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Review

The Global Epidemiology, Public Health Outcomes, Management, and Prevention of Re-Emerging Ectoparasitic Diseases

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Abstract. The ancient ectoparasitic diseases share many features in common with newly emerging infectious diseases, such as Lyme disease, including hyperendemic causative agents afforded selective advantages by changing ecological or socioeconomic conditions; origination as zoonoses; transmission by competent arthropod vectors; and introduction into new, susceptible host populations. Many ectoparasites are also developing increased resistance to medical therapies, including the safest insecticides. Over the past two decades, there have been several reports of outbreaks of ectoparasitic diseases, principally myiasis, scabies, and tungiasis, both in regional communities and in travelers returning from developing nations. Today ectoparasitic diseases infest not only executives and tourists returning from travel to developed and developing nations, but also individuals immunocompromised by advancing age and institutionalization, chronic infectious and malignant disease, malnutrition and homelessness. Ectoparasitic diseases are no longer infestations of children and socioeconomically disadvantaged populations in tropical countries; they have re-emerged as unusual, but not uncommon, infectious diseases worldwide.
Key Words: Ectoparasites; Infestations, arthropod; Infectious diseases, emerging and re-emerging.

THE GLOBAL EPIDEMIOLOGY, PUBLIC HEALTH OUTCOMES, AND PREVENTION OF RE-EMERGING ECTOPARASITIC DISEASES

Today most areas of the world are accessible within 36 hours by commercial air travel [1]. As a result, international tourism to subtropical and tropical areas of both resource-poor and developed nations has tripled over the past two decades [1]. As national economies expand and personal incomes rise, there will be even more professional time for business travel and more leisure time for vacation travel to exotic locations, especially to regions with natural attractions and pleasant climates. Several authors have now reported significant outbreaks of ectoparasitic diseases, principally myiasis, scabies, and tungiasis in developing nations and in travelers returning from developing nations [1-5]. In addition to these ectoparasitoses, the worldwide prevalence of other human ectoparasitic diseases is now increasing without regard to national economic development, including pediculosis, myiasis, and other flea, mite, and miscellaneous arthropod infestations, such as bed bugs [1, 6]. This review will examine the evolving global epidemiology of human ectoparasitic diseases in order to determine the factors responsible for their re-emergence and to develop effective strategies for the control and prevention of community outbreaks.

MATERIALS AND METHODS

To chronologically evaluate the changing global epidemiology of tropical ectoparasitic diseases and to determine the human and environmental factors responsible for the re-emergence of infectious ectoparasites with increased therapeutic resistance, data was extracted from both Medline (National Library of Medicine) and Cochrane Review (The Cochrane Collaboration, Oxford, UK) search engines, 1966-2007, of case reports, case series, descriptive epidemiological investigations, analytical and pharmaceutical studies, and reviews of ectoparasitic diseases among both regional populations and international travelers.

THE EPIDEMIOLOGY OF EMERGING INFECTIOUS DISEASES

With the exception of pediculosis and scabies, most

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human ectoparasitic diseases are caused by zoonotic diseases (*e.g*., myiasis, tungiasis), and most are arthropod-borne (pediculosis, scabies, myiasis, tungiasis). The most commonly encountered arthropod ectoparasitoses and their clinical manifestations are featured in Table 1.

Ectoparasitic diseases share many of the characteristics of currently emerging infectious diseases, such as Lyme disease and West Nile virus, and even re-emerging infectious diseases, such as leptospirosis and tularemia [7, 8]. Commonly shared characteristics of both ectoparisitoses and emerging infectious diseases include: (1) origination as zoonoses with disease establishment dependent on insect vector competency; (2) introduction into new, susceptible host populations; (3) infection by endemic agents afforded selective advantages by changing ecological and socioeconomic conditions; and (4) movement from rural to urban endemic areas, usually following migrating human host populations seeking better economic opportunities [7, 8].

