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WILDERNESS MEDICAL SOCIETY CLINICAL PRACTICE GUIDELINES

Wilderness Medical Society Clinical Practice Guidelines for the Prevention and Management of Tick-Borne Illness in the United States

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The Wilderness Medical Society convened an expert panel to develop evidence-based guidelines for the prevention and management of tick-borne illness (TBI). Recommendations are graded based on quality of supporting evidence according to criteria put forth by the American College of Chest Physicians. The guidelines include a brief review of the clinical presentation, epidemiology, prevention, and management of TBI in the United States, with a primary focus on interventions that are appropriate for resource-limited settings. Strong recommendations are provided for the use of DEET, picaridin, and permethrin; tick checks; washing and drying clothing at high temperatures; mechanical tick removal within 36 h of attachment; single-dose doxycycline for high-risk Lyme disease exposures versus "watchful waiting;" evacuation from backcountry settings for symptomatic tick exposures; and TBI education programs. Weak recommendations are provided for the use of light-colored clothing; insect repellents other than DEET, picaridin, and permethrin; and showering after exposure to tick habitat. Weak recommendations are also provided against passive methods of tick removal, including the use of systemic and local treatments. There was insufficient evidence to support the use of long-sleeved clothing and the avoidance of tick habitat such as long grasses and leaf litter. Although there was sound evidence supporting Lyme disease vaccination, a grade was not offered as the vaccine is not currently available for use in the United States.

Keywords: DEET, Lyme disease, spirochetes, rickettsia, prophylaxis

Introduction

In the United States, 95% of human vector-borne diseases reported to the Centers for Disease Control and Prevention (CDC) per year are attributable to tick-borne pathogens.[1] In turn, the number of cases of tick-borne illnesses (TBIs) reported to the CDC per year in the United States has more than doubled over the past 2 decades, totaling 50,865 in 2019,[2] with formal reporting likely underestimating the disease burden of TBIs.[3] Although Lyme disease is the most commonly reported vector-borne disease in the United States, the incidence of other TBIs, including anaplasmosis, spotted fever rickettsiosis, babesiosis, tularemia, alpha-gal syndrome, and Powassan virus, continues to rise as well.[4]

Given the increased incidence of TBI, it is critical for providers to be comfortable with the prevention and management of tick bites. TBI, however, is a complex landscape with a considerable volume of literature describing relatively rare syndromes and controversial treatment regimens. In an effort to deliver a succinct clinical practice guideline (CPG), the authors have chosen to focus the scope of this article on those issues most

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relevant to the front-line provider with minimal resources: the prevention of tick bites and the management of TBI in settings where diagnostics and therapeutics are limited. Several recent articles are available for the reader interested in a comprehensive review of emerging TBIs or the management of TBI in the hospital setting.[5,6]

Methods

An expert panel was convened through the Wilderness Medical Society clinical practice guidelines committee. Panelists were selected based on clinical interest or research experience. Articles were identified in 2 searches. The first search identified articles through the PubMed and Google Scholar databases using a keyword search with the following terms: tick, tick-borne, tick bite, tick-borne disease, borreliosis, Lyme disease, Rocky Mountain spotted fever. The second search broadened these topics to include a keyword search with the following terms: tick prevention (with permethrin, tick checks, clothing, and habitat management), DEET, and Lyme vaccine. These searches were supplemented by manually searching the references of the obtained articles, existing practice guidelines, and CDC references. All articles were peer reviewed. Methodological scope included randomized controlled trials, observational studies, and case series from both human and animal trials. Given the broad scope of TBI and the limited scope of this CPG, review articles have been cited mainly to augment topics not fully covered by this CPG. However, primary literature was used to derive the graded recommendations within this guideline. All literature searches were performed by study authors, without restrictions placed on date of publication or country of origin. The panel used the American College of Chest Physicians (ACCP) (online Supplementary Table)[7] classification scheme for grading evidence and recommendations. There were instances in which the ACCP schematic did not adequately describe our recommendations. Recommendations based on reasonable clinical practice, but not supported by data, were classified as "expert opinion."

Ticks and Human Illness

TICKS AS A VECTOR FOR DISEASE

Transmission of TBIs to humans requires an interaction between pathogen, vector, and host; each tick species can serve as a vector for a number of different bacterial, viral, and parasitic pathogens (Tables 1 and 2)[8] and may seek out different hosts for blood meals in each of their 3 life stages (https://www.cdc.gov/ticks/index.html) (Figure 1).[1] Disease transmission can occur between vector and host directly through the feeding process, or between tick vectors co-feeding on the same host.[1] As a result of these vector-host interactions, the geographic distribution of TBIs closely reflects the distribution of the tick vectors that transmit disease (Figure 2).[8]

TICK BEHAVIOR AND DISEASE TRANSMISSION

Ticks find their hosts by sensing heat, exhalations, vibrations, and odors. Once a host has been found, ticks burrow their hypostome under the skin using a cutting movement.[9] Most tick bites are painless. Once the tick is feeding, a complex molecular cascade facilitates the transmission of pathogens from the gut of an infected tick vector to the host. Prostaglandins in tick saliva inhibit the host's local immune response, and tick salivary apyrase maintains blood flow into the bite site, stimulates local vasodilation, and prevents platelet aggregation. Other tick salivary enzymes inhibit the coagulation cascade, enhancing blood flow to the lesion.[10] Importantly, there is considerable variability in the time it takes to transmit pathogen from tick to human. In Lyme disease, disease transmission is believed to take >36 h[11]; in contrast, Rocky Mountain spotted fever can be transmitted in approximately 15 min.[12]

INCIDENCE OF TICK-BORNE ILLNESS

As of 2019, Lyme disease accounted for more than 69% of more than 50,000 reported cases of TBI, whereas anaplasmosis represented 16% and spotted fever rickettsiosis represented 10%.[2] Although summer has been associated with the highest risk of Lyme disease, transmission is possible year-round.[13] For example, hunters must remain vigilant for tick bites through the fall season based on seasonal tick collection surveys.

Environmental factors that influence tick and host distribution, feeding patterns, and survivability are rapidly changing. Habitat fragmentation, urbanization, and deforestation all affect the distribution and migration of tick vectors and hosts. Climate change, with its associated temperature and precipitation fluctuations, has enabled tick expansion to regions that have previously experienced little TBI.[14–16] It is thought that these anthropogenic changes are contributing to the expanding range of tick habitat as well as an increase in tick abundance in existing habitats.[17]

TICK IDENTIFICATION, COINFECTION, AND MAJOR TBIS OF THE UNITED STATES

Ticks commonly encountered in the United States are shown in Figure 3 (https://www.cdc.gov/ticks/index.

Table 1. Pathogenic organisms and geographical distribution of important US arthropods implicated in tick-borne diseases[8]

