
Ectoparasites



Pediculosis and tungiasis

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Learning objectives

After completing this learning objective participants should be able to discuss the clinical features and risk factors for body louse infestation, including rising homelessness in US coastal cities; describe the emerging evidence that head lice may act as a vector for severe infectious diseases; review geographic distribution, clinical signs, and risk factors for tungiasis; and identify promising new treatments for tungiasis.

Disclosures

Editors

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Authors

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Pediculosis is an infestation of lice on the body, head, or pubic region that occurs worldwide. Lice are ectoparasites of the order Phthiraptera that feed on the blood of infested hosts. Their morphotype dictates their clinical features. Body lice may transmit bacterial pathogens that cause trench fever, relapsing fever, and epidemic typhus, which are potentially life-threatening diseases that remain relevant in contemporary times. Recent data from some settings suggest that head lice may harbor pathogens. The epidemiology, clinical manifestations, and management of body, head, and pubic louse infestation are reviewed. New therapies for head lice and screening considerations for pubic lice are discussed. Tungiasis is an ectoparasitic disease caused by skin penetration by the female *Tunga penetrans* or, less commonly, *Tunga trimamillata* flea. It is endemic in Latin America, the Caribbean and sub-Saharan Africa and seen in travelers returning from these regions. Risk factors for acquiring tungiasis, associated morbidity, and potential strategies for prevention and treatment are discussed. (J Am Acad Dermatol 2020;82:551-69.)

Key words: body lice; ectoparasite; epidemic typhus; flea; head lice; homeless; homelessness; infestation; lice; pediculosis; *Pediculus capitis*; *Pediculus humanus*; *Phthirus pubis*; poverty; pubic lice; refugee; relapsing fever; returning traveler; trench fever; *Tunga penetrans*; *Tunga trimamillata*; tungiasis.

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HUMAN BODY LOUSE (*PEDICULUS HUMANUS HUMANUS*)

Key points

- Body lice infestation is associated with poor hygiene or neglect
- Given a homelessness epidemic, the diagnosis and management of body lice are essential dermatology skills
- Body lice are vectors for potentially life-threatening pathogens

Lice are obligate parasites, feeding exclusively on the blood of infested hosts.¹ Among thousands of lice species, only *Pediculus humanus* (*P humanus*) and *Phthirus pubis* (pubic lice) require humans as hosts. *P humanus* includes 2 morphotypes: *P humanus corporis* (body) and *P humanus capitis* (head) lice.²

Body lice are ovoid-shaped and measure 2.3 to 3.6 mm (Fig 1, A).³ They live on clothing and attach to nearby skin for blood meals. Females lay eggs (“nits”) on clothing seams (Fig 1, B). In favorable environments, eggs hatch into nymphs after 6 to 10 days, which mature into adults that live 1 to 3 months.² Detached from hosts, lice die within 3 to 5 days.² Their ideal survival conditions are 29°C to 32°C and 70% to 90% humidity; they cannot survive above 50°C or below 40% humidity.⁴

Epidemiology

Lice infestations are not reportable diseases in most countries, limiting high-quality epidemiologic data collection.⁵ Known risk factors include close contact with infested persons, infrequent showering, an inability to wash and heat-dry clothing, and cold weather, which decreases the frequency of showering and changing clothes. These are especially prevalent among homeless persons,^{4,6,7} making body lice infestation more common in this population.⁷⁻¹⁰ In urban settings of high-income countries, ≤30% of homeless persons are infested with body lice.⁸ Unsheltered persons are more frequently affected.^{8,9} Additional risk factors include substance abuse, not showering, and previous pubic lice infestation.^{9,10} Untreated mental illness, which is more prevalent among homeless people in Western countries compared with the general population,^{11,12} may contribute to poor hygiene. Known prevalence and risk factors for infestation among homeless persons are shown in Table I.

The United States is currently experiencing a homelessness “epidemic,” particularly on the West Coast, where several cities have declared states

of emergency.¹³⁻¹⁵ From 2016 to 2017, homelessness increased for the first time in 7 years, by 9%, mostly because of a rise in unsheltered homeless persons.¹⁴ Chronic (>1 year) homelessness also increased by 12%.¹⁴ During this period, homeless populations have risen in several European countries.¹⁶

Clinical manifestations

Patients with body lice infestation present with generalized pruritus and lesions distributed on the neck, shoulders, upper back, flanks, and waist—sites of close contact between clothing and the skin (Fig 2). Common lesions include excoriations and eczematous patches; papular urticaria and bullae may be seen. Prurigo nodules, lichenification, and hyperpigmentation are present in chronic infestation. Scratching predisposes to impetigo, ecthyma, and cellulitis. Diagnosis is achieved by carefully examining clothing seams (“clothing biopsy”) for lice and nits. Severe iron deficiency anemia¹⁷⁻¹⁹ and eosinophilia^{17,19} have been associated with heavy/chronic body lice infestation.

Implications

Body lice transmit several potentially life-threatening infections (Table II). Despite being rare overall, these conditions remain major public health concerns in poor hygiene conditions.

Body lice transmit *Bartonella quintana*, which causes trench fever, named for its high prevalence among soldiers fighting together in unhygienic trenches during World War I.^{20,21} More recently, trench fever was seen in refugees after the civil war in Burundi,²² and since the 1990s, “urban trench fever” has been recognized in homeless and urban poor populations worldwide.²³⁻²⁵ *B quintana* was identified in 21% of body lice in Marseilles, France,²⁶ 28.2% in Bogotá, Colombia,²⁷ 13.35% in northern Algeria,²⁸ 13.1% in Turkey,²⁹ and 15.9% in San Francisco, California.⁸ Trench fever classically manifests as headache, dizziness, conjunctival injection, severe shin pain, lymphadenopathy, a macular evanescent rash, and relapsing fevers lasting 4 to 8 days.^{2,21,30} Contemporary *B quintana* infections present with variable manifestations: asymptomatic infections, relapsing fevers, headache, leg pain, “culture-negative” endocarditis, and, in immunocompromised persons with HIV or a previous transplant, bacillary angiomatosis.^{25,31,32} Diagnosis relies on culture, serology, immunohistochemical staining, or polymerase chain reaction assay. *B quintana* grows slowly in culture, often requiring ≥14 days and up to 45 days.³³ The microbiology laboratory should be notified when

Abbreviations used:

FDA: US Food and Drug Administration
LBRF: louse-borne relapsing fever
RCT: randomized controlled trial

Bartonella is suspected because several techniques exist to optimize growth.³³ First-line treatment is gentamicin (3 mg/kg/day for 2 weeks) followed by doxycycline (200 mg/day for 4 weeks).³⁰ In bacillary angiomatosis, first-line treatment is erythromycin (500 mg 4 times daily) or doxycycline (100 mg 2 times daily) for ≤ 3 months in immunocompromised hosts.^{30,34}

The body louse also transmits *Borrelia recurrentis*, which causes louse-borne relapsing fever (LBRF). Most LBRF cases originate from the horn of Africa (Ethiopia, Eritrea, and Somalia), where *B recurrentis* is endemic and concentrated poverty contributes to sporadic epidemics.^{35,36} With the ongoing global refugee crisis, LBRF is an emerging infectious disease in Europe, seen in refugees and asylum seekers who acquired the infection in the horn of Africa, either during their journey through North Africa where migration routes into Europe join or in Europe through contact with new refugee arrivals.³⁷⁻⁴⁶ LBRF presents with an initial phase of high-grade fever, headache, dizziness, myalgias, and fatigue,³⁵ followed by shorter, less severe relapses every 7 to 19 days.² Complications include mucocutaneous hemorrhage, neurologic dysfunction, and liver or renal failure. Mortality is $\leq 40\%$ for untreated cases² and 2% to 5% when treated.⁴⁷ Serum polymerase chain reaction studies or observing organisms on blood smears confirms the diagnosis.⁴⁸ Treatment is with a single dose of intramuscular penicillin G (400,000-800,000 units) or doxycycline

200 mg once²; the optimal treatment remains unclear.⁴⁹ After antibiotic administration, observation for 4 to 6 hours is recommended⁵⁰ because a Jarisch–Herxheimer reaction is common. In LBRF, this typically occurs within 2 hours of antibiotic administration and manifests with chills/rigors, tachycardia, and hypotension that may require hospitalization for supportive care.^{51,52}

P humanus also transmits *Rickettsia prowazekii*, which causes epidemic typhus. While infected lice die within weeks, humans are the principle reservoir, remaining infected for life.²² The organism may reemerge years later during stressful periods.²² Outbreaks are associated with war, famine, crowding, refugee camps, and cold weather.^{22,53} Flying squirrels serve as a zoonotic reservoir in periods between outbreaks, when human-to-louse-to-human transmission is less common.⁵⁴ Widespread maculopapular exanthema that may become petechial are seen.²² Other features include fever, nausea, diarrhea, delirium, respiratory failure, and shock.^{2,22} Diagnosis is made via serology.⁵⁵ Mortality is $\leq 60\%$ if untreated but approximately 4% when treated.² First-line treatment is doxycycline 200 mg once.^{55,56}

Management

The management of body lice must address poor hygiene and clothing infestation. Regular showering/bathing is important. Clothes should be discarded appropriately or washed and dried using high heat ($>50^{\circ}\text{C}$).³ Associated bacterial infections should be treated with appropriate antibiotics and eczematous lesions with topical corticosteroids. Physicians should involve social services to address underlying factors driving infestation.

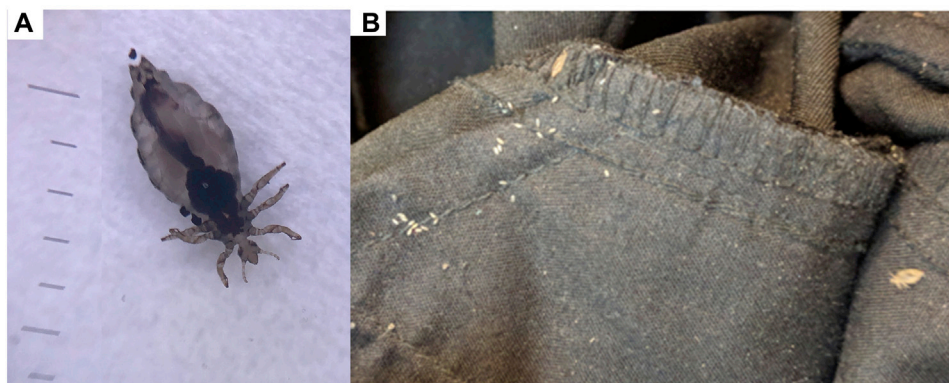


Fig 1. Body lice. **A**, Body louse morphology (courtesy of Charlotte Bernigaud, MD, Olivier Chosidow, MD, PhD, and Françoise Foulet, MD). **B**, Two lice and numerous nits (pinpoint white-yellow structures) in clothing seams (courtesy of Aileen Chang, MD).