Ectoparasitic diseases also share several specific etiological emergence factors with both emerging and re-emerging infectious diseases including: (1) the influence of human behavior on subsequent infections; (2) the effect of parasite genetic adaptation and change on therapeutic drug resistance; (3) the impact of international travel and the globalization of trade and commerce on disease dissemination; (4) the breakdown of local public health infrastructures; and (5) the increasing urbanization of the world's population with more inner city crowding in low-income housing [7-9]. Some recent specific examples of emergence factors shared by ectoparasitic diseases and emerging infectious diseases include: (1) promiscuous sexual behavior spreading the sexually transmitted ectoparasitic diseases, scabies and pubic lice, or pediculosis pubis; (2) microbial adaptations resulting in multi-insecticide resistant *Pediculus humanus capitis* (head lice); (3) furuncular myiasis in travelers returning from tropical vacations, and (4) a resurgence of scabies and tungiasis in both developing and developed nations with languishing public health infrastructures, particularly stray domestic animal and rodent control programs.

THE EPIDEMIOLOGY OF RE-EMERGING ECTOPARASITIC DISEASES

To assess the potential combined impact of increasing international travel and the relaxation of quarantine regulations for imported animals in the United Kingdom (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, during the years 1994-2000 [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]. Among the arthropod dermatoses caused by miscellaneous insects, most were due to pediculosis pubis caused by infestation with *Phthirus pubis*, the pubic 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 2 indigenous cases, and 1 case in a traveler returning from Italy [1]. The authors concluded that exotic infestations, particularly myiasis, predominated in returning travelers from Africa and Latin America, while pubic lice were domestic infestations, and bed bug infestations were both domestically and internationally acquired [1].

Recent epidemiological evidence now supports the endemicity of several ectoparasitic diseases and their arthropod vectors and human and animal reservoir hosts throughout the developing world and in many parts of the developed world, including Europe. Ectoparasitic diseases have also re-emerged in regions where they were once effectively controlled, such as in Belize, Mexico, and the Caribbean. Ectoparasitic diseases will continue to re-emerge in the developed world for several reasons including (1) the globalization of trade and commerce with ectoparasites and even their rodent hosts traveling worldwide on container ships and cargo airplanes; (2) the worldwide legitimate and illegal trade of exotic animals; (3) the accidental and intentional introduction of exotic animal species into new regions; and (4) the growing populations of accessible, and, often immunocompromised human hosts, living in crowded urban communities and mega-cities [6-10].

THE EPIDEMIOLOGY AND PUBLIC HEALTH OUTCOMES OF SPECIFIC RE-EMERGING ECTOPARASITIC DISEASES

Pediculosis Pediculosis is a complex of three different human infestations with two species of blood-sucking lice of the insect suborder Anoplura, *Pediculus humanus* and *Phthirus pubis*. After early man began to wear clothes, *Pediculus humanus* evolved into two clinically distinct ectoparasitic variants, *P. humanus corporis*, the body louse, and *P. humanis capitis*, the head louse. Although morphologically indistinct, these human lice variants do not inter-

breed and prefer unique anatomic niches on their human hosts. 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 low socioeconomic status, and primarily infest the indigent homeless, refugees from civil unrest, or the immunocompromised. Body and head lice are transmitted by direct contact between infested individuals, and, much less commonly, by indirect contact with fomites, such as bedding, clothing, headgear, combs and brushes. Pubic lice are usually trans-

Table 1. Common Arthropod Ectoparasites

Taxonomy and Representative Species of Infesting Arthropods	**Common Names of Infesting Arthropods**	**Geographic Distribution of Arthropods**	**Clinical taxonomy of the Arthropod-borne Ectoparasitoses**
Class Insecta			
Order Phtiraptera			
Suborder Anoplura	Lice		
Pediculus humanus corporis	Body louse	Worldwide	Pediculosis corporis
Pediculus humanus capitis	Head louse	Worldwide	Pediculosis capitis
Phthirus pubis	Crab (pubic) louse	Worldwide	Pediculosis pubis (phthiriasis)
Order Diptera	Flies		
Family Calliphoridae			
Auchmeromyia senegalensis	Congo floor-maggot fly	Sub-Saharan Africa, Cape Verde Islands	Larvae are nocturnal blood feeders, no myiasis
Callitroga americana	American screwworm	North & Central America	Wound (cutaneous) myiasis
Chrysomyia 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 & Central America	Furuncular myiasis
Dermatobia hominis	Human botfly	Central & South America	Furuncular myiasis
Order Heteroptera	True bugs		
Cimex lectularius	Common bedbug	Worldwide	Hemorrhagic bullous bite lesions
Cimex hemipterus	Tropical bedbug	Subtropical and tropical areas worldwide	Hemorrhagic bullous bite lesions. Potential vectors of Chagas disease, hepatitis B, and *Tsukamarella* spp. infections.
Order Siphonaptera	Fleas		
Ctenocephalides spp.	Cat & dog fleas	Worldwide	Bite groupings (mechanical vectors of dog & rat tapeworms)
Pulex irritans	Human flea	Worldwide	Bite groupings (plague vector in Chilean Andes)
Tunga penetrans	Chigoe (jigger) flea	Central & South America, Africa	Tungiasis
Xenopsylla cheopis	Oriental rat flea	Europe, Asia, Africa, Americas	Most efficient bubonic plague vector
Class Arachnida			
Order Acarina	Mites & ticks		
Sarcoptes scabiei	Itch (scabies) mite	Worldwide	Scabies
Trombicula alfreddugesi	Common chigger (redbug)	Worldwide	Chiggers
Trombicula akamushi	Asian rodent chigger	Worldwide	Potential scrub typhus vector
Trombicula deliensis	Indian rodent chigger	Southeast Asia, Australia, Indo-Pacific Islands	Potential scrub typhus vector