Tick	*Distribution*	*Pathogen*	*Disease*	*Comments*
Blacklegged tick – *Ixodes scapularis*	Eastern US	*Borrelia burgdorferi* *Borrelia mayonii* *Anaplasma phagocytophilum* *Borrelia miyamotoi* *Ehrlichia muris eauclariensis* *Babesia microti* Powassan virus	Lyme disease Anaplasmosis Relapsing fever Ehrlichiosis Babesiosis Powassan virus	Greatest risk of being bitten exists in the spring, summer, and fall in the NE, upper MW, and mid-Atlantic. However, adult ticks may be out searching for a host any time winter temperatures are above freezing. All life stages bite humans, but nymphs and adult females are most commonly found on people
Lone star tick – *Amblyomma americanum*	Eastern US Southern US	*Ehrlichia chaffeensis* *Ehrlichia ewingii* *Francisella tularensis* Heartland virus Bourbon virus	Ehrlichiosis Tularemia Heartland virus Bourbon virus STARI Alpha-gal allergy	Greatest risk of being bitten exists in early spring through late fall. A very aggressive tick that bites humans. Adult female is distinguished by a white dot ("lone star") on her back. Nymph and adult females most frequently bite humans.
American dog tick – *Dermacentor variabilis*	East of Rockies, Pacific Coast	*Francisella tularensis* *Rickettsia rickettsii*	Tularemia Rocky Mountain spotted fever	The greatest risk of being bitten occurs during spring and summer. Adult females are most likely to bite humans.
Brown dog tick – *Rhipicephalus sanguineus*	Worldwide	*Rickettsia rickettsii*	Rocky Mountain spotted fever	Dogs are the primary host for the brown dog tick in each of its life stages but may also bite humans or other mammals.
Groundhog tick – *Ixodes cookei*	Eastern half of US	Powassan virus	Powassan virus	All life stages feed on a variety of warm-blooded animals (groundhogs, skunks, squirrels, raccoons, foxes, weasels) and occasionally humans and domestic animals.
Gulf Coast tick – *Amblyomma maculatum*	SE and mid-Atlantic states, AZ	*Rickettsia parkeri*	*Rickettsia parkeri* rickettsiosis	Larvae/Nymphs feed on birds and small rodents; adults feed on deer and other wildlife. Adults can transmit disease to humans.
Rocky Mountain wood tick – *Dermacentor andersoni*	Rocky mountain states	*Rickettsia rickettsii* CO tick fever virus *Francisella tularensis*	Rocky Mountain spotted fever CO tick fever Tularemia	Adults feed on large mammals; larvae/nymphs feed on rodents. Adults can transmit disease to humans.
Soft tick – *Ornithodoros spp.*	Western US, TX	*Borrelia hermsii* *Borrelia turicatae*	Tickborne relapsing fever	Rustic cabins, cave exposure. Ticks emerge at night, feed while people sleep.
Western blacklegged tick – *Ixodes pacificus*	Pacific Coast	*Anaplasma phagocytophilum* *Borrelia burgdorferi* *Borrelia miyamotoi*	Anaplasmosis Lyme disease Relapsing fever	Larvae/Nymphs feed on lizards, birds, rodents; adults feed on deer. Nymphs/Adult females most often bite humans.

html). Tick identification can be challenging given the variety of tick life cycles and the dramatic anatomic changes that occur with engorgement. Moreover, in a significant proportion of TBIs, the patient will not recall a tick or tick bite.[18] Providers must be prepared to diagnose and manage TBI without definitive tick identification.

Several tick species are able to carry multiple pathogens (Table 1). In 1 study, nearly 25% of *Ixodes* were coinfected with some combination of the bacteria or parasites causing Lyme disease, anaplasmosis, or babesiosis.[19] Although TBI diagnosis is not the focus of this CPG, providers should be aware of high rates of coinfection[20,21]; the presence of 1 TBI should in many instances prompt testing for others. A brief clinical summary of the major TBIs found in the United States follows, supplemented by further detail in Table 2.

Table 2. Clinical syndromes, laboratory diagnosis, and treatment of common bacterial, protozoal, and viral tick-borne illnesses[8,77,104]

Disease	*Causative organism*	*Distribution*	*Incubation*	*Clinical syndrome*	*Laboratory findings*	*Laboratory diagnosis*	*Treatment*
Bacterial tick-borne illnesses							
Borrelioses							
Lyme disease	*Borrelia burgdorferi* *Borrelia mayonii*	Upper Midwest and Northeastern US 14 states accounted for 95% of Lyme disease reports in 2015: CT, DE, ME, MD, MA, MN, NH, NJ, NY, PA, RI, VT, VA, WI Some cases also reported in northern CA, OR, and WA	3–30 d	Localized: - erythema migrans - flu-like symptoms - lymphadenopathy Disseminated: - multiple secondary annular rashes - flu-like symptoms - lymphadenopathy Rheumatologic manifestations - transient, migratory mono/polyarthritis - migratory musculoskeletal pain - Baker's cyst - recurrent arthralgia Cardiac manifestations - conduction abnormalities - myocarditis, pericarditis Neurologic manifestations - Bell's palsy or other cranial neuropathy - meningitis - motor/sensory radiculoneuropathy - cognitive difficulties - encephalitis, encephalomyelitis, encephalopathy, pseudotumor cerebri Additional manifestations - conjunctivitis, keratitis, uveitis - mild hepatitis - splenomegaly	- Elevated ESR - Elevated hepatic transaminases - Microscopic hematuria/proteinuria - Lyme meningitis: lymphocytic pleocytosis, elevated protein, normal glucose in CSF	2-tiered serologic testing*: - ELISA (IgG/IgM) - Western blot if positive or equivocal * 2-tiered testing often falsely negative early in disease	*Prophylaxis* - doxycycline 200 mg, once *Localized Lyme disease* Adults: - doxycycline 100 mg bid, 10–21 d - cefuroxime 500 mg bid, 14–21 d - amoxicillin 500 mg tid, 14–21 d Children: - amoxicillin 50 $mg\cdot kg^{-1}$ daily divided into 3 doses, 14–21 d - doxycycline 4 $mg\cdot kg^{-1}$ daily divided into 2 doses, 10–21 d - cefuroxime 30 $mg\cdot kg^{-1}$ daily divided into 2 doses, 14–21 d *Lyme meningitis* Adults: - ceftriaxone 2$g\cdot d^{-1}$ IV, 10–28 d - cefotaxime 6 $g\cdot d^{-1}$ IV divided q 8 h, 10–28 d Children: - ceftriaxone 50–75 $mg\cdot kg^{-1}$ daily IV, 10–28 d - cefotaxime 150–200 $mg\cdot kg^{-1}$ daily IV divided q 8 h, 10–28 d *Cranial-nerve palsy without meningitis* Adults: - doxycycline 200 $mg\cdot d^{-1}$ divided bid, 14–21 d - amoxicillin 1500 $mg\cdot d^{-1}$ divided tid, 14–21 d - cefuroxime axetil 1000 $mg\cdot d^{-1}$ divided bid, 14–21 d Children: - doxycycline 4 $mg\cdot kg^{-1}$ daily divided bid, 14–21 d - amoxicillin 50 $mg\cdot d^{-1}$ divided tid, 14–21 d - cefuroxime axetil 30 $mg\cdot d^{-1}$ divided bid, 14–21 d *Carditis* Same oral agents as for localized Lyme disease, same parenteral agents as for Lyme meningitis, 14–21 d *Arthritis* Same oral agents as for localized Lyme disease, same parenteral agents as for Lyme meningitis, 28 d