Table I. Epidemiology of body lice infestation among homeless populations

Year	Location	Population	Prevalence of body lice	Risk factors for infestation
2014	San Francisco, California, United States ⁸	203 sheltered and unsheltered homeless persons	30% with body lice, 4.9% with head lice, and 3% with body and head lice	Male sex, African American race, and sleeping outdoors
2016	Paris, France ⁹	667 sheltered and 341 unsheltered homeless persons	5.4% of unsheltered persons with body lice and 0.15% of sheltered persons with body lice	Begging, history of pubic lice, and not taking showers in municipal baths
2017	Marseilles, France ¹⁰	2288 sheltered homeless persons	12.2% with body lice, 4.5% with head lice, and 3.2% with pubic lice	Older age, alcohol consumption, and tobacco smoking
2017	Bogotá, Colombia ²⁷	153 sheltered homeless persons	11.7% with body lice	Not reported

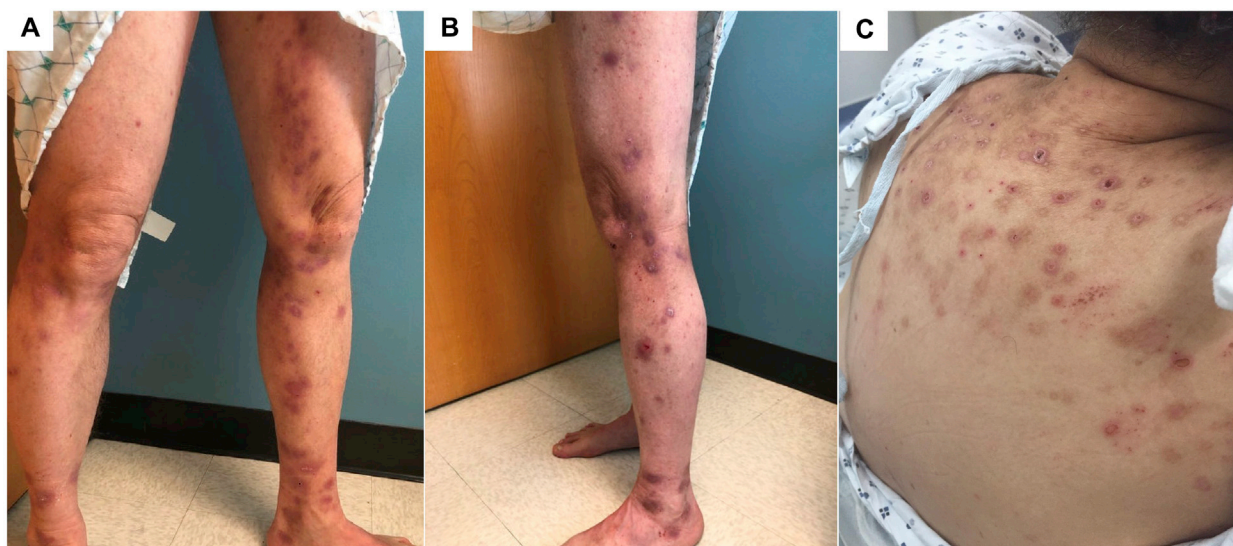


Fig 2. Clinical manifestations of body lice infestation. **A** and **B**, Bite marks along clothing seams that have evolved into prurigo nodules after chronic rubbing (courtesy of Sarah Coates, MD). **C**, Excoriations and prurigo nodules localized to upper back and shoulders in a patient with body lice infestation (courtesy of Kelly Fitzgerald, MD).

These measures can be effectively implemented during large outbreaks. In 1991, an Ethiopian refugee camp achieved large-scale delousing via shaving head and pubic hair, a 15-minute shower with soap, boiling clothing for 30 minutes, burning and replacing blankets, and spraying personal belongings with pesticide.⁵⁷ After a civil war in Burundi in the 1990s, a nationwide epidemic typhus outbreak was successfully managed with delousing interventions and doxycycline

administered to all confirmed and suspected cases of typhus.²²

In a randomized controlled trial (RCT) among sheltered homeless persons in France, persons who received permethrin-impregnated underwear were free of lice (based on the absence of living lice in underwear) on day 14 compared with placebo, but these effects were not sustained 45 days later and thus this intervention was not recommended given the lack of sustained

Table II. Human pathogens associated with body lice infestation

Pathogen	Clinical manifestations	Microbiology	Diagnosis	Treatment
<i>Bartonella quintana</i>	Trench fever: headache, dizziness, severe shin pain, lymphadenopathy, macular evanescent rash, relapsing fevers lasting 4-8 days, recurring for weeks ^{2,21,30} ; "culture-negative" endocarditis ¹⁷⁴ ; bacillary angiomatosis (in immunocompromised hosts) ^{31,32}	Facultative intracellular Gram-negative bacillus	Serology (anti- <i>Bartonella</i> antibodies); blood or tissue culture possible, but not sensitive. Slow growth in culture (≥ 14 days, ≤ 45 days) ³³	Gentamicin (3 mg/kg/day for 2 weeks), followed by doxycycline (200 mg/day for 4 weeks) ³⁰ ; erythromycin is the first-line treatment for bacillary angiomatosis
<i>Borrelia recurrentis</i>	Relapsing fever: high-grade fever, headache, dizziness, pain, anorexia, and fatigue; may progress to mucocutaneous hemorrhage, meningitis, encephalitis, liver and renal failure; relapses occur every 7-19 days ²	Spirochete	Thin and thick blood smears to identify organisms; PCR testing of serum if smear negative; CSF PCR if neurologic signs are present	Single dose IM penicillin G (400,000-800,000 units); oral doxycycline, once (200 mg for adults) monitor for Jarisch-Herxheimer reaction during first 24 hours
<i>Rickettsia prowazekii</i>	Epidemic typhus: fever, acute exanthem, diarrhea, neurologic dysfunction, respiratory failure, and shock; organism may emerge during stressful periods years later ²²	Obligate intracellular bacillus; infected lice die within 3 weeks; humans remain infected for life	Diagnose via serology; isolation in culture is impractical	Oral doxycycline 100 mg BID for 7-10 days ^{55,56} ; chloramphenicol 500 mg QID for 5 days (second-line); doxycycline 200 mg, once, in outbreaks ²

BID, Twice daily; CSF, cerebrospinal fluid; IM, intramuscular; PCR, polymerase chain reaction; QID, 4 times daily.



Fig 3. Head lice. **A**, Head louse morphology (courtesy of Arezki Izri, MD, PhD). **B**, Head louse nit attached to a hair shaft, visualized under microscopy (courtesy of Charlotte Bernigaud, MD, Olivier Chosidow, MD, PhD, and Françoise Foulet, MD). **C**, Head louse nit on a hair shaft, visualized under dermoscopy (courtesy of Aileen Chang, MD).

Table III. Treatment of head lice

Therapy	Mechanism of action	Instructions	Adverse events	Level of evidence*	Considerations
Over the counter					
Pyrethrin with piperonyl butoxide shampoo (A-200, Pronto, R&C, Rid, or Triple X)	Natural extract from the chrysanthemum flower. Blocks sodium transport, leading to depolarization of neuromembranes and respiratory paralysis	Apply to dry hair. Let sit for 10 min. Repeat in 7-10 days. Nit combing recommended (not ovicidal)	Contact dermatitis	Level 1B RCT: 50% ovicidal at day 14 ¹⁷⁵	Approved by the FDA for children >2 years of age; efficacy has waned because of resistance
Permethrin 1% lotion or cream rinse (Nix)	Synthetic pyrethrin. Blocks sodium transport, leading to depolarization of neuromembranes and respiratory paralysis	Apply after shampoo. Let sit for 10 min. Rinse. Repeat in 7 days. Nit combing recommended (not ovicidal)	Contact dermatitis	Cream rinse: level 1A RCT: 97% efficacy after 14 days ¹⁷⁶ Metaanalysis: >90% cure rates ¹⁷⁷	Approved by the FDA in 1986 for children >2 months of age; efficacy has waned because of resistance
Dimethicone 4% lotion (LiceMD)	Long-chain linear silicone dissolved in a volatile silicone base. Physical material (not an insecticide) that coats hair shafts	Apply to dry hair. Let sit for two 8-hour treatments, 7 days apart ¹⁷⁸ (reported to be ovicidal at higher concentration) ¹⁷⁹	Skin/eye irritation ¹⁷⁸	Level 1B RCT: Eradicated lice in 70% of patients ¹⁷⁸	Not approved by the FDA
Prescription					
Malathion 0.5% lotion (Ovide) or gel	Organophosphate cholinesterase inhibitor. Causes louse respiratory paralysis	Lotion: apply to dry hair. Let sit for 8-12 hours. Shampoo: repeat in 7-9 days if lice still present (partially ovicidal) Gel: Apply to dry hair. Let sit for 30 min. Repeat in 7-9 days if still present	Flammable; do not use a hair dryer/iron ⁵⁹ ; skin irritation ¹⁸⁰	Lotion: Level 1B RCT: 100% effective at 24 hours. 95.3% effective at 7 days ¹⁸⁰ Gel: Level 1B RCT: 98% response after 30 min ¹⁸¹	Withdrawn in 1995 because of flammability, then reinstated in 1999; approved for children >6 years of age ⁹⁶ Useful for permethrin-resistant lice ⁹³
Benzyl alcohol 5% lotion (Ulesfia)	Aromatic alcohol that kills lice via asphyxiation; not neurotoxic	Apply to dry hair. Let sit for 10 min. Rinse. Two applications needed (not ovicidal)	Skin irritation ¹⁸²	Level 1B Two RCTs: 75-76.2% elimination ¹⁸²	Approved by the FDA 2009 for children >6 months of age

Spinosad 0.9% suspension (Natroba)	Paralyzes lice by agonizing acetylcholine and antagonizing gamma-aminobutyric acid receptors	Apply to dry hair. Let sit for 10 min. Rinse. No nit combing required. ⁹⁷ Repeat in 7 days if lice are visualized (ovicidal) ^{97,98}	Skin/eye irritation ⁹⁷	Level 1B RCT: 84.6-86.7% effective (after 1 or 2 applications) ⁹⁷	Approved by the FDA 2011 for children >4 years of age; expensive ¹⁸³
Ivermectin 0.5% lotion (Sklice)	Binds glutamate-gated chloride channels in invertebrate nerve and muscle cells, leading to cellular hyperpolarization ⁹⁹	Apply to dry hair. Let sit for 10 min. Rinse. Two applications recommended (not ovicidal) ^{99,184}	Skin irritation ¹⁸⁴	Level 1B Two RCTs: 94.9% lice-free at day 2; 73.8% lice-free at day 15 ¹⁸⁴	Approved by the FDA in 2012 for children >6 months of age
Abametapir 0.74% lotion	Metalloproteinase inhibitor	Apply to dry hair. Let sit for 10 min. Rinse. No nit combing required (ovicidal)	Skin irritation ¹⁸⁵	Level 1B Two RCTs: 81.1-88.2% lice-free 14 days after 1 application ¹⁸⁵	Not approved by the FDA
Oral ivermectin (Stromectol)	Same as topical ivermectin above	200 µg/kg dosed once, or 400 µg/kg dosed twice at 7-day interval (not ovicidal) ¹⁰¹	Pruritus, headache	Level 1B RCT: 95.2% lice free by day 15 ¹⁰¹	Not approved by the FDA for the treatment of head lice; not for pregnant women or children weighing <15 kg ⁵⁹
Hot air delivered to the scalp	Causes louse death via overheating	30 min of heat applied to scalp (multiple delivery modalities studied)	None reported	Level 2B >88% effective in killing eggs at 14 days; variable efficacy in killing hatched lice ¹⁰⁹	Not approved by the FDA

Drug names are trademarks of their respective owners.