mitted during sexual contact, and often coexist with scabies and other sexually transmitted diseases.

Unlike head lice and pubic, or crab, lice, body lice can transmit several bacterial diseases. Homeless and refugee populations are at greatest risks of body lice infestations and epidemics of louse-borne bacterial diseases including: (1) relapsing fever caused by *Borrelia recurrentis*, (2) trench fever caused by *Bartonella quintana*, (3) epidemic typhus caused by *Rickettsia prowazekii*, (4) bacillary angiomatosis caused by *Bartonella henselae* and *Bartonella quintana*, and (5) subacute bacterial endocarditis caused by *Bartonella elizabethae*. *Bartonella* (formerly *Rochalimaea*) *henselae* and *B. quintana* have extensive domestic and wild animal reservoirs and have been recently recognized as infectious organisms of high importance not only in displaced populations, but also in immunocompromised subjects, particularly individuals with the acquired immunodeficiency syndrome (AIDS) [8].

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 industrialized countries [6]. The prevalence of head lice is now increasing in Southeast Asia and the United States (US), and resistance to many commonly used pediculicides is increasing worldwide [6, 11-18]. Both *Pediculus humanus* (head and body lice) variants and *Phthirus pubis* (pubic lice) have now demonstrated high levels of resistance worldwide to the safest topical pediculicides, specifically the natural pyrethrins and the synthetic pyrethroids (permethrin, phenothrin) [11-19]. In addition, increasing resistance to malathion, an organophosphate insecticide, both alone and combined with pyrethroids, has been reported in the UK and elsewhere [17]. The escalating resistance of head lice to pyrethrins, pyrethroids, and malathion has led to an increase in the use of lindane in treating drug resistant pediculosis capitis. Lindane is an organochlorine insecticide that bioaccumulates in adipose and nerve tissue with over-application and can cause seizure activity, especially in children [6].

Myiasis In 1994, Jelinek and coauthors, described 13 cases of cutaneous myiasis in travelers returning from tropical countries [20]. 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* [20]. The authors concluded that myiasis should be considered an increasingly common ectoparasitosis in the differential diagnosis of draining cutaneous lesions in patients returning from tropical or exotic locations [20]. Also in 1995, Gordon and coauthors reported 6 patients returning from Belize with furuncular myiasis from larval infestation with *Dermatobia hominis*, the human botfly [21]. The authors concluded that botfly myiasis was occurring more commonly in travelers returning from the American tropics, even from economically developed countries such as Belize [21].

In 2003, Tamir and coauthors reported two Israeli travelers returning from a 1-month trip to Ghana with furuncular myiasis from larval infestation by *Cordylobia rodhani*, Lund's fly [22]. The two patients had applied petroleum ointment to multiple draining lesions to partially suffocate and dislodge the larvae, which were manually extracted a few days later [22]. The authors concluded that *C. rodhani* was an uncommon cause of furuncular myiasis compared to myiasis with *Cordylobia anthropophaga* (the Tumbu fly) larval infestation in travelers returning from Africa and myiasis with *Dermatobia hominis* (human botfly) larval infestation in travelers returning from Central and South America [22]. The authors also warned that *C. rodhani* myiasis could result in multiple furuncles, thus increasing the risks of sepsis or tetanus, and recommended that travelers to Central Africa avoid direct contact with sand to prevent *C. rodhani* myiasis, iron any clothes left outside to avoid *C. anthropophaga* (Tumbu fly) myiasis, and maintain their tetanus prophylaxis status [22].