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Table 2 *(continued)*

Disease	*Causative organism*	*Distribution*	*Incubation*	*Clinical syndrome*	*Laboratory findings*	*Laboratory diagnosis*	*Treatment*
Borrelia miyamotoi disease	*Borrelia miyamotoi*	Upper Midwest, Northeast, and mid-Atlantic states Presumed in CA	Days to weeks	- Fever - Chills - Fatigue - Severe headache - Arthralgia/Myalgia - Dizziness, confusion, vertigo - Rash - Dyspnea - Nausea, abdominal pain, diarrhea, anorexia	- Leukopenia - Thrombocytopenia - Elevated hepatic transaminases	Signs/Symptoms with: - PCR for DNA, or -antibody tests	No comprehensive studies to evaluate treatment regimens; antibiotics for Lyme disease successful in case series
Tick-borne relapsing fever	*Borrelia hermsii* *Borrelia turicatae*	14 western states: AZ, CA, CO, ID, KS, MT, NV, NM, OK, OR, TX, UT, WA, WY	~7 d, followed by febrile episodes lasting ~3 d and separated by ~7 d afebrile periods	- Headache - Myalgia - Chills - Nausea, vomiting - Arthralgia - Facial palsy	- Normal-increased WBC with left shift - Increased serum bilirubin - Thrombocytopenia - Elevated ESR - Prolonged PT/PTT	- Microscopy/Culture while febrile - Peripheral blood smear - Convalescent serologic testing	Adults: - tetracycline 500 mg qid, 10 d - erythromycin 500 mg qid, 10 d - ceftriaxone 2 $g \cdot d^{-1}$ IV, 10–14 d Children: - erythromycin 12.5 $mg \cdot kg^{-1}$ qid, 10 d
Rickettsioses							
Rocky Mountain spotted fever	*Rickettsia rickettsii*	Throughout the US 5 southern states account for 60% of cases: NC, OK, AR, TN, MO	3–12 d	Early (1–4 d): - high fever - severe headache - malaise - myalgia - edema around eyes, hands - nausea, vomiting, anorexia Late (5 d and beyond): - AMS, coma, cerebral edema - pulmonary edema, ARDS - necrosis, requiring amputation - multiorgan system damage (CNS, renal failure) Rash: develops 2–5 d after symptom onset *Early rash* - maculopapular—on wrist, forearms, ankles, spreading to trunk, palms, and soles *Late rash* - petechial—signifies severe disease, develops after day 6	- Thrombocytopenia - Elevated hepatic transaminases - Hyponatremia	4× increase in IgG antibody by IFA in paired serum samples within first week of illness, and 2–4 wk later PCR for DNA in skin biopsy IHC staining from skin/ biopsy	Adults: - doxycycline 100 mg bid Children: - doxycycline 2.2 $mg \cdot d^{-1}$ bid Treat for at least 3 d after fever subsides and symptoms improve, for minimum of 5–7 d Start treatment on clinical suspicion
Rickettsia parkeri Rickettsiosis	*Rickettsia parkeri*	Southeastern and mid-Atlantic states, parts of southern AZ	2–10 d	- Inoculation eschar at site of tick attachment Several days after eschar: - fever - headache - rash (maculopapular/ papulovesicular eruption on trunk/extremities) - muscle aches	- Elevated hepatic transaminases - Mild leukopenia - Mild thrombocytopenia	PCR for DNA in eschar swab, whole blood, skin biopsy 4× increase in IgG antibody by IFA in paired serum samples within first week of illness, and 2–4 wk later	Adults: - doxycycline 100 mg bid Children: - doxycycline 2.2 $mg \cdot kg^{-1}$ bid Treat for at least 3 d after fever subsides and symptoms improve, for minimum of 5–7 d

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Table 2 *(continued)*

Disease	*Causative organism*	*Distribution*	*Incubation*	*Clinical syndrome*	*Laboratory findings*	*Laboratory diagnosis*	*Treatment*
Pacific Coast tick fever	*Rickettsia* sp 364D	CA, OR, WA	Not documented	Eschar, followed by fever, regional lymphadenopathy, headache, myalgia, fatigue	Not documented	PCR for DNA in eschar swab 4× increase in IgG antibody by IFA in paired serum samples within first week of illness, and 2–4 wk later	Adults: - doxycycline 100 mg bid Children: - doxycycline 2.2 mg·kg^{-1} bid Treat for at least 3 d after fever subsides and symptoms improve, for minimum of 5–7 d
Ehrlichiosis and *Anaplasmosis*							
Anaplasmosis	*Anaplasma phagocytophilum*	Upper Midwest, northeastern US 8 states account for 90% of cases: VT, ME, RI, MN, MA, WI, NH, NY	5–14 d	- Fever, chills, rigors - Severe headache - Malaise - Myalgia - GI symptoms: nausea, vomiting, diarrhea, anorexia - Rash	- Mild anemia - Thrombocytopenia - Leukopenia with lymphopenia and left shift - Mild elevation in hepatic transaminases	PCR for DNA in whole blood 4× increase in IgG antibody by IFA in paired serum samples within first week of illness, and 2–4 wk later IHC staining of organism from skin, tissue, or BM biopsy Morulae in granulocytes on blood smear	Adults: - doxycycline 100 mg bid, 10–14 d Children: - doxycycline 2.2 mg·kg^{-1} bid, 10–14 d Start treatment on clinical suspicion
Ehrlichiosis	*Ehrlichia chaffeensis, ewingii, muris eauclairensis*	Southeastern, south-central US, 3 states account for 35% of cases: OK, MO, AR	5–14 d	- Fever, chills - Headache - Malaise - Muscle pain - GI symptoms: nausea, vomiting, diarrhea, anorexia - AMS - Rash	- Thrombocytopenia - Leukopenia - Anemia - Mild elevation in hepatic transaminases	PCR for DNA in whole blood 4× increase in IgG antibody by IFA in paired serum samples within first week of illness, and 2–4 wk later IHC staining of organism from skin, tissue, or BM biopsy	Adults: - doxycycline 100 mg bid Children: - doxycycline 2.2 mg·kg^{-1} bid Treat for at least 3 d after fever subsides and symptoms improve, for minimum of 5–7 d Start treatment on clinical suspicion

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Table 2 *(continued)*

Disease	*Causative organism*	*Distribution*	*Incubation*	*Clinical syndrome*	*Laboratory findings*	*Laboratory diagnosis*	*Treatment*
Other							
Tularemia	*Francisella tularensis*	All states except HI	3–5 d (range 1–21 d)	- Fever, chills - Headache - Malaise, fatigue - Anorexia - Myalgia - Chest discomfort, cough - Sore throat - Vomiting, diarrhea, abdominal pain Ulceroglandular: - localized lymphadenopathy - cutaneous ulcer at infection site Oculoglandular: - photophobia - excessive lacrimation - conjunctivitis - preauricular, submandibular, or cervical lymphadenopathy Oropharyngeal: - severe throat pain - exudative pharyngitis or tonsillitis - cervical, preparotid, and/or retropharyngeal lymphadenopathy Pneumonic: - nonproductive cough - substernal tightness - pleuritic chest pain - hilar adenopathy, infiltrate, or pleural effusion on chest x-ray Typhoidal: - any combination of general symptoms (without localizing symptoms or other syndrome)	- Leukocytosis - Elevated ESR - Thrombocytopenia - Hyponatremia - Elevated hepatic transaminases - Elevated creatine phosphokinase - Myoglobinuria - Sterile pyuria	Isolation of *F tularensis* from clinical specimen or 4× increase in serum antibody titer to *F tularensis* antigen between acute and convalescent specimens Detection of *F tularensis* in clinical specimen by DFA or PCR, or single positive antibody titer to *F tularensis* antigen	Adults: - streptomycin 1 g IM bid, min 10 d - gentamicin[a] 5 $mg\cdot kg^{-1}$ IM/IV daily, min 10 d - ciprofloxacin[a] 400 mg IV or 500 mg po bid, 10–14 d Doxycycline 100 mg bid, 14–21 d Children: - streptomycin 15 $mg\cdot kg^{-1}$ IM bid, min 10 d - gentamicin[a] 2.5 $mg\cdot kg^{-1}$ IM/IV tid,[b] min 10 d - ciprofloxacin[a] 15 $mg\cdot kg^{-1}$ IV or po bid, min 10 d

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Table 2 *(continued)*

Disease	*Causative organism*	*Distribution*	*Incubation*	*Clinical syndrome*	*Laboratory findings*	*Laboratory diagnosis*	*Treatment*
Protozoan, viral, and other tick-borne illnesses							
Protozoa							
Babesiosis	*Babesia microti*, *Babesia spp.*	Upper Midwest and Northeastern US	1–9 wk	- Fever, chills, sweats - Malaise, fatigue - Myalgia, arthralgia, headache - GI symptoms: anorexia, nausea, abdominal pain, vomiting - Dark urine - Less common: cough, sore throat, emotional lability, depression, photophobia, conjunctival injection - Mild splenomegaly, hepatomegaly, jaundice	- Decreased hematocrit - Thrombocytopenia - Elevated serum creatinine and BUN - Mildly elevated hepatic transaminases	- Identification of intraerythrocytic *Babesia* parasites on peripheral blood smear, or - PCR, or - isolation of *Babesia* parasites from whole blood by animal inoculation Supportive criteria: - *Babesia*-specific antibody titer by IFA	Adults: - atovaquone 750 mg q 12 h AND azithromycin 500–1000 mg day 1, 150–1000 mg daily on subsequent days, 7–10 d Children: - clindamycin 300–600 mg IV q 6 h OR 600 mg po q 8 h AND quinine 650 mg po q 6–8 h, 7–10 d
Viruses							
Powassan virus disease	*Powassan virus*	Northeastern states and Great Lakes region	1–4 wk	- Fever, headache, vomiting, generalized weakness - Usually progresses to meningoencephalitis, may include meningeal signs, AMS, seizures, aphasia, paresis, movement disorders, cranial nerve palsies	- CSF: lymphocytic pleocytosis, mildly elevated protein, normal glucose	Virus-specific IgM antibodies in serum or CSF RT-PCR for viral RNA in acute CSF specimen or tissues	No specific antiviral treatment is available Supportive care
Colorado tick fever	*Colorado tick fever virus*	Western US, primarily CO, UT, MT, WY	1–14 d	- Fever, chills, headache, myalgias, lethargy - 50% have biphasic illness with symptoms remitting after 2–4 d, then recurring 1–3 d later - Conjunctival injection, pharyngeal erythema, lymphadenopathy - Maculopapular/Petechial rash in <20% - Prolonged convalescence with weakness and fatigue - DIC and meningoencephalitis is rare in children, can be fatal	- Leukopenia - Moderate thrombocytopenia	Culture and RT-PCR during first 2 wk of illness Serologic assays (IgM-capture EIA, IFA, plaque-reduction neutralization) on convalescent samples	No specific antiviral treatment is available Supportive care
Heartland virus disease	*Heartland virus*	Midwest, southern US	Unknown	- Fever - Fatigue - Decreased appetite - Headache - Arthralgia - Myalgia - Nausea - Diarrhea	- Leukopenia - Thrombocytopenia - Mild elevation in liver transaminases	Viral RNA and IgM/IgG antibodies	No specific antiviral treatment is available Supportive care

