TICK-BORNE DISEASES OF NORTH AMERICA

Dennis G. Maki, MD
Divisions of Infectious Diseases
and Pulmonary-Critical Care Medicine
Department of Medicine
UW School of Medicine and Public Health
dgmaki@medicine.wisc.edu
"He who studies medicine without books sails an uncharted sea, but he who studies medicine without patients does not go to sea at all."

William Osler

"Look wise, say nothing, and grunt. Speech was given to conceal thought."

William Osler
George Magnin, MD, FACP
“the internist’s internist”
TICK-BORNE DISEASES OF NORTH AMERICA

Goals -- to understand:

1. The ticks of North America that vector human diseases, their biology and epidemiology.
TICK-BORNE DISEASES OF NORTH AMERICA

Goals -- to understand:

1. The ticks of North America that vector human diseases, their biology and epidemiology.
2. Are tick-borne IDs on the rise? If so, why?
TICK-BORNE DISEASES OF NORTH AMERICA

Goals -- to understand:

1. The ticks of North America that vector human diseases, their biology and epidemiology.
2. Are tick-borne IDs on the rise? If so, why?
3. Prevention of tick-borne IDs.
TICK-BORNE DISEASES OF NORTH AMERICA

Goals -- to understand:

1. The ticks of North America that vector human diseases, their biology and epidemiology.
2. Are tick-borne IDs on the rise? If so, why?
3. Prevention of tick-borne IDs.
4. Major tick-borne IDs of North America (clinical features, diagnosis and treatment):
   - Lyme disease, STARI
   - Ehrlichiosis and Anaplasmosis
   - Babesiosis
   - Rocky Mountain Spotted Fever
   - Powassan Virus Fever
   - Colorado Tick Fever
   - Tularemia
   - Endemic Relapsing Fever
   - Tick Paralysis
NEW TICK-BORNE PATHOGENS IN NORTH AMERICA

*Borrelia miyamotoi*

*Borrelia mayonii*

Heartland Virus

*Rickettsia parkerii*

STARI agent
TICK-BORNE DISEASES OF NORTH AMERICA:

Are tick-borne IDs on the rise? If so, why?
Are Tick-borne Diseases on the Increase?
Are Tick-borne Diseases on the Increase?

Reported Cases of Anaplasmosis/Ehrlichiosis, Wisconsin, 1999 - 2014 (n=4,491)

*Total number of cases include confirmed and probable
Revised 4/3/2015
Are Tick-borne Diseases on the Increase?
Are Tick-borne Diseases on the Increase?
Are Tick-borne Diseases on the Increase?
Are Tick-borne Diseases on the Increase?

*Borrelia burgdorferi* infection rates in questing nymphs

State Average: 22%
Are Tick-borne Diseases on the Increase?

Anaplasma phagocytophilum Detected in I. scapularis adults, Wisconsin, 2011-2013 (N=542)

Borre

16/73 (22%)
1/18 (5.6%)
4/25 (16%)
3/48 (6.3%)
2/42 (4.8%)
3/9 (33%)
3/83 (3.9%)
8/65 (12%)
3/74 (4.1%)
2/9 (22%)
0/12
0/10
0/1
0/4
0/4
0/11
0/14
0/7
0/2

Maple hardwood (●)
Red Pine Stands (●)

Tick surveillance was not performed in all counties.
Are Tick-borne Diseases on the Increase?

**Babesia microti** Detected in *I. scapularis*, Wisconsin, 2011-2013

*Anaplasma phagocytophilum* qPCR positive
*I. scapularis* ticks (% infectivity)

- **Maple hardwood**: 
- **Red Pine Stands**:

- **16/73** (22%)
- **4/25** (16%)
- **1/18** (5.6%)
- **3/48** (6.3%)
- **2/42** (4.8%)
- **3/9** (33%)
- **3/83** (3.9%)

- **1/25** (4.0%)
- **2/73** (2.7%)
- **1/20** (5%)
- **2/48** (4.2%)

Tick surveillance was not performed in all counties.
IS THE INCIDENCE OF LYME DISEASE INCREASING?

- Incidence has increased >2-fold over past 25 years, now estimated ~300,000 cases/yr

*MMWR 2008, Emerg Infect Dis 2015*
Are Tick-borne Diseases on the Increase?
European-Asian Tick-borne Encephalitis
Are Tick-borne Diseases on the Increase? YES!
## FACTORS CONTRIBUTING TO EMERGENCE OF NEW INFECTIOUS DISEASES

<table>
<thead>
<tr>
<th>Categories</th>
<th>Specific examples</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Societal events</strong></td>
<td>War or civil conflict; population growth and migration; urban decay; economic impoverishment</td>
</tr>
<tr>
<td><strong>Health care</strong></td>
<td>Medical devices; organ transplantation; immunosuppression; widespread use of antibiotics</td>
</tr>
<tr>
<td><strong>Food production</strong></td>
<td>Globalization of food supplies; changes in food processing, packaging, and preparation</td>
</tr>
<tr>
<td><strong>Human behavior</strong></td>
<td>Sexual behavior; drug use; travel; diet; outdoor recreation; day-care for children; international travel</td>
</tr>
<tr>
<td><strong>Environmental changes</strong></td>
<td>Deforestation/reforestation; changes in water ecosystems; flood/drought; famine; global warming</td>
</tr>
<tr>
<td><strong>Public health infrastructure</strong></td>
<td>Curtailment or reduction of prevention programs; inadequate communicable disease surveillance; inadequate trained personnel</td>
</tr>
<tr>
<td><strong>Microbial adaptation and change</strong></td>
<td>Genuine <em>new pathogen</em> and no population immunity; changes in virulence; antiinfective drug resistance; Bioterrorism</td>
</tr>
</tbody>
</table>
## FACTORS CONTRIBUTING TO EMERGENCE OF NEW INFECTIOUS DISEASES

<table>
<thead>
<tr>
<th>Categories</th>
<th>Specific examples</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Societal events</strong></td>
<td>War or civil conflict; population growth and migration; urban decay; economic impoverishment</td>
</tr>
<tr>
<td><strong>Health care</strong></td>
<td>Medical devices; organ transplantation; immunosuppression; widespread use of antibiotics</td>
</tr>
<tr>
<td><strong>Food production</strong></td>
<td>Globalization of food supplies; changes in food processing, packaging, and preparation</td>
</tr>
<tr>
<td><strong>Human behavior</strong></td>
<td>Sexual behavior; drug use; travel; diet; outdoor recreation; day-care for children, international travel</td>
</tr>
<tr>
<td><strong>Environmental changes</strong></td>
<td>Deforestation/reforestation; changes in water ecosystems; urbanization; urbanization; global warming</td>
</tr>
</tbody>
</table>
Why are Tick-borne Diseases on the Increase?

- **Exposure:** expansion of population living in tick habitats and increased recreational activities in high risk areas
Why are Tick-borne Diseases on the Increase?

- **Exposure:** expansion of population living in tick habitats and increased recreational activities in high risk areas
URANIZATION OF AFRICA
URANIZATION OF AFRICA
URANIZATION OF NORTH AMERICA
URANIZATION OF NORTH AMERICA

- Lyme disease
- Ehrlichia
- Anaplasma
- Babesia
- Hanta virus
- West Nile Virus
Why are Tick-borne Diseases on the Increase?

- Exposure: expansion of population living in tick habitats and increased recreational activities in high risk areas
- Greatly increased deer populations?
LIFE CYCLE OF IXODES TICKS
LIFE CYCLE OF IXODES TICKS

1. Uninfected six-legged larva hatches from egg and develops
2. Larva feeds on small animal, becoming infected
3. Larva is dormant
4. Larva develops into eight-legged nymph
5. Nymph feeds on animal or human, transmitting infection
6. Nymph develops into adult tick
7. Adults feed on deer and mate
8. Female tick lays eggs

(a) The tick, *Ixodes scapularis*, has a two-year life cycle in which it requires three blood meals. The tick is infected by its first blood meal, and can pass on the infection to a human in its second.
Why are Tick-borne Diseases on the Increase?

• Exposure: expansion of population living in tick habitats and increased recreational activities in high risk areas

• Greatly increased deer populations?

• No, rapidly declining predator populations that feed on natural tick hosts with vastly increased populations of tick hosts, especially field mice

Why are Tick-borne Diseases on the Increase?

• Exposure: expansion of population living in tick habitats and increased recreational activities in high risk areas
• Greatly increased deer populations?
• No, rapidly declining predator populations that feed on natural tick hosts
• Transmission widely by migrating birds

Why are Tick-borne Diseases on the Increase?

- Exposure: expansion of population living in tick habitats and increased recreational activities in high risk areas
- Greatly increased deer populations?
- No, rapidly declining predator populations that feed on natural tick hosts
- Transmission widely by migrating birds
- **Climactic change, Global Warming**

Temperatures of the Last 10,000 Years
(Ice core data from Crete site in central Greenland)

- Holocene Climate Optimum
- Roman Climate Optimum
- Medieval Warm Period
- Modern Warm Period
- Little Ice Age
- Extent of Thermometer Record

- End of Last Ice Age
- Present Day

Daansgaard (1984), Avery (2009)
GLOBAL WARMING
GLOBAL WARMING: THE AGE OF THE INSECT
Figure 1: Schematic summary of main pathways by which climate change affects population health

- **Mitigation**
  - Anthropogenic greenhouse gas emissions
    - Changes in mean climatic conditions and variability:
      - temperature
      - precipitation
      - humidity
      - wind patterns

- **Adaptation**
  - Health effects
    - Thermal stress: deaths, illness
    - Injury/death from floods, storms, cyclones, bushfires
    - Effect of these events on food yields
  - Environmental effects
    - Extreme weather events:
      - frequency
      - severity
      - geography
    - Effects on ecosystems:
      - (land and sea), and on particular species
    - Sea-level rise:
      - salination of coastal land and freshwater; storm surges
    - Environmental degradation:
      - land, coastal ecosystems, fisheries
  - Microbial proliferation:
    - Food poisoning—Salmonella spp, etc; unsafe drinking water
  - Changes in vector-pathogen-host relations and in infectious disease geography/seasonality—e.g., malaria, dengue, tickborne viral disease, schistosomiasis
  - Impaired crop, livestock and fisheries yields, leading to impaired nutrition, health, survival
  - Loss of livelihoods, displacement, leading to poverty and adverse health: mental health, infectious diseases, malnutrition, physical risks
Risk of Malaria Transmission - This map displays the projected risk of malaria transmission in the year 2020, compared with the average risk in the years 1961 to 1990. This projection assumes a global temperature increase of 2°F and no human efforts to contain the spread of malaria. Source: Pim Martens, Maastricht University
Why are Tick-borne Diseases on the Increase?