Flea Infestations Fleas of the insect order Siphonaptera are a small group of morphologically similar wingless ectoparasites of warm-blooded animals that are not only biting nuisances, but also competent vectors of infectious diseases, most notably *Yersinia pestis*. Although fleas are often classified by host specificity (or presence or absence of head combs), all fleas can rapidly adapt from animal to convenient human hosts, especially if preferred animal hosts are exterminated by disease or pesticides. Adult fleas can blood-feed on their hosts for up to a year, and can survive apart from their hosts up to 125 days [19].

A currently re-emerging, combless ectoparasitic flea, *Tunga penetrans*, the chigoe or jigger sand flea, is endemic in the Caribbean, Africa, and South America, where it originated, and, especially, in sub-Saharan Africa, where it was introduced from South America in the late nineteenth century [23-27]. Domestic animals, especially cats, dogs, pigs, and cattle, and rodents are the major wild animal reservoirs for *Tunga penetrans* worldwide [2-5, 23-27].

Tungiasis, a painful, cutaneous infestation with the gravid female jigger flea, is a common nuisance in endemic regions of Africa, South America, and the Caribbean, and

has potentially serious sequelae, including tetanus, loss of toenails, deformation and auto-amputation of digits [4, 23]. Tungiasis has successfully re-emerged in Mexico and throughout Central America, where it was once controlled; and has been increasingly reported in travelers returning from subtropical and tropical areas worldwide [2-5, 23-27]. 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, sophisticated metropolitan capital city [25].

In order to assess the current prevalence of several ectoparasitic infections in international travelers, Heukelbach and co-authors recently conducted the first cross-sectional exit questionnaire study of 372 tourists returning home to European countries after vacations in Brazil [5]. During their stays in Brazil, 12 (3.2%) tourists contracted tungiasis and 3 (0.8%) tourists contracted cutaneous larva migrans, a subcutaneous skin infestation with the larval hookworms of dogs and cats, principally *Ancylostoma braziliensis* [5]. The length of stay in the host country was a significant predictor for tungiasis, with a 20 times higher occurrence in tourists who remained in Brazil for more than one month [5]. The authors concluded that ectoparasitic infections were not uncommon in international travelers exiting Brazil over a recent two-week study period, and that tungiasis was four times more common than cutaneous larva migrans [5].

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 re-emerging ectoparasitosis in its most severe form, Norwegian or crusted scabies, in the industrialized world, especially among the homeless and immunocompromised. In rural areas of Central and South America, the prevalence of scabies among children approaches 100% [19]. In Bangladesh, the prevalence of scabies among children exceeds the prevalence of both acute diarrheal diseases and acute respiratory infections among children throughout the year [19]. 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 [19]. Crusted scabies is now being diagnosed more commonly in homeless or displaced populations worldwide and in patients immunocompromised by human immunodeficiency virus (HIV) infection and AIDS [19].

Scabies is easily transmitted by skin-to-skin contact as with sex partners, children playing, or even health providers examining highly infectious patients with crusted scabies. Scabies may also be transmitted by fomite contact with personal grooming items, towels, clothes, and bed linens. In summary, scabies is another re-emerging ectoparasitic disease more often associated with crowding and homelessness and immunocompromise by chronic infectious diseases, such as AIDS and tuberculosis, than with poor personal hygiene.

The Global Epidemiology and Public Health Outcomes of Ectoparasitic Infections with Pesticide-Resistant Ectoparasites

Since the late 1980s, increasing resistance of head lice to the most commonly prescribed pediculicides has been reported throughout the world [6, 11-17]. In Israel, head lice resistance to permethrin increased four-fold between 1989 and 1994 [6, 14]. Head lice resistance to natural pyrethrins, permethrin, and lindane have been reported from Canada, the Czech Republic, Denmark, France, Israel, the UK, and the US [6, 11-18]. Head lice resistance to malathion has been reported from the UK and from Africa [17].

In a randomized comparison of wet-combing versus 0.5% malathion shampoos for head lice in the UK, Roberts reported a 78% cure rate for malathion shampoo versus 38% for wet-combing [6]. In an *in vitro* pediculicidal efficacy comparison of 5 pediculicides available in the US, Meinking and co-investigators reported the following results: (1) there were significant differences in the pediculicidal efficacies of the 5 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; and (4) some head lice in the US had become resistant to most pediculicides [28].