AMS, altered mental status; ARDS, acute respiratory response syndrome; bid, twice daily; BUN, blood urea nitrogen; CNS, central nervous system; CSF, cerebrospinal fluid; DIC, disseminated intravascular coagulation; EIA, enzyme-linked immunoassay; ELISA, enzyme-linked immunosorbent assay; ESR, erythrocyte sedimentation rate; GI, gastrointestinal; IFA, immunofluorescence assay; IHC, immunohistochemistry; IV, intravenous; PCR, polymerase chain reaction; PT/PTT, prothrombin time/partial thromboplastin time; tid, thrice daily; WBC, white blood cell.

[a]Not a Food and Drug Administration–approved use.

[b]Once-daily dosing could be considered in consultation with a pediatric ID specialist.

Bacteria

Anaplasmosis

Anaplasmosis, previously known as human granulocyte ehrlichiosis, is caused by the pathogen *Anaplasma phagocytophilum* carried by *Ixodes* spp ticks. Anaplasmosis is most frequently reported in the Upper Midwest and Northeastern United States, with a distribution that overlaps with Lyme disease. The incubation period is 5 to 14 d, and common symptoms include fever, rigors, headache, myalgias, vomiting, and diarrhea. Rash is uncommon. Doxycycline is first-line therapy.

Ehrlichiosis

Ehrlichiosis is the general name used to describe disease caused by *Ehrlichia chaffeensis*, *Ehrlichia ewingii*, and *Ehrlichia muris eauclairensis* bacteria and is most commonly reported in the Southeastern and South Central United States. It is transmitted by *Amblyomma americanum* (the lone star tick) and *Ixodes scapularis* (the blacklegged tick). Incubation is 5 to 14 d, and symptoms include fever, headache, myalgias, vomiting, diarrhea, and rash. Doxycycline is first-line therapy.

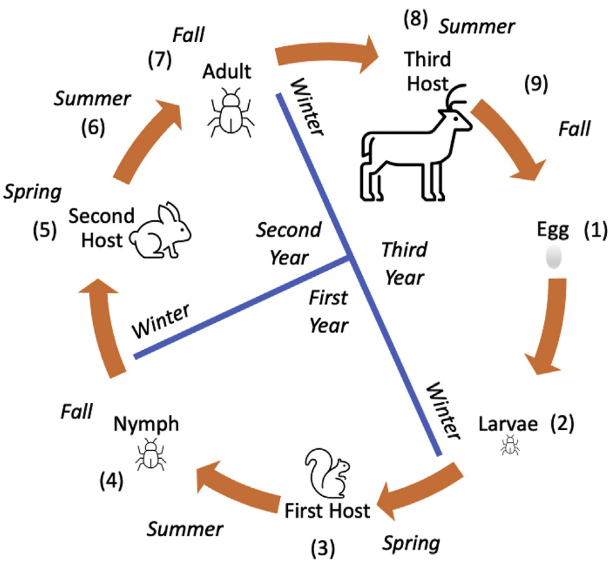

Figure 1. The 3-host life cycle. After the adult female leaves the third host to lay eggs in the fall (1), the eggs hatch into larvae and overwinter (2). The larvae will attach to the first host, usually a small rodent, the following spring (3) and remain attached until late summer, when they drop to molt into nymphs in the fall. After overwintering again, the nymphs will seek out a second host in the spring, again typically a small rodent (5), feed, and then detach later in the summer (6). Nymphs will molt into adults in late summer to fall (7), overwinter, and then seek out a third host in the spring, typically a larger mammal (8). Adult ticks will feed and mate on the third host during the summer, and the female adults will detach in the fall to continue the cycle (9). The 3 hosts are not always different species or different individuals and may be human hosts in all 3 stages.[101]

Lyme Disease and Other Emerging Borrelia Infections

Lyme disease is caused by the spirochete *Borrelia burgdorferi* and is transmitted by *I scapularis* (the blacklegged tick) and *Ixodes pacificus* (the Western blacklegged tick). The majority of cases are reported in the Midwest and Northeast, but Lyme disease is also common in California, Oregon, and Washington. Incubation ranges from 3 to 30 d.

Lyme disease presents in 3 stages: early localized, early disseminated, and late disseminated. In the early localized stage, common symptoms include fever, headache, myalgias, arthralgias, and erythema migrans (EM). EM is estimated to appear in approximately 70 to 80% of cases.[22] The rash is classically described as a "bull's eye" lesion with central clearing that occurs proximal to the site of the tick bite (Figure 4). However, misidentification of EM is relatively common[23]; atypical presentations include lesions with crusts, nodules, or a blueish coloring (Figure 4).

Early disseminated Lyme disease is characterized by multiple annular EM distant from the original tick bite, flu-like symptoms, and neurologic or cardiac manifestations such as cranial nerve palsy, meningitis, or conduction abnormalities. Lyme carditis is a rare, but it is an important cause of mortality[24] and may result in complete heart block as early as 4 d after a tick bite.[25]

Late disseminated Lyme disease occurs months or years after the initial tick bite and is characterized by arthralgias in 1 or more major joints or neurologic symptoms such as sleep disturbances, migraines, vertigo, and numbness in the hands, feet, arms, or legs.

Borrelia miyamotoi is an emerging borrelial infection with a distribution similar to Lyme disease. Although the clinical presentation is similar to Lyme disease, rash is uncommon. An early case series described a hospitalization rate of 24%.[26] *Borrelia mayonii*, recently discovered by researchers at the Mayo Clinic, also has a similar presentation to Lyme disease but has thus far been confined to the Upper Midwest. *B mayonii* infection is associated with higher concentrations of spirochetes in the blood when compared to *B burgdorferi.*[27] Doxycycline is first-line therapy.