- Exposure: expansion of population living in tick habitats and increased recreational activities in high risk areas
- Greatly increased deer populations?
- No, rapidly declining predator populations that feed on natural tick hosts
- Transmission widely by migrating birds
- Climactic change, Global Warming
TICK-BORNE DISEASES OF NORTH AMERICA

The Ticks of North America
THE VIRULENT TICKS OF THE UPPER MIDWEST

16 known species of ticks in Wisconsin, 5 species vector most of the tick-borne disease seen in the upper Midwest:

• *Ixodes scapularis* (the Deer tick)
  - *Borrelia burgdorferi*, *Borrelia miyamotoi*
  - *Ehrlichia muris*-like
  - *Anaplasmosis phagocytophilum*
  - *Babesia microti*
  - Powassan Fever Virus
THE VIRULENT TICKS OF THE UPPER MIDWEST

16 known species of ticks in Wisconsin, 5 species vector most of the tick-borne disease seen in the upper Midwest:

- *Ixodes scapularis* (the Deer tick)
  - *Borrelia burgdorferi, Borrelia miyamotoi*
  - *Ehrlichia muris-like*
  - *Anaplasmosis phagocytophilum*
  - *Babesia microti*
  - Powassan Fever Virus

- *Dermacentor variabilis* (Wood tick, American Dog tick)
  - Rocky Mt Spotted Fever
  - *Francicella tularensis*
  - Tick paralysis
  - Powassan Fever Virus
THE VIRULENT TICKS OF THE UPPER MIDWEST

16 known species of ticks in Wisconsin, 5 species vector most of the tick-borne disease seen in the upper Midwest:

• *Ixodes scapularis* (the Deer tick)
  - *Borrelia burgdorferi*, *Borrelia miyamotoi*
  - *Ehrlichia muris*-like
  - *Anaplasmosis phagocytophilum*
  - *Babesia microti*
  - Powassan Fever Virus

• *Dermacentor variabilis* (Wood tick, American Dog tick)
  - Rocky Mt Spotted Fever
  - *Francisella tularensis*
  - Tick paralysis

• *Rhipicephalus sanguineus* (Brown Dog tick)
  - *Ehrlichia chafeensis*
  - Rocky Mt Spotted Fever
  - *Bartonella henselae*
THE VIRULENT TICKS OF THE UPPER MIDWEST

16 known species of ticks in Wisconsin, 5 species vector most of the tick-borne disease seen in the upper Midwest:

- **Ixodes scapularis** (the Deer tick)
  - Borrelia burgdorferi, Borrelia miyamotoi
  - *Ehrlichia muris*-like
  - Anaplasmosis phagocytophilum
  - Babesia microti
  - Powassan Fever Virus

- **Dermacentor variabilis** (Wood tick, American Dog tick)
  - Rocky Mt Spotted Fever
  - *Francicella tularensis*
  - Tick paralysis

- **Rhipicephalus sanguineus** (Brown Dog tick)
  - *Ehrlichia chafeensis*
  - Rocky Mt Spotted Fever
  - *Bartonella henselae*

- **Amblyomma americanum** (Lone Star tick)
  - *Ehrlichia chafeensis*
  - *Francicella tularensis*
  - *Coxiella burnetti* (Q Fever)
THE VIRULENT TICKS OF THE UPPER MIDWEST

16 known species of ticks in Wisconsin, 5 species vector most of the tick-borne disease seen in the upper Midwest:

- **Ixodes scapularis** (the Deer tick)
  - *Borrelia burgdorferi*, *Borrelia miyamotoi*
  - *Ehrlichia muris*-like
  - *Anaplasmosis phagocytophilum*
  - *Babesia microti*
  - Powassan Fever Virus

- **Dermacentor variabilis** (Wood tick, American Dog tick)
  - Rocky Mt Spotted Fever
  - *Francicella tularensis*
  - Tick paralysis

- **Rhipicephalus sanguineus** (Brown Dog tick)
  - *Ehrlichia chafeensis*
  - Rocky Mt Spotted Fever
  - *Bartonella henselae*

- **Amblyomma americanum** (Lone Star tick)
  - *Ehrlichia chafeensis*
  - *Francicella tularensis*
  - *Coxiella burnetti* (Q Fever)

- **Ixodes marxi** (Squirrel tick)
  - Powassan Fever Virus
THE VIRULENT WISCONSIN TICKS

Blacklegged Tick (*Ixodes scapularis*)
- adult female
- adult male
- nymph
- larva

Lone Star Tick (*Amblyomma americanum*)

Dog Tick (*Dermacentor variabilis*)
Prevention of tick-borne IDs
PREVENTION OF TICK-BORNE DISEASE

Tick avoidance.

- If possible, avoid wooded and bushy areas or at least stay in the center of a cleared trail to avoid questing ticks.
PREVENTION OF TICK-BORNE DISEASE

Tick avoidance

• If possible, avoid wooded and bushy areas or at least stay in the center of a cleared trail to avoid questing ticks.

Personal protection

• Wear long sleeves, long pants and tuck shirts into pants and pants into shoes or socks to keep ticks on the outside of clothing.
• Use effective tick repellents (20-30% DEET) on exposed skin and for hiking, camping, fishing or hunting in high-risk areas between April and October, consider using permethrin-impregnated outer clothing or sprayed on. Tumble clothes in a dryer on high heat to kill remaining ticks.
• Daily tick checks on family members and dogs after being outdoors where ticks are present.
• Attached ticks should be immediately removed with fine-tipped tweezers, grasping the tick as close to the skin as possible.
• Lyme Vaccine…oh I forgot.
PREVENTION OF TICK-BORNE DISEASE

Tick avoidance
- If possible, avoid wooded and bushy areas or at least stay in the center of a cleared trail to avoid questing ticks.

Personal protection
- Wear long sleeves, long pants and tuck shirts into pants and pants into shoes or socks to keep ticks on the outside of clothing.
- _Use effective tick repellents_ (20-30% DEET) on exposed skin and for hiking, camping, fishing or hunting in high-risk areas between April and October, consider using permethrin-impregnated outer clothing or sprayed on. Tumble clothes in a dryer on high heat to kill remaining ticks.
- Daily tick checks on family members and dogs after being outdoors where ticks are present.
- Attached ticks should be immediately removed with fine-tipped tweezers, grasping the tick as close to the skin as possible.
- Lyme Vaccine…oh I forgot.

Tick Control
- Create tick-safe zones around homes, parks, and recreational areas. Clear overgrown grass, brush, and leaf litter from the premises or trails. Use wood chips or gravel as a barrier between lawns and wooded areas.
TICK-BORNE DISEASES
OF NORTH AMERICA

The Tick-borne IDs of North America
TICK-BORNE DISEASES OF NORTH AMERICA

Lyme disease
Ehrlichiosis and Anaplasmosis
Babesiosis
Rocky Mountain Spotted Fever
Powassan Virus Fever
Colorado Tick Fever
Tularemia
Endemic Relapsing Fever
Tick Paralysis
RECOGNITION OF TIC(K)-BORNE INFECTIONS
SEVERE FEBRILE ILLNESS

A previously-well 26-year-old Wisconsin milk truck driver has had a 3-day history of fever, chills, severe headache and profound myalgias.

110/70 108 24 41.0°C. He is a muscular, acutely-ill young white male who is anxious but lucid. Scattered petechiae are seen over the trunk. There is shotty lymphadenopathy. Throat is beefy red but there is no exudate. Neck is supple. There is mild tenderness to palpation over all muscle groups. Chest is clear. Heart is regular with a grade I SEM. He is intact neurologically.

Hematocrit 46. WBC 3.4 with 32% band forms and Doehle bodies. Platelets 64K. AST 105. Chest x-ray clear.
RECOGNITION OF TIX(C)K-BORNE INFECTIONS

Quo Vene?
WEAKNESS WITH SYNCOPE

A 49 year-old farmer with hypertension and a 50 pack-year smoking history is brought to the hospital following a syncopal episode. He has been well other than a bout of painless cellulitis over his shoulder associated with lowgrade headache and transient fever 6 weeks earlier, treated with cephalexin.

HR 32, temperature 36.8°C. He is obese but other than anxiety looks well. There are no skin lesions. Exam is unremarkable other than bradycardia.

WBC 7.4, CRP 3.5. Troponin 0.01.
**LYME DISEASE**

**AGENT:** *Borrelia burgdorferi*, *B mayonii* (*B afzenii, B garanii* … in Europe)

**EPIDEMIOLOGY:** *Ixodes scapularis* (deer tick)…*Ixodes pacificus* (lone star tick)

→ Tickbites (no transfusion-related; rare transplant transmissions)

**INCUBATION PERIOD:**

*Early Localized or Early Disseminated:* ~1 wk–2 mo

*Late:* ~months–years
LYME DISEASE

<table>
<thead>
<tr>
<th>AGENT:</th>
<th>Borrelia burgdorferi, B mayonii (B afzenii, B garanii … in Europe)</th>
</tr>
</thead>
<tbody>
<tr>
<td>EPIDEMIOLOGY:</td>
<td><em>Ixodes scapularis</em> (deer tick)…<em>Ixodes pacificus</em> (lone star tick) → Tickbites (no transfusion-related; rare transplant transmissions)</td>
</tr>
</tbody>
</table>
| INCUBATION PERIOD: | *Early Localized or Early Disseminated:* ~1 wk–2 mo  
*Late:* ~months–years |
| SYNDROME:       | *Early Localized:* Asymptomatic vs  
EM (80%), HA, myalgias…..*NO GI or RT symptoms* |
**LYME DISEASE**

