REVIEW ARTICLE

The Epidemiology, Diagnosis, Management, and Prevention of Ectoparasitic Diseases in Travelers

James H. Diaz, MD, Dr PH

Schools of Public Health and Medicine, Louisiana State University Health Sciences Center, New Orleans, LA, USA

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Ectoparasitic diseases have been reported in travelers returning from both developed and developing nations.[1–3] Ectoparasitic diseases afflict the skin and its appendages and orifices, especially the scalp, facial, and pubic hairs; external ears; nares; orbits and eyelids; and genitourinary and rectal orifices. Like endoparasites, ectoparasites may be either obligatory parasites, which need to feed on human hosts to complete their life cycles, or facultative parasites, which prefer to feed on nonhuman hosts and infest humans only as accidental or dead-end hosts.[4,5]

Patients and Methods

A MEDLINE search, 1966 to 2004, of the world's salient scientific literature of case reports, case series, laboratory investigations, epidemiological investigations, and reviews was conducted in order to determine the current global epidemiology and outcomes of ectoparasitic diseases in travelers. The ectoparasitoses studied included (1) pediculosis, (2) myiasis, (3) tungiasis and other flea infestations, (4) scabies and other mite infestations, and (5) miscellaneous true insect infestations. A classification by clinical outcomes and by taxonomy of causative species of the most commonly encountered ectoparasitoses was developed and featured in Table 1. Tick-borne infections were not treated in this review because of the unique capability of ticks to transmit both endoparasitic and other systemic infectious diseases.

The Epidemiology of Ectoparasitic Diseases in Returning Travelers

To assess the potential combined impact of increasing international travel and the relaxation of quarantine regulations for imported animals in the UK on arthropod-induced dermatoses, McGarry and colleagues described their analyses of 73 insect specimens removed from symptomatic patients and submitted to their laboratory for expert identification at the Liverpool School of Tropical Medicine and Hygiene, during the years 1994 to 2000.[1] Of the 73 specimens identified, there were 27 ticks, 24 flies, 15 miscellaneous insects, and 7 mites.[1] Most of the arthropod dermatoses originated in the UK (n = 46, 63%) and were caused by tick bites (n = 18), principally *Ixodes ricinus* (the common sheep tick), an important European vector of Lyme disease and neuroborreliosis.[1] Myiasis cases predominated in returning travelers (n = 18, 67%), principally furuncular myiasis from larval infestation by *Cordylobia anthropophaga* (n = 9), the Tumbu fly, or *Dermatobia hominis* (n = 4), the human botfly.[1] All the mite infestations originated within the UK, with most caused by *Dermanyssus gallinae*, the indigenous poultry red mite (UK), also known as the red chicken mite in the Americas (n = 4).[1] Among the arthropod dermatoses caused by miscellaneous insects, most were due to pediculosis pubis caused by infestation with *Pthirus pubis*, the crab louse (n = 7), or to hemorrhagic, bullous bite groupings caused by *Cimex lectularius*, the common bedbug (n = 3).[1] Among bedbug bite cases, there were two indigenous cases, and one case in a traveler returning from Italy.[1] The authors

Corresponding Author: James H. Diaz, MD, Dr PH, School of Public Health, Louisiana State University Health Sciences Center in New Orleans, 1600 Canal Street, Suite 800, New Orleans, LA 70112 USA. Tel: (504) 599-1067; Fax: (504) 568-6905; E-mail: jdiaz@lsuhsc.edu

Table 1 Common human ectoparasites

Taxonomy and representative species of ectoparasites	Common names of ectoparasites	Geographic distributions of ectoparasites	Clinical ectoparasitoses and/or infectious diseases transmitted by ectoparasite vectors
Class Insecta			
Order Anoplura	Lice		
Pediculus humanus var *corporis*	Body louse	Worldwide	Pediculosis corporis
			Trench fever
		Worldwide	Epidemic typhus
			Louse-borne relapsing fever
Pediculus humanus var *capitis*	Head louse	Worldwide	Pediculosis capitis
Phthirus pubis	Crab (pubic) louse		Pediculosis pubis (phthiriasis)
Order Diptera			
Family Calliphoridae	Flies		
Auchmeromyia senegalensis	Congo floor-maggot fly	Sub-Saharan Africa, Cape Verde Islands	Larvae are nocturnal blood feeders, no myiasis
Callitroga americana	American screwworm	North and Central America	Wound (cutaneous) myiasis
Chrysomya bezziana	Old World screwworm	Tropical Africa, Asia, Indonesia	Cavitary (invasive) myiasis
Cochliomyia hominivorax	New World screwworm	Central America	Cavitary (invasive) myiasis
Cordylobia anthropophaga	Tumbu (mango) fly	Africa	Furuncular myiasis
Family Oestridae			
Cuterebra spp	Rodent botfly	North and Central America	Furuncular myiasis
Dermatobia hominis	Human botfly	Central and South America	Furuncular myiasis
Family Sarcophagidae			
Sarcophaga spp	Flesh (wound) fly	Worldwide	Wound (cutaneous) myiasis
Wohlfahrtia spp	Nose (flesh) fly	Worldwide	Wound (cutaneous) myiasis
Order Heteroptera	True bugs		
Cimex lectularius	Common bedbug	Worldwide	Hemorrhagic bullous bite lesions, Chagas disease, *Tsukamurella* infections, Hepatitis B
Cimex hemipterous	Tropical bedbug	Subtropical and tropical areas worldwide	Hemorrhagic bullous bite lesions, Chagas disease, *Tsukamurella* infections, Hepatitis B
Order Siphonoptera	Fleas		
Ctenocephalides spp	Cat & dog fleas	Worldwide	Bite groupings (mechanical vectors of dog and rat tapeworms), Bubonic plague, Murine typhus may be transmitted by cat fleas (*R typhi* and *R felis*) and European mouse fleas (*R typhi*)
Pulex irritans	Human flea	Worldwide	Bite groupings, Bubonic plague vector in Chilean Andes
Tunga penetrans	Chigoe (jigger) flea	Central and South America, Africa	Tungiasis
Xenopsylla cheopis	Oriental rat flea	Europe, Asia, Africa, Americas	Most efficient bubonic plague vector
Class Arachnida			
Order Acarina	Mites and ticks		
Allodermanyssus sanguineus	Common or European mouse mite	Worldwide	Rickettsial pox (*R akari*)
Dermanyssus gallinae	Red chicken mite (Poultry red mite)	Worldwide	Potential St. Louis and Western Equine encephalitis vectors in United States and Canada
Ornithonyssus bacoti	North American rat mite	Worldwide	Papulovesicular dermatitis, potential endemic typhus (*R typhi*) vector