FDA, US Food and Drug Administration; RCT, randomized controlled trial.

*Level 1A, evidence from metaanalysis of randomized controlled trials; level 1B, evidence from ≥1 randomized controlled trial; level 2A, evidence from ≥1 controlled study without randomization; level 2B, evidence from ≥1 other type of experimental study; level 3, evidence from nonexperimental descriptive studies, such as comparative studies, correlation studies, and case-control studies; level 4, evidence from expert committee reports or opinions or clinical experience of respected authorities, or both.

response and potential promotion of permethrin resistance.⁵⁸

HUMAN HEAD LOUSE (*PEDICULUS HUMANUS CAPITIS*)

Key points

- Head lice are a public health problem affecting all demographics
- Head lice can be infected with pathogens
- Novel US Food and Drug Administration (FDA) –approved topical therapies include benzyl alcohol 5% lotion, spinosad 0.9% suspension, and ivermectin 0.5% lotion

Head lice are ovoid-shaped, 2- to 3-mm arthropods (Fig 3, A).^{59,60} Head lice are obligate human parasites that spend their entire life cycle on the scalp, feeding off blood every few hours.⁶¹ Female lice live ≤ 30 days and lay approximately 10 eggs daily.⁶¹ Nits are transparent, flask-shaped, 0.5-mm eggs found on hair shafts, typically 1 to 4 mm above the scalp, where warmth promotes survival (Fig 3, B).^{60,61}

Epidemiology

Head lice infest >100 million people worldwide and 6 to 12 million people in the United States annually.^{6,62} Transmission occurs via direct head-to-head contact, but lice can survive ≤ 4 days on fomites, including hairbrushes or headgear.⁶¹ Most cases occur in children, particularly females, likely because of cultural hair length differences.^{6,63} Head lice affect people of all socioeconomic statuses.

Clinical manifestations

Pruritus, papular urticaria, excoriations, and cervical/occipital lymphadenopathy can occur.^{60,64} Diagnosis is achieved via direct observation of lice or nits on hair shafts. Dermoscopy distinguishes eggs containing nymphs (Fig 3, C) from empty, translucent “pseudonymphs” (hair casts, hair product debris, or seborrheic dermatitis).⁶⁵⁻⁶⁸ Pruritus and disturbed sleep may cause school and work absences.⁶⁹ Severe iron deficiency anemia^{17,70,71} has been associated with heavy/chronic head lice infestation.

Implications

Head lice can carry and transmit *Staphylococcus aureus* and *Streptococcus pyogenes*.⁶¹ Recent evidence suggest head lice may also harbor other pathogens. *B recurrentis* DNA was identified in 23% of head lice from patients with LBRF in Ethiopia⁷² and the Republic of the Congo.⁷³ *B quintana* was

detected in head lice from France⁷⁴ (although it was not detected in another French study⁷⁵), Senegal (3-5% of lice in 2 studies),^{76,77} Ethiopia (9.2%),⁷⁸ San Francisco (25%),⁷⁹ Nepal,⁸⁰ and various African countries (2%).⁸¹ Investigators in Mali identified *Coxiella burnetti* and *Rickettsia aeschlimannii* in 5.1% and 0.6% of head lice, respectively.⁸² Pathogenic Acinetobacter species were identified in head lice from Thailand,⁸³ Algeria,⁸⁴ and 2 locations in France.^{75,85} Whether the presence of these organisms yields an increased risk of clinical disease remains unclear.

Management

Management of head lice requires eradication of all living lice and eggs. Mechanical removal is possible but labor intensive and is more efficacious when combined with medications.⁵⁹

Topical therapies are summarized in Table III.⁶⁰ Over-the-counter treatments include pyrethrins (neurotoxins derived from chrysanthemums), permethrin 1% lotion/cream (synthetic pyrethrin), and dimethicone (often spelled “dimeticone” outside the United States).⁵⁹ Topical pyrethrin⁸⁶⁻⁸⁹ and 1% permethrin resistance is widespread.⁹⁰⁻⁹⁴ Prescription options include malathion 0.5% lotion/gel, benzyl alcohol 5% lotion, spinosad 0.9% suspension, and ivermectin 0.5% lotion. Lindane is no longer recommended because of potential neurotoxicity.⁹⁵ Malathion was temporarily withdrawn from the US market because of flammability.⁹⁶ Ovicidal agents, including malathion and spinosad, are advantageous because they may not require repeat treatments.⁹⁷⁻⁹⁹ Persistent infestations (live lice observed 24 hours after treatment) should be retreated with a new medication class because resistance is likely.^{69,100} Oral ivermectin is effective for refractory head lice but is not approved by the FDA.¹⁰¹

Essential oils have demonstrated efficacy in eradicating head lice in many studies (Table IV).¹⁰²⁻¹⁰⁸ Most studies were in vitro, although combination Melaleuca and lavender oil, and also combination eucalyptus oil and *Leptospermum petersonii* solution, were effective in in vivo RCTs.^{106,108} No essential oils have been approved by the FDA for treating head lice, and many carry a risk of allergic contact dermatitis.⁶⁹

Conditioner should not be applied before topical medications and hair should not be rewashed for 1 to 2 days after removing the medication.⁹⁶ For patients deferring medications, delivering hot air to the scalp for 30 minutes showed up to 88% efficacy in killing lice.¹⁰⁹ Nit combs can be used every 2 to 3 days

Table IV. Efficacy of essential oils in treating head lice

Year	Compound	Study type	Instructions	Results
2007	Combined eucalyptus and peppermint in total 10% concentration, dissolved in 50% ethanol plus isopropanol in water	In vitro	N/A	Showed the greatest (93%) knockdown (lice death) 10 min after application ¹⁸⁶
2010	<i>Cinnamomum porphyrium</i> oil	In vitro	N/A	Killed >50% of lice within 2 min ¹⁰⁵
2010	Melaleuca oil and lavender oil in combination	In vivo	Applied 3 times at 7-day intervals	97.6% efficacy (louse-free) and superiority to pyrethrins at 24 hours ¹⁰⁶
2011	Citronellol and geraniol (both active components of 2 essential oils)	In vitro	N/A	>60% mortality of adult and late-stage nymphs ¹⁸⁷
2012	Tea tree (<i>Melaleuca</i>) oil, 1% concentration	In vitro	N/A	100% lice mortality at 30 min, showing both pediculicidal and ovicidal effects ¹⁰²
2016	Fumigant bioassays of leaves/fruits of <i>Schinus areira</i> (<i>Anacardiaceae</i>) and <i>Thymus vulgaris</i> (<i>Lamiaceae</i>)	In vitro	N/A	<i>S areira</i> derivatives were most toxic against adult lice (10 min to achieve 50% knockdown); <i>T vulgaris</i> was most ovicidal (0% hatching after 24 hours) ¹⁸⁸
2018	10% <i>Curcuma xanthorrhiza</i> and 10% <i>Eucalyptus globulus</i> oils	In vitro	N/A	5-min immersion halted egg hatching ¹⁰³
2018	Clove oil diluted in either coconut or sunflower oil	In vitro	N/A	90% lice mortality within 2 hours of a 30-min contact ¹⁰⁴
2018	Eucalyptus oil and <i>Leptospermum petersonii</i> solution	In vivo	Applied 3 times at 7-day intervals	More than twice as effective in curing lice than pyrethrin-containing mousse applied twice ¹⁰⁸
2018	Eucalyptus oil and <i>Leptospermum petersonii</i> solution	In vitro	N/A	Exposure in vitro killed 100% of lice and eggs ¹⁰⁸

after treatment to decrease the chance of reinfestation.⁹⁶ Along with scalp treatment, decontaminating clothing/linens at temperatures >50°C is recommended.¹¹⁰ Children should not be sent home from school early to treat infestation and should be allowed to return immediately after beginning treatment.⁵⁹ The American Academy of Pediatrics recommends against strict no-nit policies in schools.⁵⁹ All household members should be screened.

HUMAN PUBIC LOUSE (*PHTHIRUS PUBIS*)

Key points

- Pubic lice infestation can involve multiple body regions
- A diagnosis warrants screening for other sexually transmitted infections and, in children, considering the possibility of abuse

Pubic lice measure 0.8 to 1.2 mm in diameter and are wider than they are long, unlike ovoid-shaped head and body lice.⁶⁰ This crab-like shape enables them to grasp widely spaced pubic hairs (Fig 4, A). Female lice lay roughly 30 eggs during their 3- to 4-week lifespan.¹¹¹

Epidemiology

Pubic lice are transmitted via close physical contact, including sexual contact and shared sleeping arrangements.¹¹² Infestation occurs worldwide and affects both genders. Pubic hair removal via shaving, waxing, or laser has been associated with a decreased incidence.¹¹³

Clinical manifestations

P pubis infestation occurs on hairs of the scalp, axilla, chest, thighs, pubic area, eyebrows, and

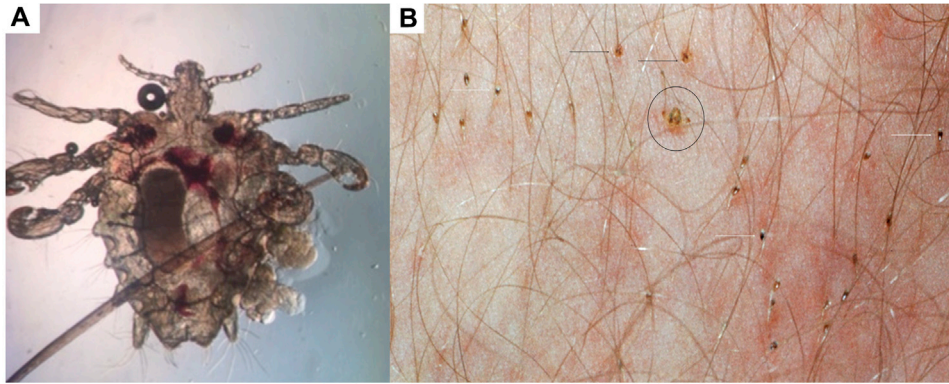


Fig 4. Pubic lice. **A**, Pubic louse morphology (courtesy of Charlotte Bernigaud, MD, Olivier Chosidow, MD, PhD, and Françoise Foulet, MD). **B**, Pubic louse (black circle) and numerous nits (several highlighted by white arrows) attached to hair shafts. Hemorrhagic macules (several highlighted by black arrows) at site of pubic lice bites (courtesy of Tim Berger, MD).

eyelashes. Symptomatic patients present with localized pruritus. Maculae ceruleae (blue-gray macules), red papules, and rust-colored feces can be seen at bite sites (Fig 4, B).^{60,114,115} In children, eyelashes are commonly involved^{112,116-118} and coinfection with head lice has been reported.¹¹⁸ Diagnosis is achieved by visualizing lice or nits on hair shafts (Fig 4, B). Bacterial superinfection of excoriations may occur. Pubic lice are not known to transmit other infections.