<table>
<thead>
<tr>
<th>AGENT:</th>
<th>Borrelia burgdorferi, B mayonii (B afzenii, B garanii … in Europe)</th>
</tr>
</thead>
<tbody>
<tr>
<td>EPIDEMIOLOGY:</td>
<td><em>Ixodes scapularis</em> (deer tick)…<em>Ixodes pacificus</em> (lone star tick) → Tickbites (no transfusion-related; rare transplant transmissions)</td>
</tr>
</tbody>
</table>
| INCUBATION PERIOD: | **Early Localized or Early Disseminated:** ~1 wk–2 mo  
**Late:** ~months–years |
| SYNDROME:       | **Early Localized:** Asymptomatic vs  
*EM (80%), HA, myalgias…..NO GI or RT symptoms*  
**Early Disseminated:** Like early localized + Multiple EM common |
LYME DISEASE

AGENT:  
*Borrelia burgdorferi*, *B mayonii* (*B afzenii*, *B garanii* … in Europe)

EPIDEMIOLOGY:  
*Ixodes scapularis* (deer tick)…*Ixodes pacificus* (lone star tick)  
→ Tickbites (no transfusion-related; rare transplant transmissions)

INCUBATION PERIOD:  
*Early Localized or Early Disseminated*: ~1 wk–2 mo  
*Late*: ~months–years

SYNDROME:  
*Early Localized*: Asymptomatic vs  
EM (80%), HA, myalgias…..NO GI or RT symptoms  
*Early Disseminated*: Like early localized + Multiple EM common  
- Polyarthralgias of major joints, fever  
- Aseptic meningitis, cranial neuropathy, cervical radiculitis  
- AV nodal heart block (transient), mild carditis

![ECG](image-url)
## LYME DISEASE

<table>
<thead>
<tr>
<th><strong>AGENT:</strong></th>
<th><em>Borrelia burgdorferi</em>, <em>B mayonii (B afzenii, B garanii ... in Europe)</em></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>EPIDEMIOLOGY:</strong></td>
<td><em>Ixodes scapularis</em> (deer tick)...<em>Ixodes pacificus</em> (lone star tick) → Tickbites (no transfusion-related; rare transplant transmissions)</td>
</tr>
</tbody>
</table>
| **INCUBATION PERIOD:** | *Early Localized or Early Disseminated:* ~1 wk–2 mo  
*Late:* ~months–years |
| **SYNDROME:** | *Early Localized:* Asymptomatic vs EM (80%), HA, myalgias.....*NO GI or RT symptoms*  
*Early Disseminated:* Like early localized + Multiple EM common  
- Polyarthralgias of major joints, fever  
- Aseptic meningitis, cranial neuropathy, cervical radiculitis  
- AV nodal heart block (transient), mild carditis  
*Late:* Oligoarthritis major joint (knee),...encephalitis, paraparesis... |
# LYME DISEASE

| **AGENT:** | *Borrelia burgdorferi*, *B mayonii* (*B afzenii, B garanii* ... in Europe) |
| **EPIDEMIOLOGY:** | *Ixodes scapularis* (deer tick)...*Ixodes pacificus* (lone star tick)  
→ Tickbites (no transfusion-related; rare transplant transmissions) |
| **INCUBATION PERIOD:** | *Early Localized or Early Disseminated:* ~1 wk–2 mo  
*Late:* ~months–years |
| **SYNDROME:** | *Early Localized:* Asymptomatic vs  
EM (80%), HA, myalgias.....**NO GI or RT symptoms**  
*Early Disseminated:* Like early localized + Multiple EM common  
☐ Polyarthritis of major joints, fever  
☐ Aseptic meningitis, cranial neuropathy, cervical radiculitis  
☐ AV nodal heart block (transient), mild carditis  
*Late:* Oligoarthritis major joint (knee),...encephalitis, paraparesis...  
**Post-Lyme Disease Syndrome:** Fatigue, myalgias, cognitive >6 mo |
# Lyme Disease

<table>
<thead>
<tr>
<th><strong>AGENT:</strong></th>
<th>Borrelia burgdorferi, B mayonii (B afzenii, B garanii ... in Europe)</th>
</tr>
</thead>
</table>
| **EPIDEMIOLOGY:** | *Ixodes scapularis* (deer tick)...*Ixodes pacificus* (lone star tick)  
Tickbites (no transfusion-related; rare transplant transmissions) |
| **INCUBATION PERIOD:** | *Early Localized or Early Disseminated:* ~1 wk–2 mo  
*Late:* ~months–years |
| **SYNDROME:** | *Early Localized:* Asymptomatic vs  
*EM (80%), HA, myalgias.....NO GI or RT symptoms*  
*Early Disseminated:* Like early localized + Multiple EM common  
☑️ Polyarthritis of major joints, fever  
☑️ Aseptic meningitis, cranial neuropathy, cervical radiculitis  
☑️ AV nodal heart block (transient), mild carditis  
*Late:* Oligoarthritis major joint (knee),...encephalitis, paraparesis...  
*Post-Lyme Disease Syndrome:* Fatigue, myalgias, cognitive >6 mo |
| **DIAGNOSIS:** | Serologic (EIA ➔ confirm by Western Blot)  
PCR blood, joint fluid or EM aspirate  
CONSIDER CO-INFECTION *(Ehrlichia/Anaplasma and Babesia)* |
High frequency of false positive IgM immunoblots for *Borrelia burgdorferi* in Clinical Practice

Y. Seriburi, N. Ndulwe, Z. Chang, M. E. Cox and G. P. Wormser
Division of Infectious Diseases, New York Medical College, Valhalla, NY, USA

*Clin Microbiol Infect* 2012; 18: 1236–1240

Abstract

Although it is known that two-tier serologic testing for Lyme disease may be associated with false positive results on the IgM immunoblot, this problem has never been systematically studied in the clinical practice setting. In a retrospective investigation of patients referred to the private adult practice of an Infectious Diseases physician for possible Lyme disease, 50 of 182 patients (27.5%, 95% CI 22.6–32.4%) had at least one positive IgM immunoblot. Of those patients, 31 (62%) had no tick exposure, no EM history and no symptoms for many months. Testing done in mid-winter.

- Screening EIA NEG
- IgG Immunoblot NEG and on repeat months later
- No tick exposure
- No EM history and asymptomatic or symptoms for many months
- Testing done in mid-winter
FALSE-POSITIVE IgM IMMUNOBLOTS (WITH NEGATIVE IgG IMMUNOBLOT)

- Screening EIA NEG
- IgG Immunoblot NEG and on repeat months later
- No tick exposure
- No EM history and asymptomatic or symptoms for many months
- Testing done mid-winter
# Lyme Disease

**Agent:** Borrelia burgdorferi, B mayonii (B afzenii, B garanii … in Europe)

**Epidemiology:**
- *Ixodes scapularis* (deer tick)…*Ixodes pacificus* (lone star tick)
- Tickbites (no transfusion-related; rare transplant transmissions)

**Incubation Period:**
- **Early Localized or Early Disseminated:** ~1 wk–2 mo
- **Late:** ~months–years

**Syndrome:**
- **Early Localized:** Asymptomatic vs EM (80%), HA, myalgias…..NO GI or RT symptoms
- **Early Disseminated:** Like early localized + Multiple EM common
  - Polyarthralgias of major joints, fever
  - Aseptic meningitis, cranial neuropathy, cervical radiculitis
  - AV nodal heart block (transient), mild carditis
- **Late:** Oligoarthritis major joint (knee),….encephalitis, paraparesis…

**Post-Lyme Disease Syndrome:** Fatigue, myalgias, cognitive >6 mo

**Diagnosis:**
- Serologic (Western Blot)
- PCR blood, joint fluid or EM aspirate
- **Consider Co-Infection** (Ehrlichia/Anaplasma and Babesia)

**Treatment:**
- **Local, Local Disseminated:** Doxycycline (14-21d) > Amox > Cefurox
- **Late Disseminated, Late:** Ceftriaxone 2 g/d (28d) or Doxycycline (14-21d)
ERYTHEMA MIGRANS
IMPORTANT ISSUES IN LYME DISEASE

• Is there co-infection with Lyme Disease? **YES.**
  In up 5-25% of cases in some areas, 2-12% with Ehrlichia/Anaplasma in Wisconsin, more often Babesia in New England

• Is there sexual transmission of *B burgdorferi*? **NO**

• Does vertical transmission with fetal infection and adverse pregnancy outcome occur? **NO**
  

• Does reinfection occur with *B burgdorferi*? **YES**
  

• Post-Lyme Disease Syndrome: **IDSA and Ad Hoc International Lyme Disease Group DEFINITION:**
  
  1. Documented and appropriately treated Lyme disease.
IMPORTANT ISSUES IN LYME DISEASE

• Is there co-infection with Lyme Disease? **YES.**

  In up 5-25% of cases in some areas, 2-12% with Ehrlichia/Anaplasma in Wisconsin, up to 50%, including with Babesia, in New England and parts of Europe
IMPORTANT ISSUES IN LYME DISEASE

• Is there co-infection with Lyme Disease? **YES.**
  In up 5-25% of cases in some areas, 2-12% with Ehrlichia/Anaplasma in Wisconsin, more often Babesia in New England

• Is there sexual transmission of *B burgdorferi*? **NO**
IMPORTANT ISSUES IN LYME DISEASE

- Is there co-infection with Lyme Disease? **YES.**
  - In up 5-25% of cases in some areas, 2-12% with Ehrlichia/Anaplasma in Wisconsin, more often Babesia in New England
- Is there sexual transmission of *B burgdorferi*? **NO**

**Does vertical transmission with fetal infection and adverse pregnancy outcome occur? **NO**

*Clin Infect Dis 1996; Am J Ob Gyn 1993, 1999*
IMPORTANT ISSUES IN LYME DISEASE

- Is there co-infection with Lyme Disease? **YES.**
  
  In up 5-25% of cases in some areas, 2-12% with Ehrlichia/Anaplasma in Wisconsin, more often Babesia in New England

- Is there sexual transmission of *B burgdorferi*? **NO**

- Does vertical transmission with fetal infection and adverse pregnancy outcome occur? **NO**
  
  *Clin Infect Dis 1996; Am J Ob Gyn 1993, 1999*

- Does reinfection occur with *B burgdorferi*? **YES**

  *Am J Trop Med Hyg 2006*

  *Clin Infect Dis 2007*

IMPORTANT ISSUES IN LYME DISEASE

• Is there co-infection with lyme Disease? YES.
  In up 5-25% of cases in some areas, 2-12% with Ehrlichia/Anaplasma in Wisconsin, more often Babesia in New England