In a 2007 Cochrane Collaboration Review on the treatment of scabies, Strong and Johnstone noted that both topical permethrin and oral ivermectin appeared most effective for individual infections; more research would be needed to compare the effectiveness of malathion to permethrin for individual infections; and there was insufficient evidence to recommend specific miticides to control community and institutional outbreaks of scabies [29]. In short, there is recent evidence of increasing resistance of head lice to the safest pediculicides, the natural pyrethrins and the synthetic pyrethroids, and more research will be required to determine the best treatment strategies for controlling outbreaks of scabies.

Malathion, an organophosphate pesticide, has demonstrated the greatest therapeutic efficacy against head lice in the US [28]. Carbaryl, a carbamate pesticide, highly effective against both head lice and scabies, is being increasingly

prescribed for *pediculosis capitis* outside of the US, especially in Europe and the UK. Lastly, therapeutic options for the control of scabies and crusted scabietic super-infestations in community, institutional, and refugee shelter outbreaks remain untested.

Unfortunately, all of the topical pesticides used to treat ectoparasitic infections share the same three characteristics as the three most commonly ingested childhood poisons. They are (1) prescribed (often over-the-counter) medications, (2) household products, and (3) pesticides. As the prevalence of ectoparasitic infections with pesticide-resistant ectoparasites in children increases, alternative pesticides, more toxic than pyrethrins and pyrethroids, may be prescribed for ectoparasitic infestations; medications will continue to be administered in households; and household accidental ingestions of more toxic pesticide formulations for head lice and scabies may increase without enhanced public health education measures.

THE CLINICAL MANIFESTATIONS, MANAGEMENT, AND PREVENTION OF RE-EMERGING ECTOPARASITIC DISEASES

Pediculosis The clinical manifestations of pediculosis capitis range from asymptomatic infestation to severe pruritus with self-inflicted, often secondarily infected, excoriations with impetigo and postoccipital lymphadenopathy. The differential diagnosis of pediculosis capitis includes, eczema, lichen simplex chronicus, dandruff, seborrheic dermatitis, and bacterial impetigo [19]. Management includes two topical or systemic treatments with pediculicides, 7-10 days apart, and removal of all viable nits by carefully combing wet hair [6, 12, 18]. Olive oil, petroleum jelly, or Hair-Clean 1-2-3® are preferred hair-wetting agents prior to combing [6, 12, 18]. Unfortunately, no ideal pediculicide with 100% killing activity against lice and nits exists [6, 12, 18, 28]. Table 2 presents the most commonly used pediculicides for lice infestations. As noted, drug resistance is increasing to the safest pediculicides, the pyrethrins and synthetic pyrethroids, and to malathion, an organophosphate insecticide with 95% efficacy against viable nits [6, 11-19, 28].

Prevention strategies for head lice include combinations of sanitizing the environment, and, more importantly, eliminating all human reservoirs of head lice in households, apartments and other housing complexes, classrooms, and schools [18, 29-31]. Some common preventive interventions include: (1) avoiding contact with potentially contaminated items, such as hats, headsets, 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 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, 18, 19, 29-32].

Prevention strategies for body and pubic lice are similar and 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 labo-

Table 2. Recommended Pediculicide Treatments for Pediculosis

Pediculicide Formulations	Therapeutic Efficacy	Safety profile	Contraindications
0.33% pyrethrins + 4% piperonyl butoxide shampoo	95% ovicidal. No residual activity. Increasing drug resistance.	Excellent	Chrysanthemum & daisy (Plant Family Compositae) allergies
1-5% permethrin cream rinse	2-week residual activity. Increasing drug resistance.	Excellent	Prior allergic reactions
0.5% malathion lotion, 1% malathion shampoo (unavailable in USA)	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 over-applications and ingestions.	Infants and children under 6 months of age. Pregnancy Breast feeding
1% lindane lotion and shampoo	95% ovicidal. No residual activity. Increasing drug resistance.	Potential for CNS toxicity from organo-chlorine poisoning, usually manifesting as seizures, with over-applications and ingestions.	Pre-existing 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 USA)	Excellent	Excellent	Safety in pregnancy uncertain. Not recommended for children weighing < 15 kg.