Management

A diagnosis of pubic lice warrants screening for other sexually transmitted infections.¹¹¹ While pediatric pubic lice infestation can occur through shared sleeping arrangements with infested individuals or contact with fomites,¹¹⁷ abuse should be considered.^{112,118}

The first-line treatment of pubic lice is topical 1% permethrin.^{111,114} Topical 0.5% ivermectin lotion is also approved by the FDA.¹¹¹ Petrolatum jelly is useful for treating eyelash infestation.¹¹⁹ Oral ivermectin is a second-line therapy but has not been approved by the FDA.^{114,120} Clean clothing should be worn when starting treatment.¹¹¹ Shaving is therapeutic and decreases the likelihood of recurrence. All clothing/linens used in the preceding 3 days should be washed and dried at temperatures >50°C.¹²¹

TUNGIASIS

Key points

- Tungiasis is an infestation, endemic to tropical world regions, caused by a flea burrowing into the skin

- Tungiasis is seen in travelers returning from endemic areas
- Tungiasis is associated with not wearing closed-toe footwear and dirt floors

Tungiasis is an ectoparasitic disease caused by skin penetration by the female *Tunga penetrans* or, less commonly, *Tunga trimamillata* flea.¹²² It is endemic in Latin America, the Caribbean, and sub-Saharan Africa¹²³ and occurs in travelers returning from endemic regions.¹²⁴⁻¹³³

Epidemiology

In travelers to endemic areas, tungiasis is usually acquired from walking barefoot or with open-toed shoes. In returning travelers between 2007 and 2011, 87 cases were reported in GeoSentinel, the largest global surveillance network for travel-related morbidity.¹³³ The top exposure countries were Brazil, Madagascar, Uganda, and Ethiopia. In returning travelers from Brazil who visited a GeoSentinel clinic between 1997 and 2013, 35 cases (2% of 1586) were diagnosed with tungiasis.¹³² At a German travel clinic, most returning travelers with tungiasis had been exposed in Africa or Latin America.¹³⁴

In endemic areas, the prevalence of tungiasis ranges from 19.1% to 58.7% (Table V).¹³⁵⁻¹⁴¹ In these settings, tungiasis is linked to inadequate health education,^{135,140} the lack of regular closed-toe footwear use,^{137,141,142} and poor housing conditions,^{135,140} including dirt floors or unclean floors,^{137,138,142} crowded living spaces,¹⁴⁰ and sleeping on the ground outside.¹⁴² Causative fleas may also exist in domestic or wild animal reservoirs, and therefore tungiasis is considered a zoonosis.¹⁴³⁻¹⁴⁵ Living on compounds with animals

Table V. Tungiasis prevalence and risk factors in endemic settings

Year	Location	Population	Prevalence of tungiasis	Risk factors for infestation
2006	Rural Northeast Brazil ¹³⁵	496 individuals in 132 households	51.0%	Poor housing conditions (OR = 4.7), lack of health education (OR = 4.1), and the presence of animals on the compound (OR = 1.9)
2006	Haiti (4 regions) ¹⁸⁹	383 patients	31.1%	Not studied
2007	Rural Western Nigeria ¹⁴²	643 individuals	42.5%	Presence of pigs (aOR = 17.98), sand or clay floor inside houses (aOR = 9.33), common resting place outside (aOR = 7.14), and no regular use of closed footwear (51% pop AR)
2012	Rural Western Tanzania ¹³⁶	586 individuals	42.5%	Age \geq 45 years (OR = 3.71)
2015	Murang'a County, Kenya ¹³⁷	508 children	19.1%	Classrooms with dusty floors (aOR = 14.657), earthen mud walled houses (aOR = 13.78), and not regularly using closed footwear (aOR = 10.45)
2015	Maguye District, Eastern Uganda ¹³⁸	422 households in 12 villages	22.5%	Cracked house floor (aOR = 6.28), dirty feet (aOR = 3.86), dirty clothes (aOR = 3.46), dirty floor (aOR = 3.21), littered compounds (aOR = 2.95), and rearing cattle (aOR = 2.38)
2017	Southwest Nigeria ¹³⁹	545 children	24.4%	Not studied
2017	Kilifi County, Kenya ¹⁴⁰	1086 individuals from 233 households in 8 villages	25.0%	Only mud puddles as a washing source (OR = 25.48), washing the body without soap (OR = 7.36), mud walls (OR = 3.35), lack of water, permitting washing only once daily (OR = 2.23), number of people sleeping per room (OR = 1.77), and sleeping on the floor (OR = 1.68)
2017	Yirgacheffe, Southern Ethiopia ¹⁵⁶	343 children	34.7%	Closed footwear associated with increased lesion number
2018	Wensho District, Southern Ethiopia ¹⁴¹	366 children	58.7%	Never using footwear (aOR = 12.55), occasionally using footwear (aOR = 7.42), cat-owning household (aOR = 4.95), illiterate mothers (aOR = 3.62), and mothers who have attended only primary school education (aOR = 2.22)

aOR, Adjusted odds ratio; OR, odds ratio; pAR, population attributable risk.

confers a higher risk.^{135,138,141} Leading animal reservoirs differ by region, including stray cats/dogs in urban Brazil,¹⁴⁶ domestic cats in southern Ethiopia,¹⁴¹ and domesticated pigs in Uganda and Nigeria.^{142,147}

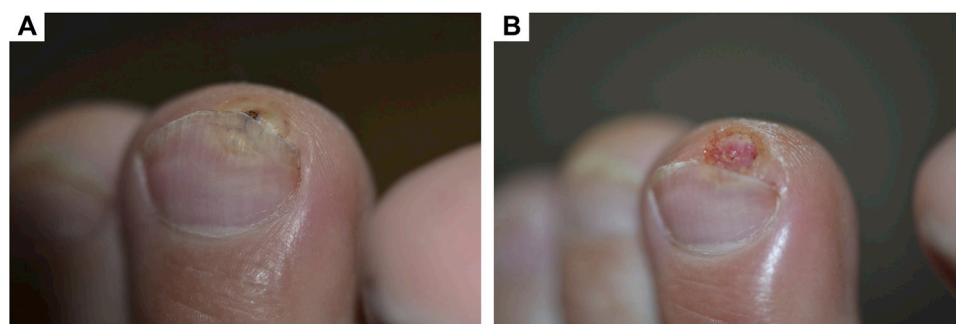
Clinical manifestations

Lesions predominantly affect the feet. Skin findings reflect the lesion's clinical stage, which is linked to the life cycle of the embedded flea and correlates with disease severity according to the Fortaleza classification (Table VI).¹⁴⁸ After painlessly burrowing into the

skin, typically on the feet, embedded fleas mature for several weeks.¹²³ An early-stage lesion is a 1-mm red-brown macule that evolves into a nodule with a central dark punctum (Fig 5, A). Subsequent flea engorgement from egg production leads to swelling, erythema, pruritus, and pain.¹²³ Eventually, egg release and parasite death trigger severe inflammation, leading to a black crusted papule that heals with a punched-out scar (Fig 5, B). Dermoscopy can be useful in diagnosing specific features, which in 1 series included dark central pores, whitish oval structures, silver dendritic fibers, and blue-black

Table VI. Fortaleza classification of the clinical stages of tungiasis

Stage	Duration	Parasite findings	Clinical findings	Histopathology findings
I	3-7 hours	Penetration at angle of 45°-90°; abdominal segments 2 and 3 begin separating	Itchy or painful red-brown 1-mm macule	Epidermis: hyperplasia, hyperkeratosis, parakeratosis Dermis: mild inflammatory infiltrate of neutrophils and eosinophils
II	1-2 days	Hypertrophic zone between abdominal segments 2 and 3	Pearly white nodule with surrounding erythema and a central dark punctum	Epidermis: hyperplasia, hyperkeratosis, parakeratosis, spongiosis, neutrophilic infiltrate, and intracorneal microabscesses Dermis: perivascular inflammatory infiltrate of neutrophils, lymphocytes, eosinophil, and plasma cells and mast cells
IIIa	2-3 days	Head of parasite at dermoepidermal interface	Growing, yellow-white halo around black dot. Fecal coils visible. Pain, foreign body sensation, severe itching	Epidermis: marked hyperplasia and hyperkeratosis, reactive pseudoepitheliomatosis; marked mixed inflammatory infiltrate Dermis: mixed inflammatory infiltrate
IIIb	1-2 weeks	Thickening of chitin exoskeleton	Caldera formation; soft consistency; severe pain	Epidermis: same as above Dermis: dilated vessels and neutrophils
IV	3-5 weeks	Flea dying or dead	Crusted black lesion (containing dead parasite) with or without superinfection	Epidermis: same as above, plus vascularization of stratum corneum Dermis: moderate to severe mixed infiltrate
V	6 weeks to several months	No parasite	Residual scar, punched out depression; nail dystrophy or loss; lymphedema	Epidermis: hyperplasia, thickening and blunting of epidermal ridges, and thickened stratum spinosum Dermis: residual mild inflammation

Adapted from Eisele et al.¹⁴⁸**Fig 5.** Tungiasis. **A**, Early clinical stage of tungiasis with embedded flea (courtesy of Jorg Heukelbach, MD, PhD). **B**, Immediate aftermath of tungiasis infestation after death and expulsion of an embedded flea (courtesy of Jorg Heukelbach, MD, PhD).

blotches in most cases.¹⁴⁹ The penetration site measures $\leq 500 \mu\text{m}$ in diameter,¹⁵⁰ and therefore secondary bacterial infection occurs frequently, including cellulitis and necrotizing skin and soft tissue infection.^{150,151} In

returning travelers, the differential diagnosis depends on the clinical stage and includes arthropod bite, wart, pyogenic granuloma, abscess, leishmaniasis, myxoid cyst, myiasis, and foreign body.¹⁵²

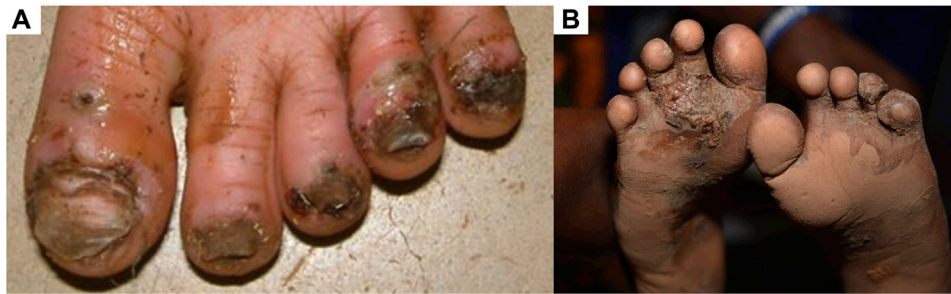


Fig 6. Tungiasis. **A**, Multiple embedded fleas on the dorsal surfaces of the toes in a person living in an endemic area (courtesy of the Regional Dermatology Training Centre, Moshi, Tanzania). **B**, Multiple embedded fleas on the plantar surface of the foot in a person living in an endemic area (courtesy of Jean Marie Rukanikigitero, MD).