• Is there sexual transmission of *B burgdorferi*? NO

• Does vertical transmission with fetal infection and adverse pregnancy outcome occur? NO
  *Clin Infect Dis 1996; Am J Ob Gyn 1993, 1999*

• Does reinfection occur with *B burgdorferi*? YES
  *Am J Trop Med Hyg 2006; Clin Infect Dis 2007*

• Post-Lyme Disease Syndrome: *IDSA and Ad Hoc International Lyme Disease Group DEFINITION:*

  1. Documented and appropriately treated Lyme disease.
  2. Unrelieved symptoms from therapy persist >6 mo: fatigue, generalized pain and cognitive complaints.
IMPORTANT ISSUES IN LYME DISEASE

- Is there co-infection with Lyme Disease? **YES.**
  - In up 5-25% of cases in some areas, 2-12% with Ehrlichia/Anaplasma in Wisconsin, more often Babesia in New England
- Is there sexual transmission of *B. burgdorferi*? **NO**
- Does vertical transmission with fetal infection and adverse pregnancy outcome occur? **NO**
- Does reinfection occur with *B. burgdorferi*? **YES**
- Post-Lyme Disease Syndrome: **IDSA and Ad Hoc International Lyme Disease Group DEFINITION:**
  1. Documented and appropriately treated Lyme disease.
  2. Unrelieved symptoms from therapy persist >6 mo.

- For patients with proven Lyme disease who remain persistently symptomatic following treatment, is a more prolonged course of treatment beneficial? **NO**
  - Multiple double-blinded RCTs have shown no benefit:
IMPORTANT ISSUES IN LYME DISEASE

- Is there co-infection with Lyme Disease? **YES.**
  - In up 5-25% of cases in some areas, 2-12% with Ehrlichia/Anaplasma in Wisconsin, more often Babesia in New England
- Is there sexual transmission of *B. burgdorferi*? **NO**
- Does vertical transmission with fetal infection and adverse pregnancy outcome occur? **NO**
- Does reinfection occur with *B. burgdorferi*? **YES**
- Post-Lyme Disease Syndrome: **IDSA and Ad Hoc International Lyme Disease Group DEFINITION:**
  1. Documented and appropriately treated Lyme disease.
  2. Unrelieved symptoms from therapy persist >6 mo.
- For patients with proven Lyme disease who remain persistently symptomatic following treatment, is a more prolonged course of treatment beneficial? **NO**
  - Multiple double-blinded RCTs have shown no benefit:

- Is there chronic Lyme Disease? **PROBABLY...**
**IMPORTANT ISSUES IN LYME DISEASE**

- Is there co-infection with lyme Disease? **YES**.
  
  In up 5-25% of cases in some areas, 2-12% with Ehrlichia/Anaplasma in Wisconsin, more often Babesia in New England

- Is there sexual transmission of *B burgdorferi*? **NO**

- Does vertical transmission with fetal infection and adverse pregnancy outcome occur? **NO**
  
  *Clin Infect Dis 1996; Am J Ob Gyn 1993, 1999*

- Does reinfection occur with *B burgdorferi*? **YES**
  
  *Am J Trop Med Hyg 2006; Clin Infect Dis 2007*

- Post-Lyme Disease Syndrome: *IDSA and Ad Hoc International Lyme Disease Group DEFINITION:*
  
  1. Documented and appropriately treated Lyme disease.
  2. Unrelieved symptoms from therapy persist >6 mo.

- For patients with proven Lyme disease who remain persistently symptomatic following treatment, is a more prolonged course of treatment beneficial? **NO**
  
  Multiple double-blinded RCTs have shown no benefit:
  

- Is there chronic Lyme Disease? **PROBABLY...BUT VERY RARE AFTER APPROPRIATE THERAPY, IF IT OCCURS AT ALL...**
IMPORTANT ISSUES IN LYME DISEASE

- Is there co-infection with Lyme Disease? **YES.**
  In up 5-25% of cases in some areas, 2-12% with Ehrlichia/Anaplasma in Wisconsin, more often Babesia in New England

- Is there sexual transmission of *B. burgdorferi*? **NO**

- Does vertical transmission with fetal infection and adverse pregnancy outcome occur? **NO**


- Does reinfection occur with *B. burgdorferi*? **YES**


- Post-Lyme Disease Syndrome: *IDSA and Ad Hoc International Lyme Disease Group DEFINITION:*
  1. Documented and appropriately treated Lyme disease.
  2. Unrelieved symptoms from therapy persist >6 mo.

- For patients with proven Lyme disease who remain persistently symptomatic following treatment, is a more prolonged course of treatment beneficial? **NO**

  - Multiple double-blinded RCTs have shown no benefit:

- Is there chronic Lyme Disease? **POSSIBLY.**

- Vaccine? **YES, but we blew it.**
A TICKBITE

An alarmed attorney calls to report he just found a feeding tick attached to the back of his 17 year-old daughter’s neck and wants to know what needs to be done, and immediately.
IMPORTANT ISSUES IN LYME DISEASE

- Is there co-infection with lyme Disease? **YES.**
  In up 5-25% of cases in some areas, 2-12% with Ehrlichia/Anaplasma in Wisconsin, more often Babesia in New England.

- Is there sexual transmission of *B. burgdorferi*? **NO**

- Does vertical transmission with fetal infection and adverse pregnancy outcome occur? **NO**

- Does reinfection occur with *B. burgdorferi*? **YES**

- Post-Lyme Disease Syndrome: **IDSA and Ad Hoc International Lyme Disease Group DEFINITION:**
  1. Documented and appropriately treated Lyme disease.
  2. Unrelieved symptoms from therapy persist >6 mo.

- For patients with proven Lyme disease who remain persistently symptomatic following treatment, is a more prolonged course of treatment beneficial? **NO**
  Multiple double-blinded RCTs have shown no benefit:

- Is there chronic Lyme Disease? **POSSIBLY.**
- Vaccine? **YES, but we blew it.**

- Does prophylaxis after tick bites prevent Lyme disease? **YES, IDSA Criteria for Prophylaxis:**
  1) *Ixodes scapularis* tick;
  2) Has fed >36h (engorged);
  3) >20% ticks in area infected;
  4) No contraindications to give doxycycline. **Clin Infect Dis 2006, 2016**
RANDOMIZED TRIAL OF A SINGLE DOSE OF DOXYCYCLINE FOR PREVENTION OF LYME DISEASE AFTER AN *IXODES SCAPULARIS* TICK BITE

<table>
<thead>
<tr>
<th></th>
<th>Placebo</th>
<th>Doxycyline 200 mg x1</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. patients treated</td>
<td>247</td>
<td>235</td>
</tr>
<tr>
<td>No developing EM at site</td>
<td>8</td>
<td>1</td>
</tr>
<tr>
<td>3.2%</td>
<td>0.4%</td>
<td></td>
</tr>
</tbody>
</table>

Nadelman et al.  
*P <0.04*
# LYME DISEASE

<table>
<thead>
<tr>
<th><strong>AGENT:</strong></th>
<th><em>Borrelia burgdorferi</em>, <em>B mayonii</em> (<em>B afzenii</em>, <em>B garanii</em> … in Europe)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>EPIDEMIOLOGY:</strong></td>
<td><em>Ixodes scapularis</em> (deer tick; field mice), <em>Ixodes pacificus</em> (lone star tick) → Tickbites (no transfusion-related; rare transplant transmissions)</td>
</tr>
</tbody>
</table>
| **INCUBATION PERIOD:** | *Early Localized or Early Disseminated:* ~1 wk–2 mo  
*Late:* ~months–years |
| **SYNDROME:** | *Early Localized:* Asymptomatic vs  
EM (80%), HA, myalgias…..NO GI or RT symptoms  
*Early Disseminated:* Like early localized + Multiple EM common  
☑ Polyarthritis of major joints, fever  
☑ Aseptic meningitis, cranial neuropathy, cervical radiculitis  
☑ AV nodal heart block (transient), mild carditis  
*Late:* Oligoarthritis major joint (knee),...encephalitis, paraparesis…  
*Post-Lyme Disease Syndrome:* Fatigue, myalgias, cognitive >6 mo |
| **DIAGNOSIS:** | Serologic (Western Blot)  
PCR blood, joint fluid or EM aspirate  
CONSIDER CO-INFECTION (Ehrlichia/Anaplasma and Babesia) |
| **TREATMENT:** | *Local, Local Disseminated:* Doxycycline (14-21d) > Amox > Cefurox  
*Late Disseminated, Late:* Cefriaxone 2 g/d (28d) |
EHRLICHIOSIS-ANAPLASMOSIS

AGENTS: \( Ehrlichia \) chefeensis (HME), \( Anaplasma \) phagocytophilum (HGA),

EPIDEMIOLOGY: \( Ehrlichia \) chefeensis (deer), \( Anaplasma \) (deer and field mice) → \textbf{tickbites} (transfusion/transplants rarely, contact with infected blood?)

INCUBATION: 5 days – 2 weeks (most < 1 week)
SEVERE FEBRILE ILLNESS

A previously-well 26-year-old Wisconsin milk truck driver presents with a 3-day history of fever, chills, severe headache and profound diffuse myalgias.

110/70 108 24 41.0°C. He is a muscular, acutely-ill young white male who is anxious but lucid. Rare petechiae are seen over the trunk. There is shotty lymphadenopathy. Throat is beefy red but there is no exudate. There is mild tenderness to palpation over all muscle groups. Chest is clear. Heart is regular with a faint flow murmer. Neck is supple, he is intact neurocognitively.

Hematocrit 46. WBC 3.4 with 32% band forms and Doehle bodies, platelets 64K, AST 105, CRP 22.5.
## EHRlichiosis-ANAPlasmosis

### Agents:

*Ehrlichia chefeensis, Ehrlichia muris-like, Ehrlichia ewingii* (HME)
*Anaplasma phagocytophilum* (HGA),

### Epidemiology:

*Ambyomma americanum* (Lone Star Tick, *Ehrlichia chafeensis*, deer), *Anaplasma* (deer and field mice)

→ *tickbites* (transfusion/transplants rarely, contact with infected blood?)