Tickborne Relapsing Fever

Tickborne relapsing fever is also a spirochetal disease most commonly caused by *Borrelia hermsii* and *Borrelia turicatae* within the United States. Tickborne relapsing fever is

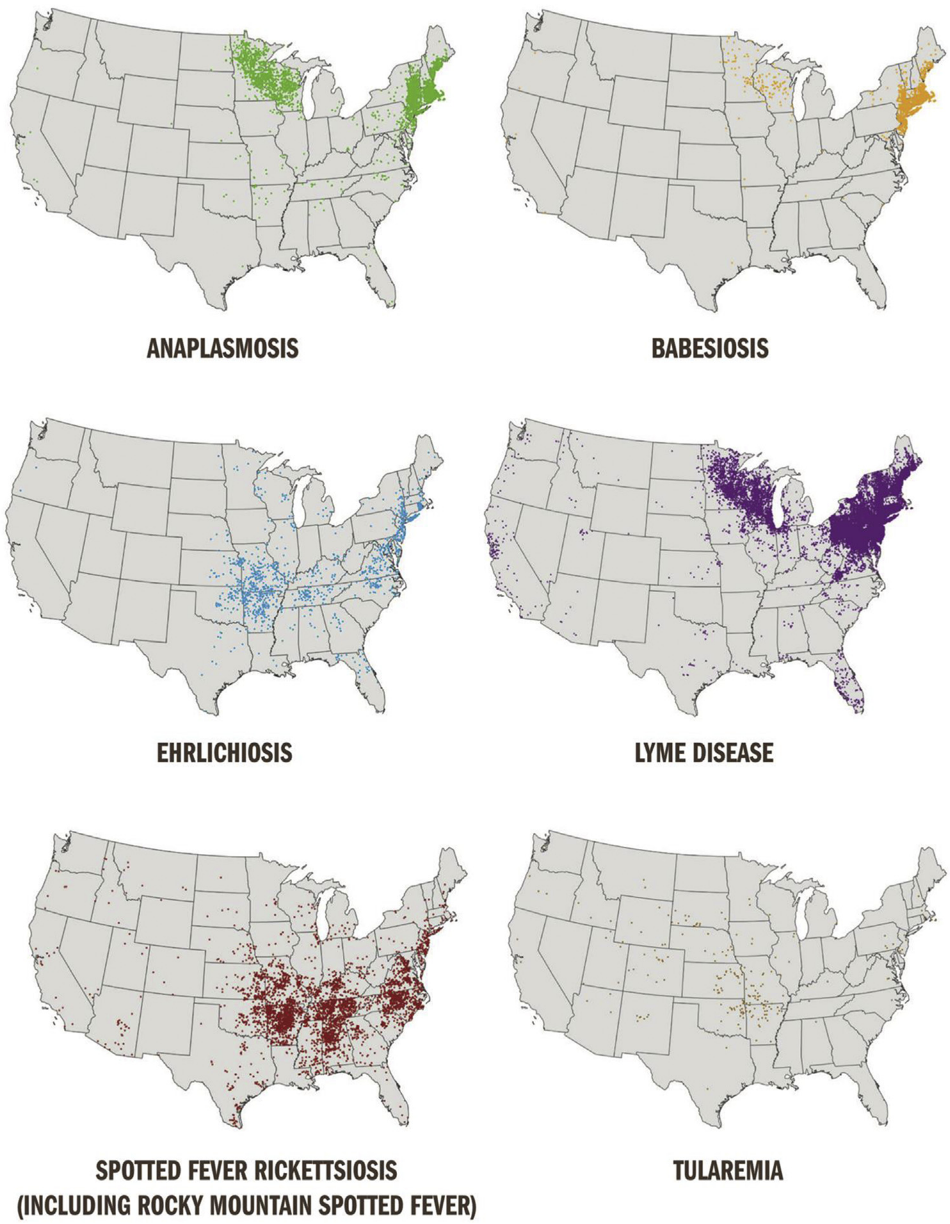

Figure 2. Geographical distribution of US tick-borne illnesses reported to the CDC in 2016.[8] Source: United States Centers for Disease Control and Prevention (public domain).

most commonly reported in western states. The disease is strongly associated with sleeping in rustic cabins or lean-tos where rodents cohabitate. In Texas, the disease is associated with caving. The disease is carried by the soft tick *Ornithodoros* and is characterized by a relapsing and recurring fever, which typically returns every 3 d. Incubation is approximately 1 wk. Other symptoms include headache, vomiting, myalgias, and arthralgias. First-line treatment is tetracycline or erythromycin.

Rocky Mountain Spotted Fever

Rocky Mountain spotted fever is caused by *Rickettsia rickettsii* and can be carried by several tick species. Despite its name, a majority of cases are found in the South Central United States; however, cases have been reported throughout the contiguous United States. The incubation period is 3 to 12 d, and symptoms include fever, headache, malaise, and myalgia. A characteristic maculopapular rash

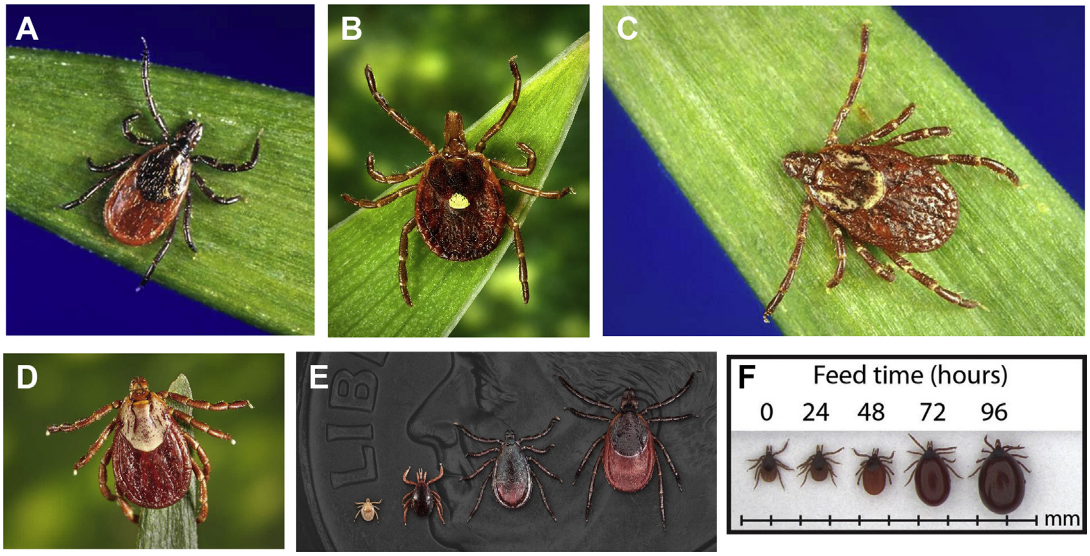

Figure 3. Medically important ticks found in the United States, including (A) *I scapularis*, (B) *A americanum*, (C) *D variabilis*, and (D) *D andersoni*. In addition to species identification, these ticks may present in different (E) life stages and (F) levels of engorgement (Centers for Disease Control and Prevention. Ticks. https://www.cdc.gov/ticks/index.html). Source: United States Centers for Disease Control and Prevention (public domain).

develops 2 to 5 d after the onset of symptoms and may progress to petechiae, heralding more severe disease. Doxycycline is first-line therapy, and delay in treatment beyond 5 d is associated with increased fatality rates.[28]

Tularemia

Tularemia is caused by the bacteria *Francisella tularensis* which is transmitted by several tick species, including *Dermacentor variabilis* (the American dog tick), *Dermacentor andersoni* (the Rocky Mountain wood tick), and *A americanum* (the lone star tick). Tularemia has been reported in all states except Hawaii. In contrast to most other TBIs, tularemia can be transmitted by other vectors, including deer flies and mammals. The incubation period is typically 3 to 5 d. Common symptoms include fever, headache, malaise, and myalgias. A cutaneous ulcer or eschar is classic for tularemia but is not always present. Ultimately, the clinical presentation is dependent on the route of inoculation. Treatment depends on the severity of illness. Doxycycline is first-line therapy for mild symptoms, and streptomycin is reserved for more severe illness.

Protozoa

Babesiosis

Babesiosis is caused most commonly by *Babesia microti* in the United States. Babesia are protozoan parasites of erythrocytes with a life cycle similar to malarial infections. Babesiosis is most frequently found in the Upper Midwest and Northeastern United States, although cases have also been reported on the West Coast. The incubation period is 1 to 9 wk, and symptoms include fever, rigors, headache, myalgias, dark urine, nausea, and diarrhea. First-line therapy is a combination of atovaquone and azithromycin.

Viruses

Colorado Tick Fever

Colorado tick fever is caused by a double-stranded RNA virus from the *Coltivirus* genus. It is transmitted by *D andersoni* (the Rocky Mountain wood tick) and is most commonly reported in Colorado, Utah, Montana, and Wyoming. The incubation period is 1 to 14 d, and early symptoms include fever (which is often biphasic), rash, and conjunctival injection. Prolonged weakness and fatigue are common in adults. Treatment is supportive.