Quality of life

Tungiasis-associated morbidity in endemic settings is caused by severe foot pain, progressive foot mutilation, and nail dystrophy, complicating walking and contributing to stigmatization.^{153,154} Children may experience teasing and have higher school absenteeism and lower school performance than unaffected peers.¹⁵⁵ Tungiasis negatively impacts children's quality of life,^{156,157} with disturbed sleep and concentration reported most frequently in 1 Kenyan study.¹⁵⁷

Treatment

Tungiasis is self-limited because the organism typically dies within 6 six weeks after penetration.¹⁴⁸ Treatment aims to reduce symptom severity and prevent secondary infection. Surgical removal of the organism is crucial. This can be achieved through shave or punch biopsy procedures. Early on, sterile needles can also be used before multiple embedded fleas or extensive inflammation occur.¹⁵⁸ Secondary bacterial infection requires appropriate antibiotics.

In endemic settings without access to appropriate equipment for surgical removal, commonplace sharp instruments, such as pins, needles, thorns, and sharpened wood pieces, are frequently reused by different people.¹⁵⁵ This can lead to bloodborne pathogen transmission,¹⁵⁵ tetanus,^{159,160} and life-threatening complications of secondary infection, including necrotizing fasciitis, gangrene, and sepsis.¹⁶⁰⁻¹⁶² As such, nonsurgical alternatives have been investigated. Topical Zanzarin, derived from coconut oil, jojoba oil, and aloe vera, decreases tungiasis incidence and morbidity^{163,164} but is no longer commercially available. Topical dimethicone has been shown to reduce inflammation and hasten parasite death in 1 RCT,¹⁶⁵ with targeted application to areas of parasite protrusion being more effective

than to the entire foot.¹⁶⁶ Oral ivermectin showed no efficacy in a RCT.¹⁶⁷

Screening and prevention

For prospective travelers to endemic areas, the best preventive measure is wearing closed-toe shoes.¹⁶⁸ Only 54% of returning travelers diagnosed with tungiasis reported a pretravel medical visit,¹³³ and even when a visit occurs tungiasis may not be discussed.¹³³ Clinicians should therefore counsel patients with relevant planned travel.

In endemic areas, where nearly 95% of lesions are restricted to the feet,¹⁶⁹ targeted evaluation of periungual feet is useful for estimating prevalence, severity, and identifying persons needing treatment.¹⁷⁰ While improving education and health care access are important, clinical knowledge does not necessarily translate into effective prevention and treatment.¹⁷¹ Large-scale provision of closed-toe footwear is essential. In a metaanalysis, footwear use was associated with significantly lower odds of acquiring tungiasis.¹⁷² However, regular shoe replacement is costly¹⁴³ and may not reduce exposure inside houses, where shoes are not typically worn.¹⁴³ As such, modifications are important, including switching to sealed cement floors and cleaning floors daily.¹⁶² Unfortunately, human hosts living in endemic areas are frequently reinfested unless proper footwear and housing changes are widely implemented.¹⁶² Finally, increasing tetanus vaccination coverage in tungiasis-endemic regions will help prevent secondary tetanus.¹⁷³

In conclusion, in the context of a global refugee crisis and homelessness epidemic, diagnosing and managing lice infestations are important dermatologic skills. Dermatologists should know that careful clothing inspection is key to diagnosing body lice and that head lice have widespread

resistance to topical pyrethrin and permethrin. Tungiasis is an infestation affecting the feet that has substantial negative impact on communities in endemic regions. It is seen in returning travelers and can be prevented by wearing closed-toe shoes.

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REFERENCES

- Rivet R, McCoy KD, Brouqui P, Raoult D. Evidence that head and body lice on homeless persons have the same genotype. *PLoS One*. 2012;7:e45903.
- Badiaga S, Brouqui P. Human louse-transmitted infectious diseases. *Clin Microbiol Infect*. 2012;18:332-337.
- Centers for Disease Control and Prevention. Body lice. Available at: <https://www.cdc.gov/parasites/lice/body/index.html>. Accessed November 3, 2018.
- Brouqui P. Arthropod-borne diseases associated with political and social disorder. *Annu Rev Entomol*. 2011;56:357-374.
- Sweilieh WM. Global output of research on epidermal parasitic skin diseases from 1967 to 2017. *Infect Dis Poverty*. 2018;7:74.
- Chosidow O. Scabies and Pediculosis. *Lancet*. 2000;355:819-826.
- Gravinatti M, Faccini-Martinez A, Ruys S, Timenetsky J, Biondo A. Preliminary report of body lice infesting homeless people in Brazil. *Rev Inst Med Trop Sao Paulo*. 2018;60:e9.
- Bonilla DL, Cole-Porse C, Kjemtrup A, Osikowicz L, Kosoy M. Risk factors for human lice and bartonellosis among the homeless, San Francisco, CA, USA. *Emerg Infect Dis*. 2014;20:1645-1651.
- Arnaud A, Chosidow O, Detrez M, et al. Prevalences of scabies and pediculosis corporis among homeless people in the Paris region: results from two randomized cross-sectional surveys (HYTPEAC study). *Br J Dermatol*. 2016;174:104-112.
- Calloix C, Badiaga S, Raoult D, Tissot-dupont H, Brouqui P, Gautret P. Changing demographics and prevalence of body lice among homeless persons, Marseille, France. *Emerg Infect Dis*. 2017;23:1894-1897.
- Fazel S, Khosla V, Doll H, Geddes J. The prevalence of mental disorders among the homeless in Western countries: systematic review and meta-regression analysis. *PLoS Med*. 2008;5:e225.
- Fazel S, Geddes J, Kushel M. The health of homeless people in high-income countries: descriptive epidemiology, health consequences, and clinical and policy recommendations. *Lancet*. 2014;384:1529-1540.
- Gee A. "Human tragedy": LA homelessness jumps to record-breaking level. Available at: <https://www.theguardian.com/us-news/2017/may/31/homelessness-count-los-angeles-record-level>. Accessed February 21, 2019.
- Henry M, Watt R, Rosenthal L, Shivji A. The 2017 Annual Homeless Assessment Report (AHAR) to Congress, part 1: point-in-time estimates of homelessness. Available at: <https://www.hudexchange.info/resources/documents/2017-AHAR-Part-1.pdf>. Accessed February 21, 2019.
- Smith N. Homelessness is a tragedy the U.S. can afford to fix. Bloomberg. Available at: <https://www.bloomberg.com/view/articles/2018-05-21/ending-homelessness-is-a-job-for-the-federal-government>. Accessed June 1, 2018.
- Feantsa and Abbe Pierre Foundation. Third overview of housing exclusion in Europe 2018. Available at: <https://www.feantsa.org/en/report/2018/03/21/the-second-overview-of-housing-exclusion-in-europe-2017>. Accessed February 21, 2019.
- Guss DA, Koenig M, Castillo EM. Severe iron deficiency anemia and lice infestation. *J Emerg Med*. 2011;41:362-365.
- Nara A, Nagai H, Yamaguchi R, et al. An unusual autopsy case of lethal hypothermia exacerbated by body lice-induced severe anemia. *Int J Leg Med*. 2016;130:765-769.
- Woodruff C, Chang A. More than skin deep: severe iron deficiency anemia and eosinophilia associated with pediculosis capitis and corporis infestation. *JAAD Case Rep*. 2019;5:444-447.
- Ruiz J. *Bartonella quintana*, past, present, and future of the scourge of World War I. *APMIS*. 2018;126:831-837.
- Anstead GM. The centenary of the discovery of trench fever, an emerging infectious disease of World War I. *Lancet Infect Dis*. 2016;16:e164-e172.
- Raoult D, Ndiokubwayo J, Tissot-Dupont H, et al. Outbreak of epidemic typhus associated with trench fever in Burundi. *Lancet*. 1998;352:353-358.
- Spach D, Kanter A, Dougherty M, et al. *Bartonella (Rochalimaea) quintana* bacteremia in inner-city patients with chronic alcoholism. *N Engl J Med*. 1995;332:424-428.
- Seki N, Sasaki T, Sawabe K, et al. Epidemiological studies on *Bartonella quintana* infections among homeless people in Tokyo, Japan. *Jpn J Infect Dis*. 2006;59:31-35.
- Ohl ME, Spach DH. *Bartonella quintana* and urban trench fever. *Clin Infect Dis*. 2000;31:131-135.
- Drali R, Sangare A, Boutellis A, et al. *Bartonella quintana* in body lice from scalp hair of homeless persons, France. *Emerg Infect Dis*. 2014;20:907-908.
- Faccini-Martinez AA, Márquez AC, Bravo-Estupiñan DM, et al. *Bartonella quintana* and typhus group Rickettsiae exposure among homeless persons, Bogota, Colombia. *Emerg Infect Dis*. 2017;23:1876-1879.
- Louni M, Mana N, Bitam I, et al. Body lice of homeless people reveal the presence of several emerging bacterial pathogens in northern Algeria. *PLoS Negl Trop Dis*. 2018;12:e0006397.
- Ulutasdemir N, Eroglu F, Tanrıverdi M, Daglı El, Koltas IS. The epidemic typhus and trench fever are risk for public health due to increased migration in southeast of Turkey. *Acta Trop*. 2018;178:115-118.
- Angelakis E, Raoult D. Pathogenicity and treatment of *Bartonella* infections. *Int J Antimicrob Agents*. 2014;44:16-25.
- Moulin C, Kanitakis J, Ranchin B, et al. Cutaneous bacillary angiomatosis in renal transplant recipients: report of three new cases and literature review. *Transpl Infect Dis*. 2012;14:403-409.
- Orsag J, Flodr P, Melter O, et al. Cutaneous bacillary angiomatosis due to *Bartonella quintana* in a renal transplant recipient. *Transpl Int*. 2015;28:626-631.
- Scola BLA, Raoult D. Culture of *Bartonella quintana* and *Bartonella henselae* from human samples: a 5-year experience (1993 to 1998). *J Clin Microbiol*. 1999;37:1899-1905.
- Koehler J, Tappero J. Bacillary angiomatosis and bacillary peliosis in patients infected with human immunodeficiency virus. *Clin Infect Dis*. 1993;17:612.
- Ramos JM, Malmierca E, Reyes F, Tesfamariam A. Louse-borne relapsing fever in Ethiopian children: experience of a rural hospital. *Trop Doct*. 2009;39:34-36.