Continued

Table 1 Continued

Taxonomy and representative species of ectoparasites	Common names of ectoparasites	Geographic distributions of ectoparasites	Clinical ectoparasitoses and/or infectious diseases transmitted by ectoparasite vectors
Pediculoides ventricossus	North American grain-itch mite	United States and Canada	Farmer's, straw-itch, or grain-itch dermatitis
Sarcoptes scabiei	Itch (scabies) mite	Worldwide	Scabies
Trombicula alfreddugesi	Common chigger (redbug)	Worldwide	"Chiggers"
Trombicula akamushi	Asian rodent chigger	Southeast Asia	Scrub typhus
Trombicula deliensis	Indian rodent chigger	Australia, Indo-Pacific Islands	Scrub typhus

concluded that both the domestic and exotic tick infestations were caused by species that could be vectors of potentially fatal diseases and that exotic infestations, particularly myiasis, predominated in returning travelers from Africa and Latin America.[1]

Recent epidemiologic evidence now clearly supports the hyperendemicity of several ectoparasitic diseases and their vectors and reservoir hosts throughout the developing world. Ectoparasitic diseases have reemerged in areas where they were once controlled, such as in Belize, Mexico, and the Caribbean. Ectoparasitic diseases have also reemerged in the developed world because of increased international trade and travel and growing populations of accessible and susceptible, and often immunocompromised, human hosts.

The Diagnosis, Management, and Prevention of Ectoparasitic Diseases in Returning Travelers

Pediculosis

Pediculosis or lice infestations remain very common in both the developing world and the industrialized world with a prevalence rate for pediculosis capitis, or head lice, exceeding 25% in the elementary schools of all industrialized countries.[6] Displaced, homeless, and refugee populations worldwide remain at greatest risks of pediculosis corporis or body lice infestations, and epidemics of body louse-borne bacterial diseases including trench fever (*Bartonella quintana*) and epidemic typhus (*Rickettsia prowazekii*) have occurred recently among refugees in Burundi (1995–1997) and Russia (1998). The prevalence of head lice is now increasing in the United States and in the UK.[4,6] Both *Pediculus humanus* (head and body lice) variants and *Pthirus pubis* (crab lice) have demonstrated high levels of resistance worldwide to the safest topical pediculocides, including the natural pyrethrins and the synthetic pyrethroids (permethrin, phenothrin).[5,7–10] In addition, increasing resistance to malathion, an organophosphate insecticide, both alone and combined with pyrethroids, has been reported in the UK and elsewhere.[11] The increasing resistance of head and pubic lice to pyrethrins, pyrethroids, and malathion has led to the increasing use of lindane in treating drug-resistant pediculosis capitis and pubis.[6,12] Lindane is an organochlorine insecticide that bioaccumulates in adipose and nerve tissue with overapplication or accidental ingestion and can cause seizure activity, especially in children.[6,12]

Pediculosis is a complex of three different human infestations with two species of blood-sucking lice of the insect order Anoplura, *Pediculus humanus* and *Pthirus pubis*, the pubic louse. Since prehistory, *Pediculus humanus* has evolved into two clinically and epidemiologically distinct, yet morphologically indistinct, ectoparasites, *Pediculus humanus* var *corporis*, the body louse, and *Pediculus humanis* var *capitis*, the head louse. The body louse may have evolved from the head louse after early man began to wear clothes. Thus, man is the definitive host reservoir for lice, and travelers are at risk of contracting pediculosis from direct contact, indirect contact via fomites, or sexual contact with lice-infested humans.

Pediculosis capitis, or head lice, is the most common type of pediculosis, afflicting millions of people annually, mostly school-aged children, in both developing and industrialized nations. Body lice infestations, or pediculosis corporis, are associated with poor hygiene and socioeconomic status, and primarily infest the indigent, displaced, homeless, and immunocompromised. Unlike head lice and pubic, or crab lice, body lice can transmit bacterial diseases, including trench fever caused by *Bartonella quintana*, epidemic typhus, caused by *R prowazekii*, and louse-borne relapsing fever, caused by *Borrelia recurrentis*. Head and body lice are transmitted by direct contact between infested individuals or by indirect contact with bedding, brushes, clothing, or headgear, according to louse species. Pubic lice are usually transmitted during sexual contact and often coexist with scabies and other sexually transmitted diseases, such as human immunodeficiency virus (HIV)/acquired immunodeficiency syndrome (AIDS).