CNS: central nervous system.

ratory testing of patients and their sexual contacts for other sexually transmitted diseases, especially scabies and 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. Relatively uncommon, intestinal myiasis is usually due to the accidental ingestion of maggot-contaminated food and is characterized by self-limited nausea, vomiting, and diarrhea. Urinary myiasis is also uncommon and may present as dysuria, hematuria, and pyuria, following larval invasion of the urethra, and/or vagina (vaginal myiasis).

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 botflies in subtropical and tropical areas of the Americas, including the human botfly, *Dermatobia hominis*, and *Cuterebra* spp. botflies [20-22, 33-35]. 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 destruction. *Cochliomyia hominivorax*, the New World screwworm, is a common cause of cavitary myiasis in the Americas; and *Chrysomya bezziana*, the Old World screwworm, is a common cause of cavitary myiasis in Africa, Asia, and Indonesia. Cavitary myiasis must be managed aggressively with surgical débridement and antibiotic therapy of secondary infections to limit tissue damage.

Although the clinical manifestations, treatments, and prevention strategies are similar in furuncular myiasis; the mechanisms of larval invasion are often different. The female Tumbu fly deposits its eggs on moist 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. 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. The botfly larvae then rapidly burrow into the skin with sharp mandibles in order to begin their developmental instar stages. After completing three instar stages, the final larval forms of both the Tumbu fly and botfly will wriggle out of their draining, boil-like furuncular swellings, drop to the ground, and pupate into adult flies within 9-14 days. Victims rarely 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 in lesions, with occlusive coatings of Vaseline® (petroleum) ointment, clear fingernail polish, tobacco tar, pork fat, or, even, bacon strips [21, 22, 33-35]. The injection of lidocaine into draining lesions has also been recommended as a successful extraction technique [35]. Nevertheless, unsuccessful occlusive therapy may asphyxiate larvae and necessitate their surgical extraction. Along with larval removal or surgical extraction, 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 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-impregnated; (7) spraying exposed skin with diethyl toluamide-containing (DEET) repellants; and (8) ironing all clothes and diapers spread left outside in Tumbu fly habitats [20-22, 33-34].

Flea Infestations 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 6-8 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, and folliculitis [19].

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 topical and/or oral antibiotics. For heavy infestations, oral therapy for 3 days with either thiabendazole (25 mg/kg/day) or albenda-

zole (400 mg/day) has been recommended [5, 19]. In addition to wearing shoes, which can be sprayed with DEET solutions or dusted with 10% DDT powder, preventive strategies for tungiasis include: (1) insecticide treatment of flea-infested domestic and stray animals and pets with 10% pyrethrin spray, or 4% malathion powder; (2) foot bathing of domestic and stray dogs and pigs with insecticide solutions; and (3) spraying or dusting households, especially those with dirt floors, with 1-4% malathion. Other strategies for the environmental control of fleas include spraying rodent runways and paths and household walkways and floors with solutions containing kerosene, fuel oil, 2% chlordane, 1% lindane, 3-4% malathion, 5% methoxychlor, or 1% trichlorfon [2, 19]. Heukelbach has emphasized the importance of controlling regional epizootic reservoirs of *T. penetrans* not only in domestic and stray dogs and cats, but also in domestic pigs and cattle, and, especially, in rats [2]. Heukelbach and co-investigators have described *T. penetrans* infestations in over 50% of rodents (*Rattus rattus*) captured in poor communities in Brazil [2].

Mite Infestations Scabies presents as nocturnal itching in a characteristic topographical distribution as 10-15 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 5-10 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. A more severe, often sexually transmitted sensitization reaction, nodular scabies, targets the external genitalia, particularly in males.

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 1-3 days of re-infestation. Characteristic lesions include linear to serpiginous intraepidermal burrows, 5-10 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- 0.5 mm in length), and surrounding eggs (0.02-0.03 mm) [19]. Scabietic nodules will develop in 7-10% of patients with scabies, usually on