36. Nordmann T, Feldt T, Bosselmann M, et al. Outbreak of louse-borne relapsing fever among urban dwellers in Arsi Zone, central Ethiopia, from July to November 2016. *Am J Trop Med Hyg.* 2018;98:1599-1602.
37. Osthoff M, Schibli A, Fadini D, Lardelli P, Goldenberger D. Louse-borne relapsing fever - report of four cases in Switzerland, June-December 2015. *BMC Infect Dis.* 2016;16:210.
38. Hoch M, Wieser A, Loscher T, et al. Louse-borne relapsing fever (*Borrelia recurrentis*) diagnosed in 15 refugees from northeast Africa: epidemiology and preventive control measures, Bavaria, Germany, July to October 2015. *Euro Surveill.* 2015;20:42.
39. Wilting K, Stienstra Y, Sinha B, Braks M, Cornish D, Grundmann H. Louse-borne relapsing fever (*Borrelia recurrentis*) in asylum seekers from Eritrea, the Netherlands, July 2015. *Euro Surveill.* 2015;20:21196.
40. Goldenberger D, Claas G, Bloch-Infanger C, et al. Louse-borne relapsing fever (*Borrelia recurrentis*) in an Eritrean refugee arriving in Switzerland, August 2015. *Euro Surveill.* 2015;20:2-5.
41. Antinori S, Mediannikov O, Corbellino M, Raoult D. Louse-borne relapsing fever among East African refugees in Europe. *Trav Med Infect Dis.* 2016;14:110-114.
42. Colomba C, Scarlata F, Di Carlo P, et al. Fourth case of louse-borne relapsing fever in Young Migrant, Sicily, Italy, December 2015. Mini Review Article. *Public Health.* 2016;139:22-26.
43. Darcis G, Hayette MP, Bontems S, et al. Louse-borne relapsing fever in a refugee from Somalia arriving in Belgium. *J Trav Med.* 2016;23. <https://doi.org/10.1093/jtm/taw009>.
44. Hytönen J, Khawaja T, Grönroos JO, Jalava A, Meri S, Oksi J. Louse-borne relapsing fever in Finland in two asylum seekers from Somalia. *APMIS.* 2017;125:59-62.
45. Antinori S, Tonello C, Edouard S, et al. Diagnosis of louse-borne relapsing fever despite negative microscopy in two asylum seekers from Eastern Africa. *Am J Trop Med Hyg.* 2017;97:1669-1672.
46. Isenring E, Fehr J, Gültekin N, Schlagenhauf P. Infectious disease profiles of Syrian and Eritrean migrants presenting in Europe: a systematic review. *Trav Med Infect Dis.* 2018;25:65-76.
47. European Centre for Disease Prevention and Control. Facts about louse-born relapsing fever. Available at: <https://ecdc.europa.eu/en/louse-borne-relapsing-fever/facts>. Accessed February 21, 2019.
48. Halperin T, Orr N, Cohen R, et al. Detection of relapsing fever in human blood samples from Israel using PCR targeting the glycerophosphodiester phosphodiesterase (GlpQ) gene. *Acta Trop.* 2006;98:189-195.
49. Guerrier G, Doherty T. Comparison of antibiotic regimens for treating louse-borne relapsing fever: a meta-analysis. *Trans R Soc Trop Med Hyg.* 2011;105:483-490.
50. Barbour A. Clinical features, diagnosis, and management of relapsing fever. Up to Date. Available at: https://www.uptodate.com/contents/clinical-features-diagnosis-and-management-of-relapsing-fever?search=relapsing%20fever&source=search_result&selectedTitle=1~41&usage_type=default&display_rank=1. Accessed February 21, 2019.
51. Negussie Y, Remick D, DeForge L, Kunkel S, Eynon A, Griffin G. Detection of plasma tumor necrosis factor, interleukins 6, and 8 during the Jarisch-Herxheimer reaction of relapsing fever. *J Exp Med.* 1992;175:1207-1212.
52. Butler T. The Jarisch-Herxheimer reaction after antibiotic treatment of spirochetal infections: a review of recent cases and our understanding of pathogenesis. *Am J Trop Med Hyg.* 2017;96:46-52.
53. Tarasevich I, Rydkina E, Raoult D. Outbreak of epidemic typhus in Russia. *Lancet.* 1998;352:1151.
54. Centers for Disease Control and Prevention. Epidemic typhus. Available at: <https://www.cdc.gov/typhus/epidemic/index.html>. Accessed March 30, 2019.
55. Bechah Y, Capo C, Mege J, Raoult D. Epidemic typhus. *Lancet Infect Dis.* 2008;8:417-426.
56. Johns Hopkins Bloomberg School of Public Health. *Rickettsia prowazekii* (epidemic typhus). Johns Hopkins Center for Health Security. Available at: <http://www.centerforhealthsecurity.org/resources/fact-sheets/pdfs/typhus.pdf>. Accessed December 9, 2018.
57. Sundnes KO, Haimanot AT. Epidemic of louse-borne relapsing fever in Ethiopia. *Lancet.* 1993;342:1213-1215.
58. Benkouiten S, Drali R, Badiaga S, et al. Effect of permethrin-impregnated underwear on body lice in sheltered homeless persons: a randomized controlled trial. *JAMA Dermatol.* 2014;150:273-279.
59. Devore CD, Schutze GE. Head Lice. *Pediatrics.* 2015;135:e1355-e1365.
60. Ko CJ, Elston DM. Pediculosis. *J Am Acad Dermatol.* 2004;50:1-14.
61. Burkhart CN, Burkhart CG. Fomite transmission in head lice. *J Am Acad Dermatol.* 2007;56:1044-1047.
62. Do-Pham G, Monsel G, Chosidow O. Lice. *Semin Cutan Med Surg.* 2014;33:116-118.
63. Tagka A, Lambrou GI, Braoudaki M, Panagiotopoulos T, Papanikolaou E, Laggas D. Socioeconomic factors associated with pediculosis (Phthiraptera: Pediculidae) in Athens, Greece. *J Med Entomol.* 2016;53:919-922.
64. Dagrosa AT, Elston DM. What's eating you? Head lice (*Pediculus humanus capitis*). *Cutis.* 2017;100:389-392.
65. Stefani A, Hofmann-Wellenhof R, Zalaudek I. Dermoscopy for diagnosis and treatment monitoring of pediculosis capitis. *J Am Acad Dermatol.* 2006;54:909-911.
66. Bakos R, Bakos L. Dermoscopy for diagnosis of pediculosis capitis. *J Am Acad Dermatol.* 2007;57:727-728.
67. Badri T, Hammami H, Benmously R, Mokhtar I, Fenniche S. Dermoscopic diagnosis of pediculosis capitis. *Acta Dermatovenerol Alp Pannonica Adriat.* 2010;19:45-46.
68. Haliasos EC, Kerner M, Jaimes-Lopez N, et al. Dermoscopy for the pediatric dermatologist part I: dermoscopy of pediatric infectious and inflammatory skin lesions and hair disorders. *Pediatr Dermatol.* 2013;30:163-171.
69. Koch E, Clark JM, Cohen B, et al. Management of head louse infestations in the United States—a literature review. *Pediatr Dermatol.* 2016;33:466-472.
70. Althomali S, Alzubaidi L, Alkhaldi D. Severe iron deficiency anaemia associated with heavy lice infestation in a young woman. *BMJ Case Rep.* 2015. bcr2015212207.
71. Burke S, Mir P. Pediculosis causing iron deficiency anaemia in school children. *Arch Dis Child.* 2011;96:989.
72. Boutellis A, Mediannikov O, Bilcha KD, et al. *Borrelia recurrentis* in Head Lice, Ethiopia. *Emerg Infect Dis.* 2013;19:796-798.
73. Amanzougaghene N, Akiana J, Mongo Ndombe G, et al. Head lice of Pygmies reveal the presence of relapsing fever *Borreliae* in the Republic of Congo. *PLoS Negl Trop Dis.* 2016;10:e0005142.
74. Angelakis E, Rolain JM, Raoult D, Brouqui P. *Bartonella quintana* in head louse nits. *FEMS Immunol Med Microbiol.* 2011;62:244-246.