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 1 mm (public lice) to 3 mm (head and body lice) in length, have three pairs of legs ending in powerful claws to grip hair shafts, and exhibit a reddish-brown hue after blood feeding. Females deposit their nits on hair shafts at the skin surface, and nymphs hatch within 6 to 10 days. Nits are oval in shape, less than 1 mm in diameter, grayish-white in color, and fluoresce in ultraviolet or Wood's light, when viable. Nymphs resemble miniature adults and grow to adulthood within 10 days. After hatching, empty egg cases remain attached to hair shafts and are not diagnostic of active infection.

Unfortunately, the ideal pediculocide with 100% killing activity against lice and nits does not exist. Table 2 presents the most commonly used pediculocides for lice infestations. As noted, drug resistance is increasing to the safest pediculocides, the pyrethrins and synthetic pyrethroids, and even to malathion, an organophosphate insecticide with 95% ovicidal efficacy against viable nits.[6,12]

Head lice and their viable nits are often attached to hairs close to the scalp, especially in occipital and postauricular locations. The clinical manifestations of pediculosis capitis range from asymptomatic infestation to severe pruritus with self-inflicted, and often secondarily infected, excoriations and impetigo, with postoccipital lymphadenopathy. The differential diagnosis of pediculosis capitis includes eczema, lichen simplex chronicus, dandruff, seborrheic dermatitis, and impetigo.[13] Management includes two topical or systemic treatments with pediculocides, 7 to 10 days apart, and removal of all viable nits by carefully combing wet hair.[6,12] Olive oil, petroleum jelly, or HairClean 1-2-3® are preferred hair-wetting agents, and plastic combs are preferred over metal combs.[6,12] Prevention strategies include combinations of (1) avoiding contact with potentially contaminated fomites, such as hats,

Table 2 Recommended pediculocide treatments for pediculosis

Pediculocide formulations	Trade names (availability)	Therapeutic efficacy	Safety profile	Contraindications
0.33% pyrethrins + 4% piperonyl butoxide shampoo	A-200® (OTC), RID® (OTC)	95% ovicidal in susceptible strains, no residual activity, increasing drug resistance	Excellent	Chrysanthemum and daisy (Plant Family Compositae) allergies
1% to 5% permethrin cream rinse	Acticin® (OTC), Elimite® (Rx), Kwellada-P® (OTC), Nix® (OTC)	2-week residual activity, increasing drug resistance	Excellent	Prior allergic reactions
0.5% malathion lotion, 1% malathion shampoo (unavailable in United States)	Ovide® (Rx)	95% ovicidal in susceptible strains, rapid (5 min) killing, good residual activity, increasing drug resistance	Flammable 78% isopropyl alcohol vehicle stings eyes, skin, and mucosa, increasing drug resistance, organophosphate poisoning risks with overapplications and ingestions	Infants and children under 6 months of age, pregnancy breast feeding
1% lindane lotion and shampoo	Kwellada (Rx)	95% ovicidal, no residual activity, increasing drug resistance	Potential for CNS toxicity from organo chlorine poisoning, usually manifesting as seizures, with overapplications and ingestions	Preexisting seizure disorder, infants and children under 6 months of age, pregnancy breast feeding
Ivermectin, 200 mcg/kg single po dose, repeated in 10 days; 0.8% shampoo (unavailable in United States)	Mectizan® (Rx), Stromectol® (Rx)	Excellent	Excellent	Safety in pregnancy uncertain, not recommended for children weighing <15 kg

CNS = central nervous system; OTC = over the counter; Rx = available by prescription only (United States).

headsets, clothing, towels, combs, brushes, bedding, and upholstery; (2) soaking all combs and brushes in isopropyl alcohol or 2% Lysol® solution; (3) sanitizing the environment by high heat-cycle washing and drying of all bedding, clothing, and headgear; and (4) inspecting high-risk school children for active head lice and viable nits.[6,12,13]

Since body lice only visit their human hosts to blood feed, adults, nymphs, and nits are found in clothing, usually aligned along inner seams. Despite the fact that body lice reside in clothing and not on the body-like head lice, pediculosis corporis is much more symptomatic than pediculosis capitis and causes severe pruritus with extensive self-inflicted excoriations. The sites of blood feeding often present as erythematous macules, papules, or areas of papular urticaria with a central hemorrhagic punctum.[13] The differential diagnosis includes eczematous dermatitis, lichen simplex chronicus, and scabies.[13] Management includes initial bathing with soap and water, followed by two topical or systemic treatments with pediculocides, 7 to 10 days apart (Table 2). Topical medications should be applied to clean affected areas, allowed to dry, and not rinsed for 8 (malathion) to 24 (pyrethrins, pyrethroids) hours. Prevention and control strategies for pediculosis corporis should include (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; and (3) institution of basic personal hygiene and sanitation measures, including showering, body washing, and clean clothing changes.