### Incubation:

5 days – 2 weeks (most < 1 week)
Objective.—To characterize the clinical presentation and course, laboratory findings, and treatment outcome of 12 patients with human granulocytic ehrlichiosis.

Setting.—The 12 patients were male, ranged in age from 29 to 91 years, and contracted their illness in Wisconsin or Minnesota.

Methods.—Cases were recognized by the presence of intracytoplasmic inclusions (morulae) in peripheral neutrophils of patients presenting with temperature of 38.5°C or higher, chills, severe headache, and myalgias. All patients had a complete blood cell count and blood chemistry profile. Blood smears were examined by light microscopy. All available paired serum samples were analyzed for presence of indirect fluorescent antibodies against *Ehrlichia chaffeensis*, *Ehrlichia phagocytophila*, and *Ehrlichia equi*. Blood samples from 12 patients were subjected to polymerase chain reaction analysis using primers specific for the *E* phagocytophila/E equi group, primers that include the agent identified in our patients, as well as *E* chaffeensis.

Results.—Varying combinations of leukopenia, anemia, and thrombocytopenia were found in all but one patient. All 12 patients demonstrated morulae in the cytoplasm of neutrophils, but not in mononuclear white blood cells. Serum assays failed to detect antibodies against *E* chaffeensis, but eight of 10 patients and seven of 10 patients tested had antibody titers of 1:80 or more for *E* phagocytophila and *E* equi, respectively. Polymerase chain reaction products obtained with primers for *E* phagocytophila, *E* equi, and the granulocytotropic *Ehrlichia* revealed that seven patients were infected with the same agent. The results of serological assays or polymerase chain reaction strongly suggest that all 12 patients were infected by *E* phagocytophila, *E* equi, or a closely related *Ehrlichia* species. Two of the 12 patients died. The other 10 patients improved rapidly with oral doxycycline treatment.

Conclusions.—We believe that all 12 patients have been infected with a granulocytic *Ehrlichia* species, reflecting a recently described new disease entity. The infective organism appears to be closely related to *E* phagocytophila and *E* equi. The geographic demesne of human granulocytic ehrlichiosis in the United States is expanding rapidly. Most cases of *E* chaffeensis infection described have been contracted in the south central and southeastern United States, and many patients have developed their illness following a tick bite. Recent studies demonstrating *E* chaffeensis in *Amblyomma americanum* ticks support the concept that human ehrlichiosis is a tick-borne illness. By 1990, at least 21 states had reported cases of human ehrlichiosis to the Centers for Disease Control and Prevention (CDC).

The upper Midwest represents a major endemic region for several tick-borne illnesses, including Lyme disease, babesiosis, and Rocky Mountain spotted fever. Human ehrlichiosis has so far not been reported from Wisconsin or Minnesota, except for one case that was imported from Florida. During the last 3 years, 12 patients either from Wisconsin or from Minnesota, all presenting with fever and symptoms suggestive of human ehrlichiosis, have been treated in Duluth, Minn. Each patient was noted to have *Ehrlichia*-like inclusions only in the cytoplasm of circulating neutrophils. This report describes the clinical presentations, course, and outcomes for these 12 patients and outlines what we know so
| **AGENTS:** | *Ehrlichia chefeensis* (HME), *Anaplasma phagocytophilum* (HGA), |
| **EPIDEMIOLOGY:** | *Ehrlichia chefeensis* (deer), *Anaplasma* (deer and field mice) → *tickbites* (transfusion/transplants rarely, contact with infected blood?) |
| **INCUBATION:** | 5 days – 2 weeks (most < 1 week) |
| **SYNDROME:** | *Fever, chills, myalgias and headache* (near-universal)  
Many also have dry cough, nausea/vomiting; *rash* (1/3 HME, *not* HGA).  
**HME can cause FUO 1-2 mo; HGA, immunosuppression with OIs**  
**Illness more severe with HGA,** severe in elderly, immunocompromised,  
Can mimic TSS, TTP or HPS/MAS in AIDS or transplant patients. |

Nasir Safdar,* Robert B. Love,* and Dennis G. Maki*

We describe a case of human ehrlichiosis in a lung transplant recipient and review published reports on ehrlichiosis in immunocompromised patients. Despite early therapy with doxycycline, our patient had unusually severe illness with features of thrombotic thrombocytopenic purpura. Of 23 reported cases of ehrlichiosis in immunocompromised patients, organ failure occurred in all patients and 6 (25%) died.

Since the discovery in 1987 of *Ehrlichia* as a cause of tick-borne disease in humans (1), *Ehrlichia* has been recognized as an increasingly important cause of acute febrile illness (2,3). The two main pathogenic species are *Ehrlichia chaffeensis*, which causes human monocytic ehrlichiosis (HME), and the as-yet-unnamed agent of human granulocytic ehrlichiosis (HGE) (4). A third species, *E. ewingii*, which has been recently described, causes clinical disease indistinguishable from infection caused by *E. chaffeensis* or the agent of human granulocytic ehrlichiosis (5).

Delineation of the epidemiology of human ehrlichiosis has greatly enhanced our understanding of this emerging infection. However, information on the manifestations of ehrlichiosis in immunocompromised patients is limited. We report a case of severe monocytic ehrlichiosis in a lung transplant recipient who had pancytopenia, acute renal failure, and encephalopathy. Despite early diagnosis and treatment with doxycycline, his illness progressed and took on features of thrombotic thrombocytopenic purpura (TTP). A review of reported cases of *Ehrlichia* infection in immunocompromised patients shows that the infection is far more severe in this population and is often fatal.

Case Report

A 38-year-old man with cystic fibrosis had undergone bilateral lung transplantation in 1998 and had been well. In September 2000, he visited a physician with a 3-day history of fever as high as 38.3°C, myalgias, and headache. A resident of Columbia, Missouri, the patient had spent much time outdoors but did not recall tick infestation or recent tick bite. His medications included cyclosporine, mycophenolate, prednisone, dilantin, trimethoprim-sulfamethoxazole, and valacyclovir.

On physical examination, the patient appeared acutely ill, with temperature 38.3°C, blood pressure 140/94, heart rate 110 per minute, and respiratory rate 20 per minute. He was lethargic but could follow commands, and his neurologic examination was unremarkable. Fine basilar crackles were present bilaterally, but heart sounds were normal. Examination of the abdomen was negative. Synovitis was not evident, and no cutaneous lesions were found.

The leukocyte count was 3.7×10⁹ per L with 68% neutrophils, hemoglobin was 64 g/L, and platelet count was 23,000/μL. Serum creatinine was 4.6 mg/dL, aspartate aminotransferase 420 U/L, alanine aminotransferase 96 U/L, and bilirubin 3.2 mg/dL. International normalized prothrombin time ratio (INR) was 1.4. Examination of a peripheral blood smear showed schistocytes and other microangiopathic changes.

Multiple blood cultures were negative. Cytomegalovirus DNA was not detected in peripheral blood. Noncontrast computed tomography of the brain was normal. Chest radiograph showed bilateral infiltrates.

The patient was treated initially with intravenous piperacillin-tazobactam and vancomycin. Cyclosporine and trimethoprim-sulfamethoxazole were discontinued. The next day, his mental status continued to deteriorate. Lumbar puncture was deferred because of thrombocytopenia. Antibiotic therapy was changed to intravenous methotrexate. Four days after admission, the bone marrow was examined because of worsening pancytopenia; intracytoplasmic morulae were seen in mononuclear cells, characteristic of monocytic ehrlichiosis (Figure). Leukocytes in a peripheral blood smear also contained morulae. Intravenous doxycycline was begun for treatment of presumed *Ehrlichia* infection. Whole-blood polymerase chain reaction (PCR) (Viromed, Minneapolis, MN) in the first week of illness was subsequently reported positive for *E. chaffeensis*.

![Figure. Bone marrow examination (Wright's stain x1000). Intracytoplasmic morulae of *Ehrlichia* can be seen (arrow) within mononuclear cells.](image-url)
# EHRlichiosis-Anaplasmosis

<table>
<thead>
<tr>
<th>AGENTS:</th>
<th><em>Ehrlichia chefeensis</em> (HME), <em>Anaplasma phagocytophilum</em> (HGA),</th>
</tr>
</thead>
<tbody>
<tr>
<td>EPIDEMIOLOGY:</td>
<td><em>Ehrlichia chefeensis</em> (deer), Anaplasma (deer and field mice)</td>
</tr>
<tr>
<td></td>
<td>→ <em>tickbites</em> (transfusion/transplants rarely, contact with infected blood?)</td>
</tr>
<tr>
<td>INCUBATION:</td>
<td>5 days – 2 weeks (most &lt; 1 week)</td>
</tr>
<tr>
<td>SYNDROME:</td>
<td><em>Fever, chills, myalgias and headache</em> (near-universal)</td>
</tr>
<tr>
<td></td>
<td>Many also have dry cough, nausea/vomiting; rash (1/3 HME, <em>not</em> HGA).</td>
</tr>
<tr>
<td></td>
<td><em>HME can cause FUO 1-2 mo; HGA, immunosuppression with OIs</em></td>
</tr>
<tr>
<td></td>
<td><em>Illness more severe with HGA</em>, severe in elderly, immunocompromised,</td>
</tr>
<tr>
<td></td>
<td>Can mimic TSS, TTP or HPS/MAS in AIDS or transplant patients.</td>
</tr>
<tr>
<td></td>
<td><strong>Triad</strong>: leukopenia (&lt;5,000)</td>
</tr>
<tr>
<td></td>
<td>thrombocytopenia (&lt;120,000)</td>
</tr>
<tr>
<td></td>
<td>elevated transaminases (80-300)</td>
</tr>
<tr>
<td></td>
<td><strong>CSF lymphocytic pleocytosis with HME</strong> (<em>not</em> HGA)</td>
</tr>
</tbody>
</table>
# EHRlichiosis-ANAPLASMOSIS

## AGENTS:

| Ehrlichia chefeensis (HME), Anaplasma phagocytophilum (HGA), |

## EPIDEMIOLOGY:

| Ehrlichia chafeensis (deer), Anaplasma (deer and field mice) | → tickbites (transfusion/transplants rarely, contact with infected blood?) |