75. Bouvresse S, Socolovski C, Berdjane Z, et al. No evidence of *Bartonella quintana* but detection of *Acinetobacter baumannii* in head lice from elementary schoolchildren in Paris. *Comp Immunol Microbiol Infect Dis*. 2011;34:475-477.
76. Diatta G, Mediannikov O, Sokhna C, et al. Short report: Prevalence of *Bartonella quintana* in patients with fever and head lice from rural areas of Sine-Saloum, Senegal. *Am J Trop Med Hyg*. 2014;91:291-293.
77. Boutellis A, Veracx A, Angelakis E, et al. *Bartonella quintana* in Head Lice from Sénégal. *Vector Borne Zoonotic Dis*. 2012;12:564-567.
78. Cutler S, Abdissa A, Adamu H, Tolosa T, Gashaw A. *Bartonella quintana* in Ethiopian lice. *Comp Immunol Microbiol Infect Dis*. 2012;35:17-21.
79. Bonilla DL, Kabeya H, Henn J, Kramer VL, Kosoy MY. *Bartonella quintana* in body lice and head lice from homeless persons, San Francisco, California, USA. *Emerg Infect Dis*. 2009;15:912-915.
80. Sesaki T, Poudel S, Iswaka H, et al. First molecular evidence of *Bartonella quintana* in *Pediculus humanus capitis* (Phthiraptera: Pediculidae), collected from Nepalese children. *J Med Entomol*. 2006;43:110-112.
81. Sangaré AK, Boutellis A, Drali R, et al. Detection of *Bartonella quintana* in African body and head lice. *Am J Trop Med Hyg*. 2014;91:294-301.
82. Amanzougaghene N, Fenollar F, Sangare AK, et al. Detection of bacterial pathogens including potential new species in human head lice from Mali. *PLoS One*. 2017;12:e0184621.
83. Sunantaraporn S, Sanprasert V, Pengsakul T, et al. Molecular survey of the head louse *Pediculus humanus capitis* in Thailand and its potential role for transmitting *Acinetobacter spp*. *Parasit Vectors*. 2015;8:127.
84. Mana N, Louni M, Parola P, Bitam I. Human head lice and pubic lice reveal the presence of several *Acinetobacter* species in Algiers, Algeria. *Comp Immunol Microbiol Infect Dis*. 2017;53:33-39.
85. Candy K, Amanzougaghene N, Izri A, et al. Molecular survey of head and body lice, *Pediculus humanus*, in France. *Vector Borne Zoonotic Dis*. 2018;18:243-251.
86. Downs AMR, Stafford KA, Hunt LP, Ravenscroft JC, Coles GC. Widespread insecticide resistance in head lice to the over-the-counter pediculocides in England, and the emergence of carbaryl resistance. *Br J Dermatol*. 2002;146:88-93.
87. Cueto G, Zerba E, Picollo M. Evidence of pyrethroid resistance in eggs of *Pediculus humanus capitis* (Phthiraptera: Pediculidae) from Argentina. *J Med Entomol*. 2008;45:693-697.
88. Durand R, Bouvresse S, Andriantsoanirina V, Berdjane Z, Chosidow O, Izri A. High frequency of mutations associated with head lice pyrethroid resistance in schoolchildren from Bobigny, France. *J Med Entomol*. 2011;48:74-75.
89. Marcoux D, Palma KG, Kaul N, et al. Pyrethroid pediculicide resistance of head lice in Canada evaluated by serial invasive signal amplification reaction. *J Cutan Med Surg*. 2010;14:115-118.
90. Mumcuoglu K, Hemingway J, Miller J, et al. Permethrin resistance in the head louse *Pediculus capitis* from Israel. *Med Vet Entomol*. 1995;9:427-432.
91. Rupes V, Moravec J, Chmela J, Ledvinka J, Zelenkova J. A resistance of head lice (*Pediculus capitis*) to permethrin in Czech Republic. *Cent Eur J Public Health*. 1995;3:30-32.
92. Pollack R, Kiszewski A, Armstrong P, et al. Differential permethrin susceptibility of head lice sampled in the United States and Borneo. *Arch Pediatr Adolesc Med*. 1999;153:969-973.
93. Yoon KS, Gao J, Lee SH, Clark JM. Permethrin-resistant human head lice, *Pediculus capitis*, and their treatment. *Arch Dermatol*. 2003;139:994-1000.
94. Bouvresse S, Berdjane Z, Durand R, Bouscaillou J, Izri A, Chosidow O. Permethrin and malathion resistance in head lice: results of ex vivo and molecular assays. *J Am Acad Dermatol*. 2012;67:1143-1150.
95. Centers for Disease Control and Prevention. Parasites: lice. Available at: <https://www.cdc.gov/parasites/lice/head/index.html>. Accessed June 17, 2018.
96. Centers for Disease Control and Prevention. Head lice: treatment. Available at: <https://www.cdc.gov/parasites/lice/head/treatment.html>. Accessed November 21, 2018.
97. Stough D, Shellabarger S, Quiring J, Gabrielsen AA. Efficacy and safety of spinosad and permethrin creme rinses for *Pediculosis capitis* (head lice). *Pediatrics*. 2009;124:e389-e395.
98. McCormack PL. Spinosad in pediculosis capitis. *Am J Clin Dermatol*. 2011;12:349-353.
99. Deeks LS, Naunton M, Currie MJ, Bowden FJ. Topical ivermectin 0.5% lotion for treatment of head lice. *Ann Pharmacother*. 2013;47:1161-1167.
100. Chosidow O, Giraudeau B. Topical ivermectin — a step toward making head lice dead lice? *N Engl J Med*. 2012;367:1750-1752.
101. Chosidow O, Giraudeau B, Cottrell J, et al. Oral ivermectin versus malathion lotion for difficult-to-treat head lice. *N Engl J Med*. 2010;362:896-905.
102. Di Campli E, Di Bartolomeo S, Delli Pizzi P, et al. Activity of tea tree oil and nerolidol alone or in combination against *Pediculus capitis* (head lice) and its eggs. *Parasitol Res*. 2012;111:1985-1992.
103. Soonwera M, Wongnet O, Sittichok S. Ovicidal effect of essential oils from Zingiberaceae plants and *Eucalyptus globulus* on eggs of head lice, *Pediculus humanus capitis* De Geer. *Phytomedicine*. 2018;47:93-104.
104. Candy K, Nicolas P, Andriantsoanirina V, Izri A, Durand R. In vitro efficacy of five essential oils against *Pediculus humanus capitis*. *Parasitol Res*. 2018;117:603-609.
105. Toloza AC, Zygadlo J, Biurrun F, Rotman A, Picollo MI. Bioactivity of Argentinean essential oils against permethrin-resistant head lice, *Pediculus humanus capitis*. *J Insect Sci*. 2010;10:185.
106. Barker SC, Altman PM. A randomised, assessor blind, parallel group comparative efficacy trial of three products for the treatment of head lice in children - melaleuca oil and lavender oil, pyrethrins and piperonyl butoxide, and a "suffocation" product. *BMC Dermatol*. 2010;10:6.
107. Barker SC, Altman PM. An ex vivo, assessor blind, randomised, parallel group, comparative efficacy trial of the ovicidal activity of three pediculicides after a single application—melaleuca oil and lavender oil, eucalyptus oil and lemon tea tree oil, and a "suffocation" pediculicide. *BMC Dermatol*. 2011;11:14.
108. Greive KA, Barnes TM. The efficacy of Australian essential oils for the treatment of head lice infestation in children: a randomised controlled trial. *Australas J Dermatol*. 2018;59:e99-e105.
109. Goates BM, Atkin JS, Wilding KG, et al. An effective nonchemical treatment for head lice: a lot of hot air. *Pediatrics*. 2006;118:1962-1970.
110. Izri A, Chosidow O. Efficacy of machine laundering to eradicate head lice: recommendations to decontaminate

- washable clothes, linens, and fomites. *Clin Infect Dis*. 2006;42:e9-e10.
111. Centers for Disease Control and Prevention. Pubic "crab" lice. Available at: <https://www.cdc.gov/parasites/lice/pubic/index.html>. Accessed November 3, 2018.
 112. Klaus S, Shvil Y, Mumcuoglu KY. Generalized infestation of a 3 1/2-year-old girl with the pubic louse. *Pediatr Dermatol*. 1994; 11:26-28.
 113. Dholakia S, Buckler J, Jeans JP, Pillai A, Eagles N, Dholakia S. Pubic lice: an endangered species? *Sex Transm Dis*. 2014;41: 388-391.
 114. Salavastru CM, Chosidow O, Janier M, Tiplica GS. European guideline for the management of pediculosis pubis. *J Eur Acad Dermatol Venereol*. 2017;31:1425-1428.
 115. Brown TJ, Yen-Moore A, Tyring SK. An overview of sexually transmitted diseases. Part II. *J Am Acad Dermatol*. 1999;41: 661-677.
 116. Michali G, Lacarrubba F. Phthiriasis palpebrarum in a child. *N Engl J Med*. 2015;373:e35.
 117. Yi JW, Li L, Luo DW. Phthiriasis palpebrarum misdiagnosed as allergic blepharoconjunctivitis in a 6-year-old girl. *Niger J Clin Pr*. 2014;17:537-539.
 118. Ryan MF. Phthiriasis palpebrarum infection: a concern for child abuse. *J Emerg Med*. 2014;46:e159-e162.
 119. Ma DL, Vano-Galvan S. Infestation of the eyelashes with Phthirus pubis. *CMAJ*. 2010;182:E187.
 120. Burkhart C, Burkhart C. Oral Ivermectin therapy for phthiriasis palpebrum. *Arch Ophthalmol*. 2000;118:134-135.
 121. Karabela Y, Yardimci G, Yildirim I, Atalay E, Karabela SN. Treatment of phthiriasis palpebrarum and crab louse: petrolatum jelly and 1 % permethrin shampoo. *Case Rep Med*. 2015;2015:287906.
 122. Fioravanti M, Pampiglione S, Trentini M. A second species of *Tunga* (Insecta, Siphonaptera) infecting man: *Tunga trimamillata*. *Parasite*. 2003;10:282-283.
 123. Karunamoorthi K. Tungiasis: a neglected epidermal parasitic skin disease of marginalized populations — a call for global science and policy. *Parasitol Res*. 2013;112:3635-3643.
 124. Sachse MM, Guldbakke KK, Khachemoune A. *Tunga penetrans*: a stowaway from around the world. *J Eur Acad Dermatol Venereol*. 2007;21:11-16.
 125. Veraldi S, Valsecchi M. Imported tungiasis: a report of 19 cases and review of the literature. *Int J Dermatol*. 2007;46: 1061-1066.
 126. Grupper M, Potasman I. Outbreak of tungiasis following a trip to Ethiopia. *Trav Med Infect Dis*. 2012;10:220-223.
 127. Sood A, Raman DK, Joshi RK, Gupta D. Tungiasis: outbreak investigation of a zoonosis during overseas deployment. *Med J Armed Forces India*. 2017;73:375-379.
 128. Belaz S, Gay E, Robert-Gangneux F, Beaucournu JC, Guiguen C. Tungiasis outbreak in travelers from Madagascar. *J Trav Med*. 2015;22:263-266.
 129. Kulakov EL, Mann J, Bagla N, Cooper AJ. Painful nodule on the foot of a traveller returning from Africa. *Clin Exp Dermatol*. 2013;38:436-438.
 130. Jackson A, Stevenson L, Coggeshall P, Whitman TJ. A patient returning from Africa finds a mass imbedded in the skin of her right foot. *Clin Infect Dis*. 2012;55:1227.
 131. Peccerillo F, Zambito Spadaro F, Fabrizi G, Feliciani C, Pagliarello C, Stanganelli I. Not a simple plantar wart: a case of tungiasis. *J Eur Acad Dermatol Venereol*. 2018;32:e113-e114.
 132. Wilson ME, Chen LH, Han PV, et al. Illness in travelers returned from Brazil: the geosentinel experience and implications for the 2014 FIFA World Cup and the 2016 Summer Olympics. *Clin Infect Dis*. 2014;58:1347-1356.
 133. Leder K, Torresi J, Libman M, et al. GeoSentinel surveillance of illness in returned travelers, 2007-2011. *Ann Intern Med*. 2013; 158:456-468.
 134. Herbinger K, Siess C, Nothdurft H, von Sonnenburg F, Loscher T. Skin disorders among travellers returning from tropical and non-tropical countries consulting a travel medicine clinic. *Trop Med Int Health*. 2011;16:1457-1464.
 135. Muehlen M, Feldmeier H, Wilcke T, Winter B, Heukelbach J. Identifying risk factors for tungiasis and heavy infestation in a resource-poor community in northeast Brazil. *Trans R Soc Trop Med Hyg*. 2006;100:371-380.
 136. Mazigo HD, Bahemana E, Konje ET, et al. Jigger flea infestation (tungiasis) in rural western Tanzania: high prevalence and severe morbidity. *Trans R Soc Trop Med Hyg*. 2012; 106:259-263.
 137. Mwangi JN, Ozwara HS, Gicheru MM. Epidemiology of *Tunga penetrans* infestation in selected areas in Kiharu constituency, Murang'a County, Kenya. *Trop Dis Trav Med Vaccin*. 2015;1:13.
 138. Wafula ST, Ssemugabo C, Namuhani N, Musoke D, Ssempebwa J, Halage AA. Prevalence and risk factors associated with tungiasis in Mayuge district, Eastern Uganda. *Pan Afr Med J*. 2016;24:77.
 139. Ugbomoiko US, Ariza L, Babamale AO, Heukelbach J. Prevalence and clinical aspects of tungiasis in south-west Nigerian schoolchildren. *Trop Doct*. 2017;47:34-38.
 140. Wiese S, Elson L, Reichert F, Mambo B, Feldmeier H. Prevalence, intensity and risk factors of tungiasis in Kilifi County, Kenya: I. Results from a community-based study. *PLoS Negl Trop Dis*. 2017;11:e0005925.
 141. Girma M, Astatkie A, Asnake S. Prevalence and risk factors of tungiasis among children of Wensho district, southern Ethiopia. *BMC Infect Dis*. 2018;18:456.
 142. Ugbomoiko US, Ariza L, Ofoezie IE, Heukelbach J. Risk factors for tungiasis in Nigeria: identification of targets for effective intervention. *PLoS Negl Trop Dis*. 2007; 1:1-7.
 143. Feldmeier H, Heukelbach J, Ugbomoiko US, et al. Tungiasis—a neglected disease with many challenges for global public health. *PLoS Negl Trop Dis*. 2014;8:e3133.
 144. Mutebi F, Krücken J, von Samson-Himmelstjerna G, et al. Animal and human tungiasis-related knowledge and treatment practices among animal keeping households in Bugiri District, South-Eastern Uganda. *Acta Trop*. 2018; 177:81-88.
 145. Pilger D, Schwalfenberg S. Investigations on the biology, epidemiology, pathology, and control of *Tunga penetrans* in Brazil: VII. The importance of animal reservoirs for human infestation. *Parasitol Res*. 2008;102:875-880.
 146. Heukelbach J, Costa AML, Wilcke T, Mencke N, Feldmeier H. The animal reservoir of *Tunga penetrans* in severely affected communities of north-east Brazil. *Med Vet Entomol*. 2004;18: 329-335.
 147. Mutebi F, Krücken J, Feldmeier H, et al. Animal reservoirs of zoonotic tungiasis in endemic rural villages of Uganda. *PLoS Negl Trop Dis*. 2015;9:e0004126.
 148. Eisele M, Heukelbach J, Van Marck E, et al. Investigations on the biology, epidemiology, pathology and control of *Tunga penetrans* in Brazil: I. Natural history of tungiasis in man. *Parasitol Res*. 2003;90:87-99.
 149. Kosumi H, Iwata H, Miyazawa H, et al. Dermoscopic features of tungiasis. *J Eur Acad Dermatol Venereol*. 2018;32:e313-e314.