Crab lice infestation (pediculosis pubis or phthriasis) is moderately symptomatic compared to pediculosis corporis; it affects all hair-bearing regions, most commonly the pubic area, but also the upper eyelashes (*pediculosis palpebrarum*), and the hairy areas of the axilla and chest. More extensive infestations occur in males 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 mouthparts embedded in skin. Clinical manifestations include papular urticaria and self-inflicted, often infected, excoriations at blood feeding sites, and regional, usually inguinal, lymphadenopathy. Pathognomic findings may include *Maculae cerulea* (*taches bleues*), bluish-gray irregularly shaped macules, 0.5 to 1 cm in diameter, scattered over the lower abdominal wall, buttocks, and upper thighs. *M cerulea* may be caused by subcutaneous tissue staining from heme pigments altered by louse saliva and digestion.[13] Differential diagnosis of crab lice includes eczematous dermatitis, seborrheic dermatitis, tinea cruris, folliculitis, molluscum contagiosum, and scabies, which frequently coexists with phthriasis.[13] Management includes initial bathing with soap and water, followed by two topical or systemic treatments with pediculocides, 7 to 10 days apart (Table 2). Prevention and control strategies should include (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; and (4) examination and laboratory testing of patients and their sexual contacts for other sexually transmitted diseases, especially scabies and HIV/AIDS.

Myiasis

Myiasis is defined as human tissue invasion by the dipterous larvae or maggots of flies and may be classified clinically as furuncular (subcutaneous) myiasis, wound (superficial cutaneous) myiasis, cavitary (atrial or invasive) myiasis, intestinal myiasis, urinary myiasis, and vaginal myiasis. Intestinal myiasis is uncommon, usually due to the accidental ingestion of maggot-contaminated food and is characterized by self-limited nausea, vomiting, and diarrhea. Urinary myiasis and vaginal myiasis are also uncommon, may coexist, and present as dysuria, hematuria, vaginal bleeding, dyspareunia, and pyuria, following larval invasion of the urethra and/or vagina.

In the past, iatrogenic, superficial myiasis was intentionally induced with the maggots of greenbottle flies (*Lucilla* spp) and bluebottle flies (*Calliphora* spp) in order to cleanse septic wounds of necrotic tissues. Although not causing myiasis, the larvae of the Congo floor maggot fly, *Auchmeromyia senegalensis*, do feed preferentially on humans but do not invade deeper epidermal tissues. The Congo floor maggot fly lays its eggs on the soil floors of huts and tents; its maggots blood feed on floor-sleeping human victims, usually at night, and return to the soil after each blood meal.

The most common forms of human myiasis are furuncular myiasis and cavitary, or invasive, myiasis. Furuncular myiasis is often caused by subcutaneous larval invasion by the Tumbu fly, *Cordylobia anthropophaga*, in Africa, and the human botfly, *Dermatobia hominis*, in the subtropical and tropical areas of the Americas. 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, necrotizing tissue destruction. *Cochliomyia*

hominivorax, the New World screwworm, is a common cause of cavitary myiasis, particularly in domestic livestock, in the Americas, and *Chrysomya bezziana*, the Old World screwworm, is a common cause of cavitary myiasis in Africa, Asia, and Indonesia. Since cavitary myiasis often results in human suffering and disfigurement as well as economic loss in livestock, several successful screwworm eradication programs were launched by the US Department of Agriculture throughout the Americas, beginning in the southeastern United States in 1957 and extended over the years by cooperative agreements with Mexico, Guatemala, Belize, El Salvador, Honduras, Nicaragua, Costa Rica, and, most recently, Panama.[14] Cavitary myiasis must be managed aggressively with surgical debridement and antibiotic therapy of secondary infections to limit subcutaneous tissue and cartilaginous damage and permanent facial disfigurement.

In 1995, Jelinek and colleagues described 13 cases of cutaneous myiasis in travelers returning from tropical countries.[3] Of the 13 cases, 6 patients returning from Africa presented with furuncular myiasis from larval infestation with *Cordylobia anthropophaga*, the Tumbu fly; 6 patients returning from the American tropics presented with furuncular myiasis caused by *Dermatobia hominis*, the human botfly; and one patient returning from Nepal presented with cutaneous myiasis due to infestation with *Hypoderma lineatum*.[3] The authors concluded that myiasis should be considered an increasingly common ectoparasitosis in the differential diagnosis of cutaneous lesions in patients returning from tropical or exotic locations.[3] Also in 1995, Gordon and coauthors reported 6 patients returning from Belize with furuncular myiasis from larval infestation with *Dermatobia hominis*.[15] The authors concluded that botfly myiasis was occurring more commonly in travelers returning from the American tropics, even from countries, such as Belize, where botfly ectoparasitosis was relatively uncommon.[16]

In 2002, Schwartz and Gur reported their experiences treating 12 patients with *Dermatobia hominis* myiasis who returned to Israel from travel to the Amazon River Basin of Bolivia during the 6-year period of 1994 to 1999.[17] In all patients, the mean time from exposure to diagnosis was 1.5 months, and every patient presented with furuncular myiasis characterized by draining, nonhealing lesions, unresponsive to antibiotic treatment.[17] The authors concluded that *Dermatobia hominis*–induced furuncular myiasis was increasing among Israeli travelers to South America and that the Amazon Basin region of Bolivia, an area, like Belize, not considered at high risk for botfly myiasis, should be designated as a new high-risk area for botfly myiasis for travelers.[17]

In 2003, Tamir and colleagues reported two Israeli travelers returning from a 1-month trip to Ghana with furuncular myiasis from larval infestation by *Cordylobia rodhani*, Lund's fly.[16] The two patients had multiple draining lesions to which they applied petroleum ointment to partially suffocate and dislodge the larvae, which were manually extracted a few days later.[16] The authors concluded that *Cordylobia rodhani* was an uncommon cause of furuncular myiasis compared to myiasis with *Cordylobia anthropophaga* larval infestation in travelers returning from Africa and myiasis with *Dermatobia hominis* larval infestation in travelers returning from Central and South America.[16] The authors also warned that *Cordylobia rodhani* myiasis could result in multiple furuncles increasing risks of sepsis or tetanus and recommended that travelers to Central Africa avoid direct contact with sand to prevent *Cordylobia rodhani* myiasis, iron any clothes left outside to avoid *Cordylobia anthropophaga* myiasis, and maintain their tetanus prophylaxis status.[16]