## INCUBATION:

| 5 days – 2 weeks (most < 1 week) |

## SYNDROME:

| Fever, chills, myalgias and headache (near-universal) |
| Many also have dry cough, nausea/vomiting; rash (1/3 HME, not HGA). |
| **HME can cause FUO 1-2 mo; HGA, immunosuppression with OIs** |
| **Illness more severe with HGA**, severe in elderly, immunocompromised, Can mimic TSS, TTP or HPS/MAS in AIDS or transplant patients. |

**Triad:** leukopenia (<5,000)  
Thrombocytopenia (<120,000)  
Elevated transaminases (80-300)  
**CSF lymphocytic pleocytosis with HME** (not HGA)  

## DIAGNOSIS:

| Morulae in peripheral blood: HME (monos, <10%); HGA (PMNs, 20-80%) |
| PCR blood (both for Ehrlichia and Anaplasma, ~75% sensitive) |
| Serology IFA (if PCR negative but strong suspicion) |
| ALWAYS CONSIDER CO-INFECTION (Lyme or Babesia) |
**EHRLICHIOSIS-ANAPLASMOsis**

| AGENTS: | *Ehrlichia chefeensis* (HME), *Anaplasma phagocytophilum* (HGA), |
| EPIDEMIOLOGY: | *Ehrlichia chefeensis* (deer), *Anaplasma* (deer and field mice) → **tickbites** (transfusion/transplants rarely, contact with infected blood?) |
| INCUBATION: | 5 days – 2 weeks (most < 1 week) |
| SYNDROME: | **Fever, chills, myalgias and headache** (near-universal) Many also have dry cough, nausea/vomiting; rash (1/3 HME, **not** HGA). **HME can cause FUO 1-2 mo; HGA, immunosuppression with OIs** **Illness more severe with HGA**, severe in elderly, immunocompromised, Can mimic TSS, TTP or HPS/MAS in AIDS or transplant patients. **Triad**: leukopenia (<5,000) thrombocytopenia (<120,000) elevated transaminases (80-300) **CSF lymphocytic pleocytosis with HME** (not HGA) |
| DIAGNOSIS: | **Morulae** in peripheral blood: HME (monos, <10%); HGA (PMNs, 20-80%) **PCR** blood (*both* for *Ehrlichia* and *Anaplasma*, ~75% sensitive) **Serology** IFA (if PCR negative but strong suspicion) **ALWAYS CONSIDER CO-INFECTION** (*Lyme* or *Babesia*) |
| TREATMENT: | **Doxycycline** 100 mg BID mg (14d), 100 mg TID for patients >100 kg **Rifampin** 600 mg/d (14d), if intolerance of doxycycline |
A 66-year-old Type 2 insulin-dependent diabetic with Parkinsons disease, CAD s/p recent CABG and remote history of Hodgkins lymphoma in childhood was admitted to a rural southern Wisconsin hospital with weakness and anemia.

110/70 110 18 38.0°C. While quite pale, there is no rash, and there is no lymphadenopathy or splenomegally. There is a healing sternotomy wound and an old midline epigastric scar.

Hgb 4.8, MCV 96, WBC 5.6 with mild lymphopenia and platelets 104K, reticulocyte count 14%, LDH 850, bilirubin 6.9 with normal transaminases and haptoglobin undetectable.
## BABESIOSIS

### AGENT:
- *Babesia microti* (Midwest, East coast)
- *Babesia duncani* (West coast)

### EPIDEMIOLOGY:
- *Ixodes scapularis* (*B microti*, field mouse), *I. pacificus* (*B duncani*)
- **Tickbites** or **Transfusion** or **Transplantation**
- **Congenital Infection** (rare)

### INCUBATION PERIOD:
- **Tickbite-acquired:** ~3 – 30 days
- **Transfusion or Transplant-acquired:** ~37 days
TRANSFUSION-RELATED BABESIOSIS

• Risk nationwide ~1: 1 million units transfused
  (1: 15,000 in RI)
• Incubation period ~ 37 days
• Most affected are immunocompromised, CV surgery,
  hemoglobinopathies, newborns or infants or elderly;
• Mortality ~20%
• Blood banks do not yet routinely screen donated blood
  for Babesia infection but RI bloodbanks defer if history
  of babesiosis
### BABESIOSIS

| **AGENT:** | Babesia microti (Midwest, East coast)  
|            | Babesia duncani (West coast) |
| **EPIDEMIOLOGY:** | Ixodes scapularis (B microti, field mouse), I. pacificus (B duncani)  
|                | → Tickbites or Transfusion or Transplantation  
|                | Congenital Infection (rare) |
| **INCUBATION PERIOD:** | Tickbite-acquired: ~3 – 30 days  
|                        | Transfusion or Transplant-acquired: ~37 days |
| **SYNDROME:** | Most infections asymptomatic  
|               | Fever, HA, myalgias.... **weakness, pallor, dark urine.**  
|               | **High risk for severe disease and hemolysis:**  
|                   | - Asplenia  
|                   | - Immunosuppression (AIDS, transplants, steroids, anti-TNF drugs)  
|                   | - Elderly, high-risk neonates  
| **Complications:** | CHF, ACS, ARDS, AKI, DIC, splenic rupture  
| **COINFECTION VERY COMMON** | (Lyme, Ehrlichia/Anaplasma) |

# BABESIOSIS

## AGENT:
- Babesia microti (Midwest, East coast)
- Babesia duncani (West coast)

## EPIDEMIOLOGY:
- *Ixodes scapularis* (*B microti*, field mouse), *I. pacificus* (*B duncani*)
  - Tickbites or Transfusion or Transplantation
  - Congenital Infection (rare)

## INCUBATION PERIOD:
- Tickbite-acquired: ~3 – 30 days
- Transfusion or Transplant-acquired: ~37 days

## SYNDROME:
- Most infections asymptomatic
- Fever, HA, myalgias...weakness, pallor, dark urine.
  - High risk for severe disease and hemolysis:
    - Asplenia
    - Immunosuppression (AIDS, transplants, steroids, anti-TNF drugs)
    - Elderly, high-risk neonates
  - Complications: CHF, ACS, ARDS, AKI, DIC, splenic rupture
  - COINFECTION VERY COMMON (Lyme, Ehrlichia/Anaplasma)

## DIAGNOSIS
- Peripheral blood smear
- Serologic (IFAT)
- PCR 18s rRNA peripheral blood (>95% sensitive)
## BABESIOSIS

### AGENT:

| Babesia microti (Midwest, East coast) |
| Babesia duncani (West coast) |

### EPIDEMIOLOGY:

- *Ixodes scapularis* (*B microti*, field mouse), *I. pacificus* (*B duncani*)
- **Tickbites** or **Transfusion** or **Transplantation**
- **Congenital Infection** (rare)

### INCUBATION PERIOD:

- **Tickbite-acquired**: ~3 – 30 days
- **Transfusion or Transplant-acquired**: ~37 days

### SYNDROME:

Most infections asymptomatic
- Fever, HA, myalgias...*weakness, pallor, dark urine.*
- **High risk for severe disease and hemolysis:**
  - Asplenia
  - Immunosuppression (AIDS, transplants, steroids, anti-TNF drugs)
  - Elderly, high-risk neonates
- **Complications**: CHF, ACS, ARDS, AKI, DIC, splenic rupture
- **COINFECTION VERY COMMON** (Lyme, Ehrlichia/Anaplasma),

### DIAGNOSIS

- Peripheral blood smear
- Serologic (IFAT)
- PCR 18s rRNA peripheral blood (>95% sensitive)

### TREATMENT:

- **Mild**: Atovaquone 750 mg BID + Azithro 500 mg d1 and 250-500 mg/d
- **Severe**: Clinda 600 mg TID + Quinine 650 mg TID (7-10d)
SEVERE ANEMIA

A 66-year-old Type 2 insulin-dependent diabetic with Parkinson's disease, CAD s/p recent CABG and remote history of Hodgkins lymphoma in childhood was admitted to a rural southern Wisconsin hospital with weakness and anemia.

110/70 110 18 38.0°C. While quite pale, there is no rash, and there is no lymphadenopathy or splenomegally. There is a healing sternotomy wound and an old midline epigastric scar.

Hgb 4.8, MCV 96, WBC 5.6 with mild lymphopenia and platelets 104K, reticulocyte count 14%, LDH 850, bilirubin 6.9 with normal transaminases and haptoglobin undetectable.

He was transferred to UWHC where he begun on quinine and clindamycin and transfused 3 U pRBCs but continued to hemolyze and require 2-3 units pRBCs a day over 7 days....
Post-Babesiosis Warm Autoimmune Hemolytic Anemia

Ann E. Woolley, M.D., Mary W. Montgomery, M.D., William J. Savage, M.D., Ph.D., Maureen O. Achebe, M.D., Kathleen Dunford, B.S., Sarah Villeda, B.S., James H. Maguire, M.D., and Francisco M. Marty, M.D.
# Babesiosis

## Agent:
- *Babesia microti* (Midwest, East coast)
- *Babesia duncani* (West coast)

## Epidemiology:
- *Ixodes scapularis* (*B microti*, field mouse), *I. pacificus* (*B duncani*),
  - **Tickbites** or **Transfusion** or **Transplantation**
  - **Congenital Infection** (rare)

## Incubation Period:
- **Tickbite-acquired:** ~3 – 30 days
- **Transfusion or Transplant-acquired:** ~37 days

## Syndrome:
- Most infections asymptomatic
- Fever, HA, myalgias... weaknesses, pallor, dark urine.
- **High risk for severe disease and hemolysis:**
  - Asplenia
  - Immunosuppression (AIDS, transplants, steroids, anti-TNF drugs)
  - Elderly, high-risk neonates
- **Complications:** CHF, ACS, ARDS, AKI, DIC, splenic rupture
- **Coinfection very common** (Lyme, Ehrlichia/Anaplasma),
  - **Autoimmune hemolytic anemia** if asplenia

## Diagnosis:
- Peripheral blood smear
- Serologic (IFAT)
- PCR 18s rRNA peripheral blood (>95% sensitive)

## Treatment:
- **Mild:** Atovaquone 750 mg BID + Azithro 500 mg d1 and 250-500 mg/d
- **Severe:** Clinda 600 mg TID + Quinine 650 mg TID (7-10d)
CRITICAL ILLNESS WITH RASH

A 55 year-old long-distance hiker became unwell over 5 days with severe frontal headache, fever and rigors, abdominal pain and profound myalgias, with a maculopapular rash.