Powassan Virus

Powassan, the only tick-borne encephalitis found in the United States, is caused by a flavivirus related to West Nile virus and dengue, and it is most commonly transmitted by *Ixodes* ticks (the blacklegged and groundhog ticks). The virus is rare, but it is most commonly reported in the Upper Midwest and Northeast. Incubation is 1 to 4 wk. Fever, headache, and vomiting are often followed by an encephalitis syndrome characterized by altered mental status,

Figure 4. Erythema migrans (EM) is classically described as a flat, blanchable, annular, erythematous skin lesion with or without central clearing, occurring at the site of *B burgdorferi* inoculation. The rash typically develops 3 to 30 d after exposure and can reach up to 30 cm in diameter (Centers for Disease Control and Prevention. Lyme disease. https://www.cdc.gov/lyme/index.html) (A and B).[102] Atypical presentations include vesicular lesions (C), confluent erythematous lesions (D), urticarial lesions, transient EM, and disseminated EM (E), itself a feature of disseminated Lyme disease.[23] EM may be easily misdiagnosed in patients with darker skin (F).[103] Source: United States Centers for Disease Control and Prevention (public domain).

seizure, and localized weakness. The fatality rate approaches 10%. Treatment is supportive.

Other

Alpha-gal Syndrome

Alpha-gal syndrome is a transmissible hypersensitivity reaction most commonly spread by a bite from *A americanum* (the lone star tick) or *I scapularis* (the blacklegged tick). In this recently recognized syndrome,[29] the tick regurgitates the sugar molecule alpha-gal into its human host. In some individuals, the alpha-gal molecule induces a hypersensitivity to meat (and less frequently dairy), which can lead to a spectrum of allergic reactions from hives to anaphylaxis. In contrast to most food-based allergies, which manifest in minutes, the alpha-gal–induced reaction to meat can be delayed by several hours.

Recommendations for the Prevention and Management of TBIs in Resource-Limited Settings

Frontline providers may encounter TBIs in a variety of scenarios, ranging from the truly austere and remote to an office setting. Our recommendations assume that care is being provided with few resources and therefore focus on simple, medically relevant interventions that can be applied at the individual and community levels to prevent the transmission of TBIs from vector to human host. A cornerstone of preventing TBIs is avoiding tick bites themselves; in a 2019 systematic review, use of insect repellents and protective clothing was associated with lower incidences of Lyme disease when compared to other preventative strategies.[30] However, strategies such as tick checks, timely tick removal, prophylactic antibiotics, vaccination, and education are important in a multimodal strategy for TBI prevention (Table 3). Many large-scale tick control strategies such as vegetation management and host population management are outside of the scope of medical practice and therefore not addressed in this CPG.

LONG-SLEEVED CLOTHING

Although wearing long-sleeved clothing is recommended by the CDC to limit a tick's ability to latch,[31] this strategy has not been directly studied as a means to prevent TBI. However, the intervention is cheap, practical, and has minimal to no risk; therefore, the authors support the use of long-sleeved clothing when traveling in tick habitat.

Recommendation. Wear long-sleeved clothing when traveling in tick habitat. Recommendation grade: Expert opinion.

LIGHT-COLORED CLOTHING

Wearing light-colored clothing has been recommended to improve the visualization of ticks during tick checks, but this relationship has not been formally studied in TBI prevention. In a randomized, cross-over, cohort study of clothing color, the authors found that *Ixodes ricinus* may in fact be attracted to light-colored clothing.[32] In a subsequent case control study of risk factors associated with Lyme disease, the use of light-colored clothing was not associated with a reduced risk of Lyme disease.[33]

Recommendation. Although light-colored clothing may not reduce the risk of tick bites, it does make it easier to identify ticks on clothes during tick checks. Recommendation grade: 2C.

TICK AND INSECT REPELLENTS

Deet

N, N-Diethyl-meta-toluamide, also known as DEET, has been in use since 1956 and has been widely shown to be an effective tick repellent.[34–37] Seizure and neurotoxicity are possible side effects,[38] but with approximately 30% of the US population having used DEET at some point in their lives,[39] the absolute risk is likely quite low.

The concentration of DEET is associated with estimated protection time. For example, 30% DEET will protect the user for an estimated 6 h, whereas 5% DEET offers 2 h of protection. The American Academy of Pediatrics does not recommend the use of DEET in children 2 mo of age or younger, and DEET concentrations of 10% or less should be used in those 12 y of age and younger.[40] Additionally, DEET is known to be corrosive to synthetic or technical fabrics such as Gore-Tex.

Recommendation. DEET is an effective tick repellent. DEET should be reapplied based on the concentration of formulation. Recommendation grade: 1B.

Recommendation. DEET can be used in children over the age of 2 mo. Recommendation grade: 1B.

Picaridin

Picaridin (also known as icaridin) was developed in the 1980s as an alternative to DEET, and it became available in US markets in the mid-2000s. As with DEET, the degree of protection for picaridin is based on concentration[41]: 20% picaridin offers approximately 10 h of protection against arthropods. Although picaridin has a much shorter track record of use than DEET, it appears to be nontoxic and, when compared to DEET, has a superior safety profile.[42] Picaridin is also

Table 3. General tick-borne disease prevention strategies

Personal protection	Wearing long-sleeved shirts and pants, light-colored clothing
	Applying insecticides to skin and/or clothing
	Performing tick checks
	Bathing immediately after spending time outdoors
	Washing and drying clothing at high temperatures
	Prompt tick removal
Community interventions	Educational programs
Medical prevention	Vaccination
	Prophylactic antibiotics
Environmental strategies	Spraying acaricides
	Vegetation management (mowing, clearing leaf litter)
	Host management (fencing, hunting)

odorless and, in contrast to DEET, is not harmful to synthetic fabrics.

Recommendation. Picaridin is an effective tick repellent and is comparably efficacious to DEET. Recommendation grade: 1B.

Recommendation. Picaridin may have a superior safety profile when compared to DEET. Recommendation grade: 2B.

Permethrin

Permethrin is both a repellent and an insecticide that is impregnated into clothing. Permethrin-treated clothing has been thoroughly studied in a number of populations and has been shown to reduce the rate of tick attachment and tick bites in studies of volunteers,[43] military personnel,[44] and forestry workers.[45,46] However, head-to-head studies comparing permethrin to DEET or other repellents suffer from heterogeneous methodologies.[47,48] Overall, permethrin and DEET appear to be comparatively efficacious; the consumer must choose based on cost, relative risk of toxicity, and the timing of their potential exposure. When used in combination, permethrin-treated clothing and topical DEET were found to be more effective in preventing mosquito bites compared to either used alone.[49] Although not specifically studied for tick bite prevention, it stands to reason that the combination of permethrin-treated clothing and topical DEET (or picaridin) may be synergistic to reduce the risk of TBI.

Recommendation. Permethrin-treated clothing is an effective tick repellent. Recommendation grade: 1A.

Recommendation. Permethrin-treated clothing, when used in combination with a skin-based tick repellent such as DEET or picaridin, may further reduce the risk of TBI. Recommendation grade: 1C.

Other Repellents

Several essential oils, citriodiol (oil of eucalyptus), and IR3535 are also commercially available as tick repellents.[50] Others, such as nootkatone, are in development. A review of every available tick repellent is beyond the scope of this CPG. However, each of the aforementioned repellents shares a common trait of either lower repellent efficacy or a significantly shorter duration of action when compared to DEET.[51] Ideally a natural, effective, and nontoxic tick repellent will be available in the future; unfortunately, such a product is not currently available.

Recommendation. Essential oils, citriodiol, nootkatone, and IR3535 have either a lower repellent efficacy or significantly shorter duration of action. Given these attributes, wilderness recreationalists should avoid these products as first-line tick repellents. Recommendation grade: 2B.

TICK CHECKS

Another highly recommended method of personal protection against tick bites is tick checks. The tick check procedure is as follows[52]:

1. Remove clothing for overall visual inspection.
2. Systematically scan the body for ticks, paying special attention to warm places (armpits, knees, under underwear, around hairline of neck, ears, and navel).
3. Remove any identified ticks by grasping at the head of the tick with pointed tweezers or tick remover and pulling perpendicular to the skin (Figure 5).