Although the clinical manifestations, treatments, and prevention strategies are similar in cavitary and furuncular myiasis, the mechanisms of larval invasion are often different. The female Tumbu fly deposits its eggs on moist soil or on wet clothing (eg, cloth diapers) hung outside to dry. When the human victim dons the 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. The intermediate vector then delivers the botfly eggs to its blood meal victims, where the eggs hatch immediately and release larvae on warm-blooded hosts. The botfly larvae then rapidly burrow into the skin with sharp mandibles in order to begin their developmental instar stages. Victims may recall a flying insect bite that preceded 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. Anterior hooklets anchor the maggots in place subcutaneously, making manual removal, even with forceps, difficult.

Management strategies for furuncular myiasis include coaxing embedded larvae to emerge from furuncles by covering their respiratory spiracles, often visible as dark specks within lesions, with occlusive coatings of Vaseline® (petroleum) ointment,

clear fingernail polish, tobacco tar, pork fat, or, even, bacon strips.[18] Unsuccessful occlusive therapy may, however, asphyxiate the larvae and necessitate surgical removal of the retained dead maggot. Although lidocaine injections into draining furuncles to dislodge larvae were recommended in a case report, the most successful management strategies for furuncular myiasis have included initial asphyxiation techniques to dislodge superficially embedded larvae, followed by sterile surgical extraction and debridement to remove deeply embedded larvae and any larval fragments or necrotic tissues.[15–19] Along with larval extraction, all myiasis wounds should be cleansed and conservatively debrided, tetanus prophylaxis administered, and bacterial secondary infections treated with antibiotics. Prevention and control strategies for myiasis include (1) control of domestic and livestock animal larval infestations; (2) sanitary disposal of animal carcasses and offal to deny flies of their preferred breeding grounds; (3) proper management of any open human wounds or cutaneous infections; (4) cementing floors to deny floor maggot flies of 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-impregnated; (7) spraying exposed skin with diethyl toluamide-containing (DEET) repellants; and (8) ironing all clothes and diapers left outside to air-dry in Tumbu fly habitats.

Flea Infestations

Household infestations with cat fleas (*Ctenocephalides felis*) and dog fleas (*Ctenocephalides canis*) remain common but are easily preventable in households by using domestic pet insecticidal dusting powders, or solutions, and flea collars. By contrast, outbreaks of human flea (*Pulex irritans*) infestations, especially in hospitals, are rare. In 2000, Thomas and colleagues reported an outbreak of human flea infestation in 24 patients and staff in a Welsh convalescent hospital.[20] The flea infestation was initially managed as a zoonotic infestation with institutional spraying with pyrethroids and a search for domestic cat or dog fleas imported from household pets by staff or patients.[20] Despite these control and investigation efforts, the outbreak continued unabated until a temporary worker, the worker's locker, and the worker's entire home and family were all discovered heavily infested with human fleas.[20] Control efforts were then successfully redirected toward the index case and the household source of infestation.[20] The authors cautioned against assuming that all institutional flea infestations are due to imported domestic animal fleas and assuming that the index case will always be symptomatic.[20]

Fleas of the insect order Siphonaptera are a small group of morphologically similar wingless ectoparasites of warm-blooded animals that are not only biting or burrowing nuisances but also competent vectors of infectious diseases, most notably *Yersinia pestis*, the bubonic plague agent. Fleas are laterally flattened, brown insects, 1 to 3 mm in length. Fleas are perfectly adapted for blood feeding, with powerful third pairs of hind legs for springing onto hosts and siphoning mouthparts, often surrounded by hairy combs, for feeding. Fleas' preferred hosts are domestic and wild furred animals, especially squirrels, chipmunks, prairie dogs, and other ground-dwelling rodents. Although fleas are often classified by host specificity (or presence or absence of head combs), all fleas can rapidly adapt from animal to nearby human hosts, especially if preferred hosts are exterminated by diseases or pesticides. Adult fleas can blood feed on their hosts for up to a year and can survive apart from their hosts for up to 125 days.

Both male and female adult fleas can transmit infectious diseases through bites or other wounds by a variety of mechanisms including infected feeding mouthparts, infectious fecal contamination, crushed bodies of infected fleas, and, most importantly, regurgitation of bacteria that have multiplied in and blocked the proventriculus of the flea's foregut. Although the human flea, *Pulex irritans*, may transmit plague from rodents or domestic animals to man, the oriental rat flea, *Xenopsylla cheopis*, is the most efficient arthropod vector of *Y pestis*, transmitting plague from rodent to rodent and rodent to man. Rat fleas will attempt to blood feed repeatedly on convenient hosts, yet remain insatiate, regurgitating plague organisms at every bite. Endemic or murine typhus caused by *R typhi* is most commonly transmitted among rodents and from rodents to man by both *X cheopis* and *Nosopsyllus fasciatus*, the northern rat flea, by contamination of bites or scratch wounds with infectious flea feces or the crushed bodies of infected fleas.