On examination he was alert and conscious with VS 38.5°C, 84/45, 136, 22 SpO2 97%. There was striking conjunctival injection and a generalized MP rash most pronounced on the face, palms, and soles, with numerous petechial and coalescent hemorrhagic lesions. No lymphadenopathy. Chest was clear. Diffuse deep abdominal tenderness. He was intact neurocognitively, without meningismus.

Hgb 11.5, WBC 7.4 with 45% bands and 10% metamyelocytes. platelets 22,000, CRP 25.5
# ROCKY MOUNTAIN SPOTTED FEVER

<table>
<thead>
<tr>
<th><strong>AGENT:</strong></th>
<th><em>Rickettsia rickettsii</em></th>
</tr>
</thead>
</table>
| **EPIDEMIOLOGY:** | *Dermacentor variabilis* (Dog tick, eastern, south central)  
*Dermacentor andersoni* (Wood Tick, west of Mississippi) |
| **INCUBATION PERIOD:** | 5 - 7 days |

![Map of Rocky Mountain Spotted Fever distribution in the United States](image)
## ROCKY MOUNTAIN SPOTTED FEVER

<table>
<thead>
<tr>
<th>AGENT:</th>
<th>Rickettsia rickettsii</th>
</tr>
</thead>
</table>
| EPIDEMIOLOGY:| *Dermacentor variabilis* (American Dog tick, eastern and south central)  
*Dermacentor andersoni* (Wood Tick, west of Mississippi)  
*Rhipicephalus sanguineus* (Brown dog tick) |
| INCUBATION PERIOD: | 5 - 7 days |
| SYNDROME:    | Fever, **RASH (90%)** – *characteristically involves palms, myalgias, headache* (near-universal)  
…also dry cough, abdominal pain, nausea/vomiting, AMS, |
## ROCKY MOUNTAIN SPOTTED FEVER

<table>
<thead>
<tr>
<th>AGENT:</th>
<th><em>Rickettsia rickettsii</em></th>
</tr>
</thead>
</table>
| **EPIDEMIOLOGY:** | *Dermacentor variabilis* (Dog tick, eastern, south central)  
*Dermacentor andersoni* (Wood Tick, west of Mississippi) |
| **INCUBATION PERIOD:** | 5 - 7 days |
| **SYNDROME:**   | *Fever, RASH (90%), myalgias, headache* (near-universal)  
...also dry cough, abdominal pain, nausea/vomiting, AMS  
*Can mimic* meningococcemia, other bacterial sepsis, ehrlichiosis, anaplasmosis, leptospirosis, TTP, measles, Kawasaki disease  
*CSF pleocytosis* (near universal but CSF glucose normal)  
**Severe disease:** elderly, children<10, alcoholism, native Americans  
- Native Americans, African-Americans  
- Alcoholism, G-6-PD deficiency  
- Elderly, children<10 yo  
**Complications:** shock, ARDS, seizures, hyponatremia, CHF, AKI |
# ROCKY MOUNTAIN SPOTTED FEVER

<table>
<thead>
<tr>
<th><strong>AGENT:</strong></th>
<th><em>Rickettsia rickettsii</em></th>
</tr>
</thead>
</table>
| **EPIDEMIOLOGY:** | *Dermacentor variabilis* (Dog tick, eastern, south central)  
*Dermacentor andersonii* (Wood Tick, west of Mississippi) |
| **INCUBATION PERIOD:** | 5 - 7 days |
| **SYNDROME:** | *Fever, RASH (90%), myalgias, headache* (near-universal)  
...also dry cough, abdominal pain, nausea/vomiting, AMS  
*Can mimic* meningococcemia, other bacterial sepsis, ehrlichiosis, anaplasmosis, leptospirosis, TTP, measles, Kawasaki disease  
*CSF pleocytosis* (near universal but CSF glucose normal)  
*Severe disease:* elderly, children<10, alcoholism, native Americans  
- Native Americans, African-Americans  
- Alcoholism, G-6-PD deficiency  
- Elderly, children<10 yo  
*Complications:* shock, ARDS, seizures, hyponatremia, CHF, AKI, |
| **DIAGNOSIS:** | Biopsy rash with DFA (80% sensitive)  
Serology (IgM not + for 5-7d)  
PCR (poor sensitivity)  
*MUST SUSPECT CLINICALLY AND TREAT EMPIRICALLY* |
## Rocky Mountain Spotted Fever

<table>
<thead>
<tr>
<th><strong>Agent:</strong></th>
<th>Rickettsia rickettsii</th>
</tr>
</thead>
</table>
| **Epidemiology:** | Dermacentor variabilis (Dog tick, eastern, south central)  
Dermacentor andersoni (Wood Tick, west of Mississippi) |
| **Incubation:** | 5 - 7 days |
| **Syndrome:** | Fever, RASH (90%), myalgias, headache (near-universal)  
... also dry cough, abdominal pain, nausea/vomiting, AMS  
*Can mimic* meningococcemia, other bacterial sepsis, ehrlichiosis, anaplasmosis, leptospirosis, TTP, measles, Kawasaki disease  
*CSF pleocytosis* (near universal but CSF glucose normal)  
**Severe disease:** elderly, children<10, alcoholism, native Americans  
- Native Americans, African-Americans  
- Alcoholism, G-6-PD deficiency  
- Elderly, children<10 yo  
**Complications:** shock, ARDS, seizures, hyponatremia, CHF, AKI, |
| **Diagnosis:** | Biopsy rash with DFA (80% sensitive)  
Serology (IgM not + for 5-7d)  
PCR (poor sensitivity)  
**MUST SUSPECT CLINICALLY AND TREAT EMPIRICALLY** |
| **Treatment:** | *Doxycycline* 100 mg BID mg (until afebrile 3d), 100 mg TID for >100 kg  
*Chloramphenicol* 0.5-1 g Q6h, if intolerance of doxycycline |
<table>
<thead>
<tr>
<th><strong>AGENT:</strong></th>
<th>Francisella tularensis</th>
</tr>
</thead>
</table>
| **EPIDEMIOLOGY:** | Bites by multiple species ticks, fleas, flies, mosquitos  
Direct contact, contaminated water or food, airborne |
| **INCUBATION PERIOD:** | 3 – 5 days |
The bacterium *F. tularensis* may be transmitted between rodents and rabbits and to humans by ticks. Drinking contaminated water, contact with infected animals, eating undercooked rabbit meat, and inhaling contaminated dust are ways in which tularemia can be contracted.
# TULAREMIA

<table>
<thead>
<tr>
<th><strong>AGENT:</strong></th>
<th><em>Francisella tularensis</em></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>EPIDEMIOLOGY:</strong></td>
<td><em>Bites by multiple species ticks, fleas, flies, mosquitos Direct contact, contaminated water or food, airborne</em></td>
</tr>
<tr>
<td><strong>INCUBATION PERIOD:</strong></td>
<td>3 – 5 days</td>
</tr>
<tr>
<td><strong>SYNDROME:</strong></td>
<td><strong>Multiple Forms:</strong></td>
</tr>
<tr>
<td></td>
<td><strong>Ulceroglandular</strong>: local ulcer + proximal (axillary/groin/cervical) lymphadenopathy, often minimal constitutional symptoms</td>
</tr>
<tr>
<td></td>
<td><strong>Glandular</strong>: Like ulceroglandular, without ulcer</td>
</tr>
<tr>
<td></td>
<td><strong>Oculoglandular</strong>: Ocular inflammation with proximal lymphadenopathy</td>
</tr>
<tr>
<td></td>
<td><strong>Typhoidal</strong>: bacteremic infection, with or without ulcer glandular</td>
</tr>
<tr>
<td></td>
<td><strong>Pneumonic</strong>: complicates all forms, especially bacteremic, often lung nodules, can also be airborne aquisition</td>
</tr>
</tbody>
</table>

![Image of Tularemia symptoms](www.wired.com)
## TULAREMIA

<table>
<thead>
<tr>
<th><strong>AGENT:</strong></th>
<th><em>Francisella tularensis</em></th>
</tr>
</thead>
</table>
| **EPIDEMIOLOGY:** | *Bites by multiple species ticks, fleas, flies, mosquitos*  
*Direct contact, contaminated water or food, airborne* |
| **INCUBATION PERIOD:** | 3 – 5 days |
| **SYNDROME:** | **Multiple Forms:**  
**Ulceroglandular:** local ulcer + proximal (axillary/groin/cervical) lymphadenopathy, often minimal constitutional symptoms  
**Glandular:** Like ulceroglandular, without ulcer  
**Oculoglandular:** Ocular inflammation with proximal lymphadenopathy  
**Typhoidal:** bacteremic infection, with or without ulceroglandular  
**Pneumonic:** complicates all forms, especially bacteremic, often lung nodules, can also be airborne acquisition |
| **DIAGNOSIS:** | **SeroLogic** (excellent but not + for 7-14 days)  
**PCR** (excellent, if available)  
**Cultures** (grows slowly, very hazardous for lab workers)  
**MUST SUSPECT CLINICALLY AND TREAT EMPIRICALLY** |
<table>
<thead>
<tr>
<th>TULAREMIA</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>AGENT:</strong></td>
</tr>
</tbody>
</table>
| **EPIDEMIOLOGY:** | *Bites by multiple species ticks, fleas, flies, mosquitos*  
*Direct contact, contaminated water or food, airborne* |
| **INCUBATION PERIOD:** | 3 – 5 days |
| **SYNDROME:** | **Multiple Forms:**  
*Ulceroglandular:* local ulcer + proximal (axillary/groin/cervical) lymphadenopathy, often minimal constitutional symptoms  
*Glandular:* Like ulceroglandular, without ulcer  
*Oculoglandular:* Ocular inflammation with proximal lymphadenopathy  
*Typhoidal:* bacteremic infection, with or without ulceroglandular  
*Pneumonic:* complicates all forms, especially bacteremic, often lung nodules, can also be airborne aquisition |
| **DIAGNOSIS:** | *Sero logic* (excellent but not + for 7-14 days)  
*PCR* (excellent, if available)  
*Cultures* (grows slowly, very hazardous for lab workers)  
**MUST SUSPECT CLINICALLY AND TREAT EMPIRICALLY** |
| **TREATMENT:** | *Doxycycline* 100 mg BID (7-14d), 100 mg TID for >100 kg  
*Gentamicin* 5 mg/kg IV daily (pregnancy, children)  
*Ciprofloxacin* 500-750 mg/d  
*Chloramphenicol* 0.5-1 g QID  
*Doxycycline + Gentamicin* for Meningitis |
# Tularemia

**Agent:** *Francisella tularensis*

**Epidemiology:** Bites by multiple species ticks, fleas, flies, mosquitoes. Direct contact, contaminated water or food, airborne.