The data supporting this behavior, however, are scant, and its efficacy has not been rigorously studied. In a study of various personal protective methods in definite, possible, and unlikely Lyme disease patients, no difference in tick check performance was found between patients with Lyme disease and controls, suggesting that inspection is not an effective strategy in preventing Lyme disease.[53] On the other hand, EM-free control subjects were found to be more likely to perform tick checks within 36 h and bathe within 2 h of being outdoors than those with EM, suggesting a protective effect of these behaviors.[54]

Recommendation. Evidence supporting tick checks is contradictory; however, when combined with bathing within 2 h of being outdoors, these measures may help prevent TBI. Recommendation grade: 1C.

Recommendation. Shower or bathe within 2 h of returning from tick habitat. Recommendation grade: 2C.

CLOTHING CARE

In an early study of *A americanum* and *I scapularis* survival after exposure to automatic washer and dryer conditions, all nymphs were killed after 1 h of drying at 40 to 42°C.[55] Although large proportions of nymphs survived hot water washes in this study, a more recent study showed 100% effectiveness of hot water washes at temperatures greater than 54°C (130°F) in killing both nymphal and adult *I scapularis*.[56] These studies did not look specifically at the effect of these interventions on the incidence of TBI, however.

Recommendation. Washing clothes at temperatures over 54°C/130°F and drying clothing in high heat for 10 min kills ticks and therefore may reduce the risk of TBIs. Recommendation grade: 1C.

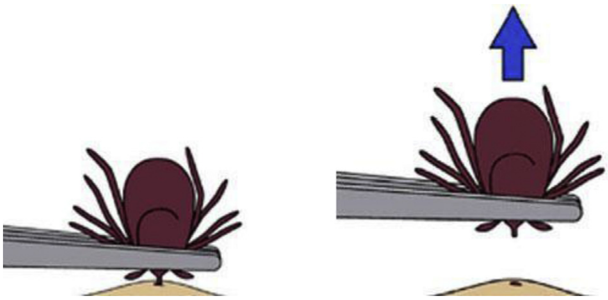

Figure 5. Forceps removal (Centers for Disease Control and Prevention. Ticks. https://www.cdc.gov/ticks/index.html). Source: United States Centers for Disease Control and Prevention (public domain).

OTHER BEHAVIORAL MODIFICATIONS

Although data are limited to support these practices, the CDC recommends several behaviors to avoid either primary contact with ticks or to limit tick latching.

Recommendation. When feasible, avoid areas with high grass or leaf litter. When in tick habitat, walk in the middle of trails to mitigate the chance of contact with ticks. Recommendation grade: Expert opinion.

TICK REMOVAL

A number of anecdotal methods for tick removal have been described,[57–64] inspiring the creation of several commercial devices that use different strategies in grasping and removing embedded ticks. Case series comparing various mechanical tick removal techniques, however, have yielded disparate results.[65–69] Data are mixed on whether the method of tick removal is associated with the occurrence of TBI.[70] However, in a case series, tick removal with forceps was associated with a lower rate of spirochetal and rickettsial infections.[71] Passive methods such as application of petroleum jelly, fingernail polish, 70% isopropyl alcohol, or a hot kitchen match,[67,72] local infiltration of anesthetics,[73] and administration of oral ivermectin[74] failed to exhibit any efficacy in encouraging tick detachment. Overall, mechanical removal is largely accepted by experts,[75] and forceps removal has been endorsed by the CDC (Figure 5)[8]; however, to date no professional organization has adopted strong practice guidelines regarding optimal tick removal techniques.[76]

Recommendation. Mechanical removal by pulling upward, or perpendicular to skin, directly on an embedded tick with forceps is the best currently available method. Mechanical removal using commercial devices may also work, but evidence suggesting superiority does not exist. Recommendation grade: 1C.

Recommendation. Pulling embedded ticks with straight, steady pressure is preferred over a twisting motion.[8] Recommendation grade: 1C.

Recommendation. Passive removal techniques, particularly chemical strategies that involve exposing attached ticks to petroleum jelly, fingernail polish, isopropyl alcohol, gasoline, or methylated spirits are ineffective and not recommended. Recommendation grade: 2C.

Recommendation. Using local or systemic medications such as locally infiltrated anesthetics or systemic ivermectin is not effective in removing or exterminating attached ticks. No evidence exists to suggest any benefit to these strategies. Recommendation grade: 2C.

TIMING OF TICK REMOVAL AND RISK OF LYME TRANSMISSION

Prophylactic strategies for Lyme disease have been modeled on the risk of infection after a tick bite relative to the length of time the tick is attached to its host. Generally, the risk of Lyme disease transmission is correlated with the duration of tick attachment. Although the risk of transmission of *B burgdorferi* after a tick bite in Lyme endemic regions is estimated to be 1 to 3%,[77] the risk increases to 20% when infected *I scapularis* ticks are attached longer than 72 h.[78] Early animal studies of *I scapularis* ticks infected with *B burgdorferi* suggested that at least 48 h of attachment were necessary for transmission of disease.[79–81] However, a recent review highlighted that Lyme transmission is possible within 24 h of attachment and that a definitive study describing a minimum tick attachment time for the transmission of Lyme in humans has not been published.[82] Until these data are available, the CDC recommends tick removal within 36 h of attachment to reduce the risk of Lyme disease.[8]

Recommendation. Once discovered, ticks should be removed as soon as possible. To meaningfully reduce the risk of Lyme disease, ticks should be removed within 36 h of attachment. Recommendation grade: 1B.

PROPHYLACTIC ANTIBIOTICS FOR LYME DISEASE

Efforts were made in the 1990s to determine whether prophylactic antibiotics could prevent the development of Lyme disease in patients after an *I scapularis* tick bite. In a randomized, placebo-controlled trial comparing a single 200 mg dose of doxycycline to placebo in patients who had been exposed to *I scapularis* for less than 72 h, 8 of 247 (3%) placebo patients developed EM, compared to 1 of 235 (0.4%) patients receiving doxycycline, a statistically significant reduction in EM in treated patients.[83] A more recent meta-analysis including 1082 patients revealed a 2% risk of Lyme disease in placebo patients versus a 0.2% risk of Lyme disease in those receiving prophylaxis, although the number needed to treat to prevent 1 case of Lyme disease was 49.[84] A more recent randomized controlled trial supports the conclusion that doxycycline prophylaxis can reduce the risk of Lyme borreliosis.[85] In their guidelines, the Infectious Disease Society of America recommends a single dose of doxycycline 200 mg within 72 h of tick removal for adults and children older than 8 years if the following high-risk criteria are met: (1) the tick bite was from an identified Ixodes vector species, (2) it occurred in an endemic area, and (3) the tick was attached for ≥36 h.[86]

Recommendation. A single dose of 200 mg doxycycline orally is recommended after a high-risk tick bite if given within 72 h to reduce the risk of Lyme disease. Recommendation grade: 1B.

Recommendation. If a provider is unable to identify the tick, or if the time of attachment is unknown, then a period of "watchful waiting" is recommended instead of prophylaxis. Should the patient develop fever, EM, or arthralgias within 30 d of the presumed tick bite, treatment with doxycycline can then be initiated. Recommendation grade: 1B.

PROPHYLACTIC ANTIBIOTICS FOR OTHER TBI

Currently, there are no compelling data to suggest that antibiotic prophylaxis is effective for any TBI other than Lyme disease.[8]

Recommendation. Providers should not employ prophylactic antibiotics for management of anaplasmosis, ehrlichiosis, Rocky Mountain spotted fever, relapsing fever, or any other TBI. Outside of high-risk Lyme disease exposures, prophylactic antibiotics are not indicated. Recommendation grade: 1C.