Although adult fleas resemble each other, they differ in the presence and pattern of combs or spines (ctenidia) on their heads and in their length. The combed fleas include the ubiquitous dog (*Ctenocephalides canis*) and cat (*Ctenocephalides felis*) fleas, the European mouse flea (*Leptopsylla segnis*), the northern rat flea (*Nosopsyllus fasciatus*), and the squirrel flea [*Daimanus* (*Oropsylla*) *montanus*]. Cat and dog fleas readily bite man and pets, often act as mechanical vectors for the dog (*Dipylidium canium*) and rat (*Hymenolepis diminuta*) tapeworms, and may, rarely,

transmit these endoparasites to man after accidental ingestion of tapeworm-infected fleas. The cat flea and the European mouse flea can also transmit murine typhus caused *by R typhi*, and the cat flea has recently been found to transmit murine typhus caused by *R felis*.[21] Rat and squirrel fleas maintain epizootic plague in rodents and can also potentially transmit plague to man and domestic pets. The combless fleas are all potential plague vectors and include *X cheopis*, *Pulex irritans*, and a related New World species, *Pulex simulans*, and *Tunga penetrans*, the chigoe or jigger sand flea. *Tunga penetrans* is now hyperendemic in East Asia, India, and South America, where it originated, and in Sub-Saharan Africa, where it was introduced from South America in the late 19th century.[22]

Tungiasis is caused by the penetration of the gravid female chigoe flea into the epidermis to feed on blood and tissue juices, usually on the feet and under the toenails or in the interdigital web spaces. The embedded flea will produce a subcutaneous papule or vesicle 6 to 8 mm in diameter with a central black dot pinpointing the exteriorized segments. The papule darkens with intralesional hemorrhage, and, if squeezed, will extrude eggs, feces, and internal organs through exteriorized posterior abdominal segments. The differential diagnosis of tungiasis includes staphylococcal skin infections, bacterial and fungal paronychia, cercarial dermatitis, fire ant bites, folliculitis, and scabies.[13]

Tungiasis is now hyperendemic 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. In 1987, Schuller-Petrovic and colleagues were among the first investigators to report increasing cases of tungiasis in German travelers returning from vacations in subtropical and tropical areas of the developing world.[23] In 1996, Ibanez-Bernal and Velasco-Castrejon reported the first new cases of tungiasis ($n = 3$) in Mexico since 1948, with one infection acquired in Mexico City, a highly developed metropolitan area. [24]

Management strategies for tungiasis include extracting all embedded fleas immediately with sterile needles or curettes, administering tetanus prophylaxis, and treating secondary wound infections with appropriate antibiotics. For heavy infestations with multiple lesions, oral therapy for 3 days with either thiabendazole (25 mg/kg/day) or a single oral dose of niridazole (30 mg/kg) has been recommended.[13,25] In addition to wearing shoes, which can be sprayed with DEET solutions or dusted with 10% dichlorodiphenyltrichloroethane (DDT) powder, preventive strategies for tungiasis include (1) insecticide treatment of flea-infested domestic (and stray, if feasible) animals and pets with 10% pyrethrin spray, or 4% malathion powder; (2) foot bathing of domestic dogs and pigs, if feasible, with insecticidal solutions; and (3) spraying or dusting households, especially those with dirt floors, with 1% to 4% malathion. Other strategies for community control of fleas include spraying rodent runways and paths and household walkways and floors with solutions containing kerosene, fuel oil, 2% chlordane, 1% lindane, 3% to 4% malathion, 5% methoxychlor, or 1% trichlorfon. Although toxic to humans in overdose, the organophosphate pesticides, such as malathion, do have a better safety profile than the organochlorine pesticides, such as DDT and lindane, and do not bioaccumulate in the environment. Prior to initiating any rodent control program, the local environment must be treated thoroughly to kill all fleas and to ensure that as rats die off, their fleas will not seek new human hosts and transmit their zoonotic diseases to humans, particularly, plague and endemic typhus.

Mite Infestations

Scabies, an infestation by the itch mite, *Sarcoptes scabiei*, has remained a major public health problem throughout the developing world and has become a significant reemerging ectoparasitosis in its most severe form, Norwegian or crusted scabies, in the industrialized world, especially among the homeless and immunocompromised. Scabies is easily transmitted by skin-to-skin contact as with sex partners, children playing, or health providers examining highly infectious patients with crusted scabies. Unlike lice transmission, scabies is less easily transmitted by contact with personal grooming items, towels, or bed linens.

Scabies presents as nocturnal itching in a characteristic anatomic distribution as 10 to 15 fertile female mites are transferred from infected patients to new hosts. Female mites burrow into the thinner areas of the epidermis, usually no deeper than the stratum granulosum, to lay their eggs at the end of tunnels 5 to 10 mm long. The preferred distribution of infestation 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, and buttocks. Female mites live 4 to 6 weeks, burrow 2 to 3 mm a day dropping fecal pellets (scybala) along the way, and lay 40 to 50 eggs at the end of their tunnels, which hatch into six-legged larvae after 72 to 96 hours.

With primary infestations, the onset of pruritus and characteristic lesions are delayed up to 21 days, but following initial sensitization, symptoms and lesions return within 1 to 3 days of reinfestation. Characteristic lesions include linear to serpiginous intraepidermal burrows, 5 to 10 mm long, dotted with fecal lithes or scybala and terminating in raised papules hiding ovipositing females. Diagnosis is confirmed by microscopic examination of a burrow skin scraping, which excavates the female mite (2 to 0.5 mm in length), and surrounding eggs (0.02 to 0.03 mm) and fecal pellets.