150. Feldmeier H, Heukelbach J, Eisele M, Sousa AQ, Barbosa LMM. Bacterial superinfection in human tungiasis. *Trop Med Int Health*. 2002;7:559-564.
151. Nyangacha RM, Odongo D, Oyieke F, et al. Secondary bacterial infections and antibiotic resistance among tungiasis patients in Western, Kenya. *PLoS Negl Trop Dis*. 2017;11:e0005901.
152. Brane S, Adams B, Bazemore A. Tungiasis in the returning traveler. *J Am Acad Dermatol*. 2005;52:1105.
153. Heukelbach J, Ugbomoiko U. Tungiasis in the past and present: a dire need for intervention. *Nig J Parasitol*. 2007;28:1-5.
154. Feldmeier H, Eisele M, Sabóia-Moura RC, Heukelbach J. Severe tungiasis in underprivileged communities: case series from Brazil. *Emerg Infect Dis*. 2003;9:949-955.
155. Feldmeier H, Sentongo E, Krantz I. Tungiasis (sand flea disease): a parasitic disease with particular challenges for public health. *Eur J Clin Microbiol Infect Dis*. 2013;32:19-26.
156. Walker SL, Lebas E, De Sario V, et al. The prevalence and association with health-related quality of life of tungiasis and scabies in schoolchildren in southern Ethiopia. *PLoS Negl Trop Dis*. 2017;11:e0005808.
157. Wiese S, Elson L, Feldmeier H. Tungiasis-related life quality impairment in children living in rural Kenya. *PLoS Negl Trop Dis*. 2018;12:e0005939.
158. Vasievich MP, Villarreal JDM, Tomecki KJ. Got the travel bug? A review of common infections, infestations, bites, and stings among returning travelers. *Am J Clin Dermatol*. 2016;17:451-462.
159. Tonge B. Tetanus from chigger flea sores. *J Trop Pediatr*. 1989;35:94.
160. Pallangyo P, Nicholas P. Disseminated tungiasis in a 78-year-old woman from Tanzania: a case report. *J Med Case Rep*. 2016;10:354.
161. Veraldi S, Dassoni F, Çuka E, Nazzaro G. Two cases of imported tungiasis with severe *Staphylococcus aureus* superinfection. *Acta Derm Venereol*. 2014;94:463-464.
162. Elson L, Wright K, Swift J, Feldmeier H. Control of tungiasis in absence of a roadmap: grassroots and global approaches. *Trop Med Infect Dis*. 2017;2:1-13.
163. Thielecke M, Raharimanga V, Rogier C, Stauss-Grabo M, Richard V, Feldmeier H. Prevention of tungiasis and tungiasis-associated morbidity using the plant-based repellent Zanzarin: a randomized, controlled field study in rural Madagascar. *PLoS Negl Trop Dis*. 2013;7:e2426.
164. Buckendahl J, Heukelbach J, Ariza L, Kehr JD, Seidenschwang M, Feldmeier H. Control of tungiasis through intermittent application of a plant-based repellent: an intervention study in a resource-poor community in Brazil. *PLoS Negl Trop Dis*. 2010;4:e879.
165. Thielecke M, Nordin P, Ngomi N, Feldmeier H. Treatment of tungiasis with dimeticone: a proof-of-principle study in rural Kenya. *PLoS Negl Trop Dis*. 2014;8:e3058.
166. Nordin P, Thielecke M, Ngomi N, Mudanga GM, Krantz I, Feldmeier H. Treatment of tungiasis with a two-component dimeticone: a comparison between moistening the whole foot and directly targeting the embedded sand fleas. *Trop Med Health*. 2017;45:1-7.
167. Heukelbach J, Franck S, Feldmeier H. Therapy of tungiasis: a double-blinded randomized controlled trial with oral ivermectin. *Mem Inst Oswaldo Cruz*. 2004;99:873-876.
168. Leung A, Woo T, Robson W, Trotter M. A tourist with tungiasis. *CMAJ*. 2007;177:343-344.
169. Heukelbach J, Wilcke T, Eisele M, Feldmeier H. Ectopic localization of tungiasis. *Am J Trop Med Hyg*. 2002;67:214-216.
170. Ariza L, Wilcke T, Jackson A, et al. A simple method for rapid community assessment of tungiasis. *Trop Med Int Health*. 2010;15:856-864.
171. Kimani B, Nyagero J, Ikamari L. Knowledge, attitude and practices on jigger infestation among household members aged 18 to 60 years: case study of a rural location in Kenya. *Pan Afr Med J*. 2012;13(suppl 1):1-5.
172. Tomczyk S, Deribe K, Brooker SJ, Clark H, Rafique K, Davey G. Association between footwear use and neglected tropical diseases: a systematic review and meta-analysis. *PLoS Negl Trop Dis*. 2014;8:e3285.
173. Joseph JK, Bazile J, Mutter J, et al. Tungiasis in rural Haiti: a community-based response. *Trans R Soc Trop Med Hyg*. 2006;100:970-974.
174. Jenkins N, Ferguson D, Alp N, Harrison T, Bowler I. Urban trench fever presenting as culture-negative endocarditis. *QJM*. 2009;102:63-65.
175. Pitman N, Hernandez A, Hernandez E. Comparison of pediculicidal and ovicidal effects of two pyrethrin-piperonyl-butoxide agents. *Clin Ther*. 1987;9:368-372.
176. Taplin D, Meinking T, Castillero P, Sanchez R. Permethrin 1% creme rinse for the treatment of *Pediculus humanus var capitis* infestation. *Pediatr Dermatol*. 1986;3:344-348.
177. Stichele RHV, Dezeure EM, Bogaert MG. Systematic review of clinical efficacy of topical treatments for head lice. *BMJ*. 1995;311:604-608.
178. Burgess IF, Brown CM, Lee PN. Treatment of head louse infestation with 4% dimeticone lotion: randomised controlled equivalence trial. *BMJ*. 2005;330:1423-1425.
179. Heukelbach J, Sonnberg S, Becher H, Melo I, Speare R, Oliveira FA. Ovicidal efficacy of high concentration dimeticone: a new era of head lice treatment. *J Am Acad Dermatol*. 2011;64(4):e61-e62.
180. Taplin D, Castillero PM, Spiegel J, Mercer S, Rivera AA, Schachner L. Malathion for treatment of *Pediculus humanus var capitis* infestation. *JAMA*. 1982;247:3103-3105.
181. Meinking TL, Vicaria M, Eyerdam DH, Villar ME, Reyna S, Suarez G. A randomized, investigator-blinded, time-ranging study of the comparative efficacy of 0.5% malathion gel versus Ovide Lotion (0.5% malathion) or Nix Creme Rinse (1% permethrin) used as labeled, for the treatment of head lice. *Pediatr Dermatol*. 2007;24:405-411.
182. Meinking TL, Villar ME, Vicaria M, et al. The clinical trials supporting benzyl alcohol lotion 5% (Ulesfia TM): a safe and effective topical treatment for head lice (pediculosis humanus capitis). *Pediatr Dermatol*. 2010;27:19-24.
183. Popescu C, Popescu R. Efficacy and safety of spinosad cream rinse for head lice. *Arch Dermatol*. 2012;148:1070.
184. Pariser D, Meinking T, Bell M, Ryan W. Topical 0.5% ivermectin lotion for treatment of head lice. *N Engl J Med*. 2012;367:1687-1693.
185. Bowles VM, VanLuvanee LJ, Alsop H, et al. Clinical studies evaluating abametapir lotion, 0.74%, for the treatment of head louse infestation. *Pediatr Dermatol*. 2018;35:616-621.
186. Gonzalez Audino P, Vassena C, Zerba E, Picollo M. Effectiveness of lotions based on essential oils from aromatic plants against permethrin resistant *Pediculus humanus capitis*. *Arch Dermatol Res*. 2007;299:389-392.
187. Gonzalez-Audino P, Picollo MI, Gallardo A, Toloza A, Vassena C, Mougabure-Cueto G. Comparative toxicity of oxygenated monoterpenoids in experimental hydroalcoholic

- lotions to permethrin-resistant adult head lice. *Arch Dermatol Res.* 2011;303:361-366.
188. Gutiérrez MM, Werdin-González JO, Stefanazzi N, Bras C, Ferrero AA. The potential application of plant essential oils to control *Pediculus humanus capitis* (Anoplura: Pediculidae). *Parasitol Res.* 2016;115:633-641.
189. Louis SJ, Bronsnick T, Louis FJ, Rao B. Tungiasis in haiti: a case series of 383 patients. *Int J Dermatol.* 2014;53:999-1004.

Answers to CME examination

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