THE DECISION TO EVACUATE

The decision to evacuate versus expectant monitoring can be the most important decision the provider can make in resource-limited settings. This decision involves a careful balance of risks and benefits, including consideration of the possible harms of an inaccurate diagnosis and the potential for delayed care. No randomized controlled trials exist comparing field management to evacuation for patients with potential systemic TBI. Available cohort studies from the National Outdoor Leadership School describe a low incidence of TBI in the field, but in nearly all cases those with suspected TBI were evacuated.[87–89] This is appropriate because management of TBI requires accurate clinical and laboratory diagnosis, prompt treatment, and close follow-up. Life-threatening complications such as Lyme carditis could also potentially develop within the timeframe of a multiday backcountry trip. An algorithmic decision tree outlining the field management of tick bites is summarized in Figure 6.

Recommendation. Individuals who develop systemic or high-risk symptoms (fever, generalized rash, arthralgias, cranial nerve palsy, dyspnea, or syncope) related to a suspected TBI should be evacuated to a higher level of medical care. Recommendation grade: 1C.

Recommendation. Individuals who develop symptoms suggestive of Lyme carditis such as dyspnea, dizziness, or syncope should receive a screening ECG as soon as possible and would benefit from a thorough cardiovascular evaluation in an appropriate clinical setting. Recommendation grade: 1C.

VACCINATION

Vaccination strategies offer an attractive option for disease control both at the individual and the public health level. The efficacy of a tick-borne encephalitis vaccine, available in central Europe, Russia, and China, has been well documented.[90,91] Vaccine programs for TBIs in the United States, however, have had limited success. In 1999, 2 large randomized controlled trials of Lyme disease vaccines based on the *B burgdorferi* outer surface protein (OspA), both with and without adjuvant, found the vaccines to be highly efficacious in preventing Lyme disease in endemic populations.[90,92] These vaccines, however, were removed from the market in 2002 due to low sales related to the need for frequent boosters, musculoskeletal side effects, high cost, and litigation.[91]

Vaccine development continues despite these shortfalls. There is commercial and scientific interest in developing a vaccine that could protect against both North American and European *Borrelia* serotypes.[93] Although a candidate vaccine using fused OspA molecules is no longer in development,[94] other candidates involving other OspA and OspC antigens are currently under study.[95–98]

Recommendation. While no vaccine for tick-borne encephalitis is currently available in the United States, vaccines such as Encepur appear to be efficacious for inducing seroconversion against tick-borne encephalitis. Recommendation grade: 2A.

Recommendation. Although not currently available in the United States, Lyme vaccination is efficacious at reducing the risk of infection. Recommendation grade: 2A.

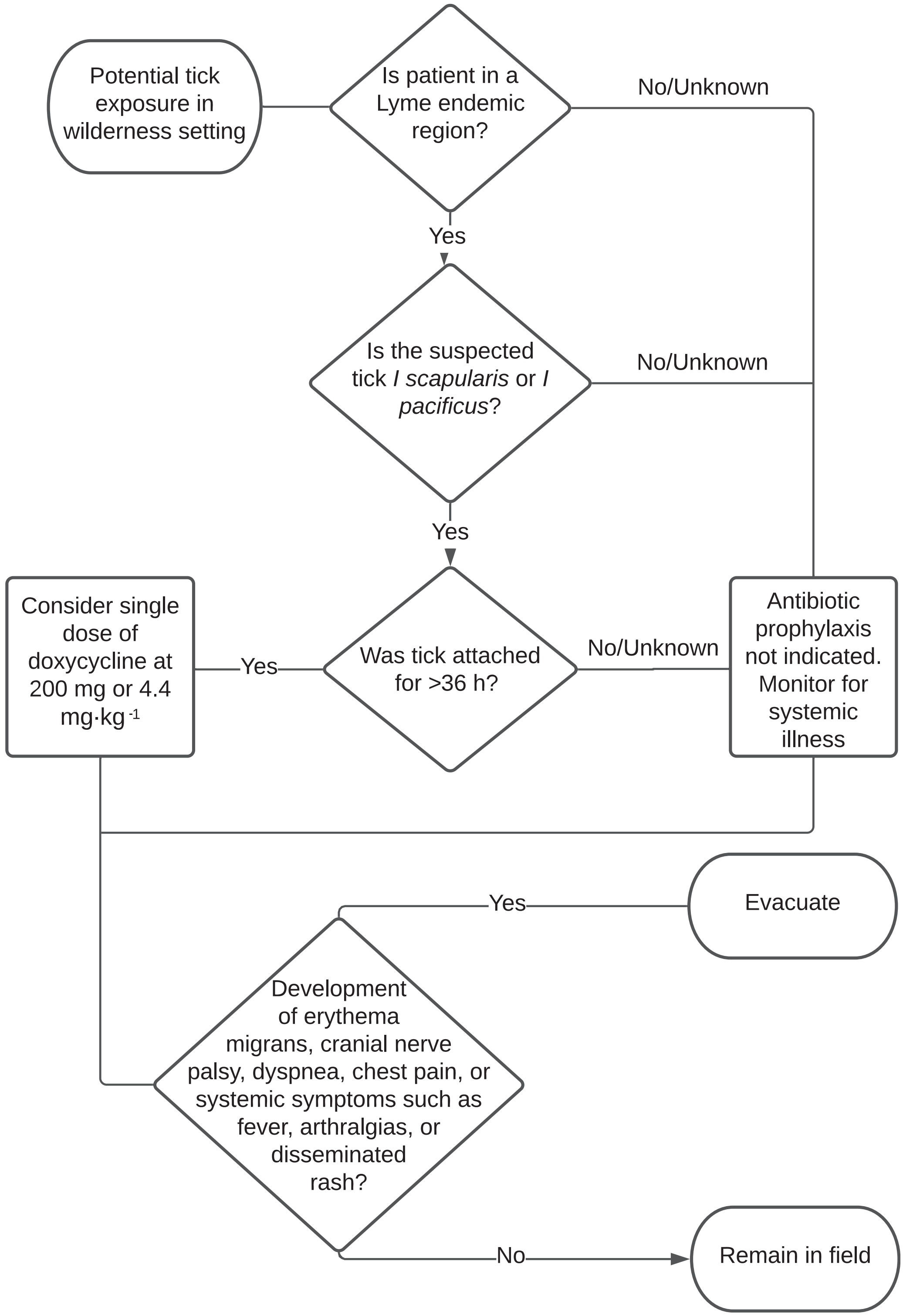

Figure 6. Wilderness management algorithm for tick-borne illness.

EDUCATIONAL PROGRAMS

TBI prevention behavior is inconsistent among individuals; in a cross-sectional study assessing knowledge, attitudes, and behaviors regarding TBI prevention strategies among persons living in Lyme-endemic areas, behaviors ranged widely from the use of tick control products on pets (83%) and tick checks (58%), to lower rates of compliance with showering or bathing after spending time outdoors (42%), applying insect repellents (31%), and using chemical or natural pesticides on yards (23 and 15%, respectively).[99] Simple educational programs, however, have been shown to be effective not only in changing behavior, but also in reducing disease burden. In a randomized controlled trial, individuals who received a 15-min Lyme disease and tick specific educational presentation were found to have lower rates of TBI compared to those receiving a control program.[100] Given the vast range of preventative behavior and the costs of TBIs to individuals and communities, programs such as these provide an effective and economical method for disease prevention.

Recommendation. Educational programs can change behavior and lower rates of TBI and should be encouraged. Recommendation grade: 1B.

Conclusion

TBI is a broad medical topic, influenced by the environment, geography, climate, ecology, and animal and human behavior. Given the interactions between humans and the environment, TBI cannot be completely avoided. However, with certain behavioral and medical adaptations, the overall burden of disease related to TBIs can be reduced. With a changing climate that continues to influence the epidemiology of TBI, promotion and strict adherence to simple prevention measures is important. The recommendations presented in this CPG are largely consistent with those presented by the CDC (https://www.cdc.gov/ticks/index.html) and other practice guidelines,[76] but they specifically highlight concepts most relevant to providers who encounter ticks in backcountry, austere, and limited-resource settings. Despite the limitations of the existing literature, these guidelines provide a starting point for front-line providers to mitigate the transmission and reduce the disease burden of TBIs through low-risk interventions.

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