A more severe, often sexually transmitted sensitization reaction, nodular scabies, will develop in 7% to 10% of patients with scabies and target the external genitalia, particularly in males. Scabietic nodules, usually on the penis and scrotum, will appear as darkened, tender nodules 5 to 20 mm in diameter, often with a raised female mite burrow on top.

In Africa and Southeast Asia, where T-cell leukemia-lymphoma virus (HTLV-1) is prevalent, generalized crusted (Norwegian) scabies is often a heralding marker of adult T-cell leukemia or lymphoma. Crusted scabies is now being diagnosed more commonly in homeless or displaced populations worldwide and in patients immunocompromised by HIV infection and AIDS. Unlike typical scabies, crusted scabies spreads to the face, scalp, neck, and trunk, with the most heavily infested areas capped by well-demarcated psoriatic-like plaques, which crust and scale. The differential diagnosis of scabies is extensive and includes drug reactions, eczematous dermatitis, fiberglass dermatitis, dermatitis herpetiformis, pediculosis, lichen planus, and pityriasis rosea.[13] As noted, crusted scabies may resemble psoriasis.

Recommended management strategies for scabies are listed in Table 3. With the increasing resistance of head lice and itch mites to 1% to 5% permethrin formulations, two-dose oral ivermectin, 200 mg po initially followed by another ovicidal dose in 10 days, is becoming a safe, simple alternative therapy for both pediculosis and scabies. The two-dose oral ivermectin regimen has also been recommended for the institutionalized elderly and for the control of institutional outbreaks of lice or itch mite infestations. The safety of ivermectin therapy in pregnancy has not been established. At present, ivermectin therapy is not recommended during pregnancy or for children weighing less than 15 kg. Prevention and control strategies for scabies include (1) aggressive treatment of infested patients, especially those with highly infectious crusted and nodular scabies; (2) disposal of or hot wash-dry sterilization of all contaminated clothing and bedding; and (3) provision of improved access for personal hygiene for all displaced, homeless, or institutionalized persons. Although the control of mange in domestic and stray dogs and other companion animals was once recommended as a control strategy for human scabies, recent investigations have demonstrated that mange mite infestations in animals are epidemiologically and genetically distinct from human itch mite infestations and rarely cause human zoonoses.

Less common infestations with a variety of other animal mites, some of which can serve as competent rickettsial or viral vectors, are listed in Table 1. The trombiculid, chigger, or redbug mites can transmit scrub typhus caused by *Oreintia* (formerly *Rickettsia*) *tsutsugamushi* in Southeast Asia, Australia, and the Indo-Pacific, where the most common mite vectors include *Trombicula akamushi* and *Trombicula deliensis*.[22] Scrub typhus may pose a potentially serious threat to international travelers to Southeast Asia, especially those traveling or backpacking in hyperendemic areas, such as Thailand, and exposed to secondary vegetation overgrowth in cleared or deforested areas.[26–28] Scrub typhus remains a common arthropod-borne infectious disease in Asia, with 1 billion people at risk and an estimated 1 million cases per year.[27] Scrub typhus is the most common cause of acute undifferentiated febrile illness in Thailand and has a high case fatality rate, if untreated.[28]

Among the chiggers, the adults and nymphs live in scrub brush, and blood feed on rodents and small mammals, and only the developing larvae feed on humans when incidentally encountered. Rather than burrowing into the skin, chigger larvae insert their mouthparts or capitula into the skin to feed on body fluids and epithelial cells, which accumulate in a stylosome, a tube-like feeding reservoir created by the host's inflammatory reaction to chigger saliva.[22] Chigger larvae feed on humans in the warmest topographic areas, especially in areas of tight clothing, such as the ankles, axillae, waistline, and perineum.[22]

As noted by McGarry and colleagues in Liverpool, the ubiquitous poultry red mite (UK), or red chicken mite (United States), *Dermanyssus gallinae*, caused most of the mite bites in a descriptive analysis of arthropod dermatoses in the UK over the period 1994 to 2000.[1] The red chicken mite can also cause a pruritic dermatitis usually on the backs of the hands and forearms in poultry workers and can transmit both St. Louis encephalitis and western equine

Table 3 Recommended miticide treatments for scabies

Pediculocide formulations	Trade names (availability)	Therapeutic efficacy	Safety profile	Contraindications
5% permethrin cream	Acticin® (OTC), Elimite® (Rx)	Apply from neck down, wash off after 8 to 12 h, good residual activity	Excellent	Prior allergic reactions
1% lindane lotion or cream	Kwellada® (Rx)	Apply from neck down, wash off after 8 h, no residual activity, increasing drug resistance	Potential for CNS toxicity from organo chlorine poisoning, usually manifesting as seizures, with overapplications and ingestions	Preexisting seizure disorder. Infants and children under 6 months of age, pregnancy, breast feeding
10% crotamiton cream	Eurax® (OTC)	Apply from neck down on 2 consecutive nights, wash off 24 h after second application	Excellent exacerbates pruritus	
2% to 10% sulfur in petrolatum	Generic (OTC)	Apply for 2 to 3 days, then wash	Excellent	Preexisting sulfur allergy
10% to 25% benzoyl benzoate	Generic (OTC)	2 applications for 24 h with 1 day to 1 wk interval	Irritant exacerbates pruritus, can induce contact irritant dermatitis	
0.5% malathion lotion, 1% malathion, shampoo (unavailable in United States)	Ovide® (Rx)	95% ovicidal, rapid (5 min) killing, good residual activity, increasing drug resistance	Flammable 78% isopropyl alcohol vehicle stings eyes, skin, and mucosa; increasing drug resistance; organophosphate poisoning risks with overapplications and ingestions	Infants and children under 6 months of age, pregnancy, breast feeding
2% sulfiram lotion	Generic (Rx)		Disulfiram effect	Ethanol consumption within 48 h of application
Ivermectin, 200 mcg/kg single po dose, repeated in 10 days, 0.8% lotion (unavailable in United States)	Mectizan® (Rx), Stromectol® (Rx)	Excellent, recommended for endemic or epidemic scabies in institutions and refugee camps	Excellent	Safety in pregnancy uncertain, not recommended in children weighing <15 kg