Table 3. Recommended Miticide Treatments for Scabies

Miticide Formulations	Therapeutic Efficacy	Safety profile	Contraindications
5% permethrin cream	Apply from neck down; wash off after 8-12 hours. Good residual activity.	Excellent	Prior allergic reactions
1% lindane lotion or cream	Apply from neck down; wash off after 8 hours. No residual activity. Increasing drug resistance.	Potential for CNS toxicity from organo-chlorine poisoning, usually manifesting as seizures, with over-applications and ingestions.	Pre-existing seizure disorder. Infants and children under 6 months of age. Pregnancy Breast feeding
10% crotamiton cream	Apply from neck down on 2 consecutive nights; wash off 24 hours after 2nd application.	Excellent Exacerbates pruritus.	
2-10% sulfur in petrolatum	Apply for 2-3 days then wash.	Excellent	Pre-existing sulfur allergy.
10-25% benzoyl benzoate	2 applications for 24 h with 1 day to 1-week interval.	Irritant Exacerbates pruritus. Can induce contact irritant dermatitis.	
0.5% malathion lotion, 1% malathion shampoo (unavailable in USA)	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 over-applications and ingestions.	Infants and children under 6 months of age. Pregnancy Breast feeding
2% sulfiram lotion		Any ethanol consumption within 48 hours of application may cause severe nausea and vomiting (the disulfiram effect).	Ethanol consumption within 48 hours of application.
Ivermectin, 200-mcg/kg single po dose, repeated in 10 days.. 0.8% lotion (unavailable in USA)	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.

the penis and scrotum, and appear as darkened, tender nodules 5-20 mm in diameter, often with a raised female mite burrow on top.

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 corporis, lichen planus, and pityriasis rosea [19].

Recommended management strategies for scabies are listed in Table 3. Prevention and control strategies for scabies include: (1) aggressive treatment of infested patients, especially those with highly infectious crusted scabies; (2) disposal or hot wash-dry sterilization of all contaminated clothing and bedding; (3) provision of improved access for personal hygiene for all displaced, homeless, or institutionalized persons; and (4) aggressive control of outbreaks of zoonotic scabies caused by itch and mange mites of various domestic animals, especially cats, dogs, camels, pigs, and horses.

Although of limited clinical significance, a number of other mite species can cause bothersome dermatitis and even transmit infectious diseases, including scrub typhus, endemic typhus, and rickettsial pox [36]. The trombiculid species of chigger mites, or redbugs, can transmit scrub typhus caused by *Rickettsia tsutsugamushi* in North America (*Trombicula* spp.) and in Southeast Asia, Australia, and the Indo-Pacific (*T. akamushi*, *T. deliensis*) [36]. 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 then burrowing into the skin, chigger larvae insert their mouthparts or capitula into the skin to feed on body fluids and epithelial cells that accumulate in a stylosome, a tube-like feeding reservoir created by the host's inflammatory reaction to chigger saliva [36]. Chigger larvae feed on humans in the warmest topographic areas, especially in areas of tight clothing, such as the ankles, axillae, waistline, and perineum.

Miscellaneous Arthropod Infestations: Bedbugs As noted, increasing international trade and travel might be associated with increasing bedbug infestations in the UK, Europe, and the US [1, 37]. Bedbugs can easily transfer to humans for blood feeding from luggage as well as from local and imported furniture and bedding. All patients presenting with nocturnally acquired hemorrhagic, bullous bite clusters and rashes, especially in areas where clothing is tight or constricting, such as the axillae and waist, should be evaluated for acquired or domestic *Cimex* or bedbug infestations [37].

Historically, bedbugs were regarded as potential mechanical carriers but not as biological vectors of human infectious diseases. However, bedbugs can transmit Chagas disease, caused by *Trypanosoma cruzi*, in endemic regions [38]. Although uncommon and unconfirmed microbiologically, bedbugs may also transmit hepatitis B and *Tsukamarella* spp. (commensal bacteria in bedbug gastrointestinal tracts) infections during blood-feeding [39]. The most commonly infesting bedbug species are listed in Table 1.

CONCLUSIONS

The ectoparasitoses are no longer diseases afflicting only barefoot children, displaced people, and socioeconomically disadvantaged residents of tropical countries. Human ectoparasitoses, such as, myiasis, scabies, and tungiasis, have now re-emerged as unusual, and often misdiagnosed, diseases among tourists, executives, missionaries, and soldiers from industrialized nations returning from vacations or job-related assignments in locations throughout developed and developing nations, including popular resort destinations. Ectoparasitic diseases are significant sources of morbidity in humans, particularly children, and should not be neglected as international outbreaks will continue to occur with resistant strains that are difficult to control.

Clinicians should be aware of the re-emergence of human ectoparasitoses in order 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 executives, vacationers, missionaries, aid workers, refugees, soldiers, long-term care residents, and the immunocompromised.

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