CNS = central nervous system; OTC = over the counter; Rx = available by prescription only (United States).

encephalitis.[22] The rat mite, *Ornithonyssus bacoti*, is also ubiquitous in the temperate areas of Europe and the Americas, can cause a papulovesicular dermatitis in stockyard and warehouse workers, and can transmit endemic typhus caused by *R typhi*.[22] The North American grain-itch mite, *Pediculoides ventricosus*, feeds preferentially on the larvae of insects that infest hay and straw (and some grains) and can burrow superficially into the skin causing a papulovesicular dermatitis, especially in persons sleeping on straw mattresses or riding on hay wagons. The ubiquitous dust mite, *Dermatophagoides farinae*, is a common cause of dust-borne allergic reactions and childhood asthma.[29] Last, the mouse mite, *Allodermanyssus sanguineus*, readily adapts to blood feeding on humans rather than preferred mice hosts and can transmit rickettsial pox caused by *R akari*.[22]

Prevention and control strategies for these miscellaneous mite ectoparasites include (1) household spraying of pyrethrin and pyrethroid-containing insecticides; (2) spraying pyrethrin and pyrethroid-containing repellants to clothing; (3) applying DEET-containing insect repellants to exposed skin; (4) scrubbing exposed or infested areas of the body with soap and water to remove stylosome-attached mites; (5) improving rodent control in homes, apartments, and, especially, in crowded public housing; and (6) treating straw beds and mattresses and hay-ride wagons with pyrethrin and pyrethroid-containing insecticides.

Miscellaneous Arthropod Infestations

In 2000, Paul and Bates reported their experience in identifying *Cimex lectularius*, the common bedbug,

infestations at the Brighton Public Health Laboratory during 1998 to 1999.[30] The authors reported only one specimen submitted to their laboratory and identified as *Cimex lectularius* in 1998.[30] From February to October 1999, however, the authors identified *Cimex lectularius* as the etiologic ectoparasite from four separate infestations, including the infestations of two travelers returning from Australia, one patient sleeping on an infested bed imported from the United States, and the home infestation of a healthcare worker without travel or exposure history.[30] The authors concluded that (1) bedbugs can transfer to humans from luggage as well as from bedding and furniture; (2) patients presenting with nocturnally acquired hemorrhagic bite clusters and rashes, especially in the axillae and around the waist, should be evaluated for household bedbug infestations; and (3) increasing holiday travel and international trade might be associated with increasing bedbug infestations in the UK.[30] Bedbugs can transmit Chagas disease, caused by *Trypanosoma cruzi*.[31] Although uncommon and unconfirmed, bedbugs may transmit hepatitis B and *Tsukamurella* infections, bedbug gastrointestinal commensals, during blood feeding.[32] The most commonly infesting species of bedbugs are listed in Table 1.

Conclusions

Ectoparasites have plagued wild and domestic animals and man ever since prehistory. Although humans are the preferred host reservoirs for lice and scabies, other ectoparasites are endemic wherever there are preferred animal hosts, such as rodents, small mammals, domestic animals, and livestock. However, when offered the selective advantages of better living and feeding conditions, most animal ectoparasites quickly readapt their habitats, life cycles, and feeding behaviors to human hosts. Ectoparasites are now hyperendemic in many areas of the subtropical and tropical world, where rodent and stray animal control programs have languished. The ectoparasitoses are no longer diseases afflicting only barefoot children, the displaced, the socioeconomic ally disadvantaged, and the immunocompromised. Human ectoparasitoses, such as myiasis and tungiasis, have now reemerged as unusual, and frequently misdiagnosed, diseases of tourists, executives, missionaries, and soldiers from industrialized nations returning from vacations or job-related assignments in exotic locations in developing nations. Human ectoparasitoses, such as crusted scabies, have also reemerged as opportunistic diseases of patients immunocompromised by advancing age, cancer, or infectious diseases, especially HIV/AIDS, who may acquire ectoparasitoses in their own homes from bedding, fomites, and casual, or sexual, contacts.

Some of the most common ectoparasites, principally flea, lice, and mite infestations, have become increasingly resistant to the safest insecticides, such as the natural pyrethrins and synthetic pyrethroids. Since fleas and lice can transmit a variety of infectious diseases, including bubonic plague, trench fever, endemic and epidemic typhus, and scrub typhus; disease prevention and control strategies now require the mass applications of more toxic pesticides, including the organophosphates and organochlorines.

Clinicians should be aware of the possibility of human ectoparasitoses in travelers to make timely diagnoses and institute proper therapies ranging from surgical therapies for myiasis and tungiasis to topical and systemic therapies for pediculosis and scabies. In addition, public health officials should be informed of regional ectoparasitic disease outbreaks in order to institute investigation, prevention, and control strategies to protect vulnerable populations, including vacationers, missionaries, aid workers, refugees, soldiers, the homeless, the institutionalized, and the immunocompromised.

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Declaration of Interests

The author states that he has no conflicts of interest.

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