**Incubation period:** 3 – 5 days

**Syndrome:** Multiple Forms:

- **Ulceroglandular:** local ulcer + proximal (axillary/groin/cervical) lymphadenopathy, often minimal constitutional symptoms
- **Glandular:** Like ulceroglandular, without ulcer
- **Oculoglandular:** Ocular inflammation with proximal lymphadenopathy
- **Typhoidal:** bacteremic infection, with or without ulceroglandular
- **Pneumonic:** complicates all forms, especially bacteremic, often lung nodules, can also be airborne aquisition

**Diagnosis:** Serologic (excellent but not + for 7-14 days)

- **PCR** (excellent, if available)
- **Cultures** (grows slowly, very hazardous for lab workers)

*MUST SUSPECT CLINICALLY AND TREAT EMPIRICALLY*

**Treatment:**

- **Doxycycline** 100 mg BID (7-14d), 100 mg TID for >100 kg
- **Gentamicin** 5 mg/kg IV daily (pregnancy, children)
- **Ciprofloxacin** 500-750 mg/d
- **Doxycycline + Gentamicin**
- **Chloramphenicol** 0.5-1 g QID for Meningitis
A 44-year-old healthy man returned from vacation in a remote cabin at 5500 feet in the mountains of San Juan, Colorado, in June 2017 with headache, myalgias, vomiting and violent chills over the past 2 days, one week after he had recovered from a similar 5-day illness while on vacation. He reported no known tick or other insect exposure.

39.2°C, 90, 131/80. Examination was unremarkable, with no rash, lymphadenopathy or meningismus.

Hgb 14.0, WBC 6.0 with 10% bandemia, platelets 37K. CRP 17.0. Bilirubin 2.1, AST 50.
TICK-BORNE RELAPSING FEVER

AGENT: Borrelia hermsii (Western states)  
           Borrelia turicatae (Southwest and south central states)

EPIDEMIOLOGY: Ornithodoros spp (soft-bodied ticks, squirrels, other mammals)

INCUBATION PERIOD: 3 - 5 days
TICK-BORNE RELAPSING FEVER

AGENT:  
- *Borrelia hermsii* (Western states)  
- *Borrelia turicatae* (Southwest and south central states)

EPIDEMIOLOGY:  
*Ornithodoros spp* (soft-bodied ticks, squirrels, other mammals)

INCUBATION PERIOD:  
3 - 5 days

SYNDROME:  
*Bouts of Relapsing Fever* x3-6 d every 4-14 days, defervescence by “crisis” with profound chills, diaphoresis and hypotension, HA, myalgias, dry cough.
# Tick-Borne Relapsing Fever

| **Agent:** | *Borrelia hermsii* (Western states)  
*Borrelia turicatae* (Southwest and south central states) |
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Epidemiology:</strong></td>
<td><em>Ornithodoros</em> spp (soft-bodied ticks, squirrels, other mammals)</td>
</tr>
<tr>
<td><strong>Incubation Period:</strong></td>
<td>3 - 5 days</td>
</tr>
</tbody>
</table>
| **Syndrome:** | **Bouts of Relapsing Fever** *x3-6 d every 4-14 days*, defervescence by “crisis” with profound chills, diaphoresis and hypotension, HA, myalgias, dry cough.  
**Complications:** meningitis, cranial neuropathy, radiculopathy, shock, ARDS |

# TICK-BORNE RELAPSING FEVER

| **AGENT:** | *Borrelia hermsii* (Western states)  
*Borrelia turicatae* (Southwest and south central states) |
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>EPIDEMOIOLOGY:</strong></td>
<td><em>Ornithodoros spp</em> (soft-bodied ticks, squirrels, other mammals)</td>
</tr>
<tr>
<td><strong>INCUBATION PERIOD:</strong></td>
<td>3 - 5 days</td>
</tr>
</tbody>
</table>
| **SYNDROME:** | *Bouts of Relapsing Fever* x3-6 d every 4-14 days,  
defervescence by “crisis” with profound chills, diaphoresis and hypotension, HA, myalgias, dry cough.  
**Complications:** meningitis, cranial neuropathy, radiculopathy, shock, ARDS |
| **DIAGNOSIS:** | *Peripheral blood smear, thick smear, buffy coat dark field*  
Mouse inoculation or culture (research labs)  
Serologic (insensitive)  
PCR (>95% sensitive) |
SEVERE ENCEPHALITIS

A 67-year-old woman from Aitkin, Minn, was admitted to hospital in May 2011 with a 3-day history of headache, dizziness, fever and chills, nausea and confusion with slurred speech. She had removed many deer ticks after gardening or hiking in the woods that spring.

On admission, she was alert lucid. 100°F, 138/77, 83 SpO2 98%r. Examination was normal.

*B burgdorferi*, Ehrlichia and Anaplasma antibodies were negative. WBC 10.8 with normal differential. CSF 80 NCs, 89% PMNs, with protein 63 and glucose 78, NOS on gram stain. Brain MRI revealed nonspecific inflammatory changes within the thalamus, midbrain, and cerebellum.

Overnight she became apneic and required intubation. Examination revealed absent deep tendon reflexes, ocular deviation, positive Babinskis and bilateral flaccid paralysis. Pupillary light and corneal reflexes remained intact. No spontaneous respirations were initiated. EEG epileptiform discharges. She remained unresponsive with flaccid paralysis and areflexia, and 13 days from the onset of illness she was extubated and died.
POWASSAN VIRUS FEVER

AGENT: Powassan Virus (Flavivirus, related to Dengue, Zika)

EPIDEMIOLOGY: *Ixodes scapularis* (deer tick, field mice and deer), other *Ixodes* spp, *Dermacentor andersoni*

INCUBATION PERIOD: ~1 week
POWASSAN FEVER

<table>
<thead>
<tr>
<th>AGENT:</th>
<th>Powassan Virus (Flavivirus, related to Dengue, Zika)</th>
</tr>
</thead>
<tbody>
<tr>
<td>EPIDEMIOLOGY:</td>
<td><em>Ixodes scapularis</em> (deer tick, field mice and deer), other <em>Ixodes</em> spp, <em>Dermacentor andersoni</em></td>
</tr>
<tr>
<td>INCUBATION PERIOD:</td>
<td>~ 1 week</td>
</tr>
<tr>
<td>SYNDROME:</td>
<td>Powassan virus infection characteristically produces an aseptic/lymphocytic <strong>meningoencephalitis</strong></td>
</tr>
<tr>
<td></td>
<td><em>Fever, severe headache and AMS</em>, myalgias, weakness, nausea and vomiting, occasional rash</td>
</tr>
<tr>
<td></td>
<td><strong>Complications</strong>: coma, seizures, hemiplegia, flaccid paralysis, cognitive residual, up to 15% fatal</td>
</tr>
</tbody>
</table>
# POWASSAN FEVER

<table>
<thead>
<tr>
<th><strong>AGENT:</strong></th>
<th>Powassan Virus (Flavivirus, related to Dengue, Zika)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>EPIDEMILOGY:</strong></td>
<td><em>Ixodes scapularis</em> (deer tick, field mice and deer), other <em>Ixodes</em> spp, <em>Dermacentor andersoni</em></td>
</tr>
<tr>
<td><strong>INCUBATION PERIOD:</strong></td>
<td>~ 1 week</td>
</tr>
</tbody>
</table>
| **SYNDROME:** | Powassan virus infection characteristically produces an aseptic/lymphocytic *meningoencephalitis*  

*Fever, severe headache and AMS,* myalgias, weakness, nausea and vomiting, occasional rash  

*Complications:* coma, seizures, hemiplegia, flaccid paralysis, cognitive residual, 0-15% fatal |
| **DIAGNOSIS:** | *SEROLOGIC* (IgM in serum or CSF, 4-fold seroconversion, confirmation by PRNT)  
*PCR* (CSF) |
# POWASSAN FEVER

<table>
<thead>
<tr>
<th><strong>AGENT:</strong></th>
<th>Powassan Virus (Flavivirus, related to Dengue, Zika)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>EPIDEMIOLOGY:</strong></td>
<td><em>Ixodes scapularis</em> (deer tick, field mice and deer), other <em>Ixodes</em> spp, <em>Dermacentor andersoni</em></td>
</tr>
<tr>
<td><strong>INCUBATION PERIOD:</strong></td>
<td>~ 1 week</td>
</tr>
<tr>
<td><strong>SYNDROME:</strong></td>
<td>Powassan virus infection characteristically produces an aseptic/lymphocytic <em>meningoencephalitis</em></td>
</tr>
<tr>
<td></td>
<td><em>Fever, severe headache and AMS</em>, myalgias, weakness, nausea and vomiting, occasional rash</td>
</tr>
<tr>
<td></td>
<td><strong>Complications:</strong> coma, seizures, hemiplegia, flaccid paralysis, cognitive residual, 0-15% fatal</td>
</tr>
<tr>
<td><strong>TREATMENT:</strong></td>
<td><em>Supportive, only</em></td>
</tr>
<tr>
<td><strong>Serologic</strong></td>
<td>(IgM in serum or CSF, 4-fold seroconversion, confirmation by PRNT)</td>
</tr>
<tr>
<td><strong>PCR</strong></td>
<td>(CSF)</td>
</tr>
</tbody>
</table>
**COLORADO TICK FEVER**

<table>
<thead>
<tr>
<th><strong>AGENT:</strong></th>
<th>Colorado Tick Fever Virus</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>EPIDEMIOLOGY:</strong></td>
<td><em>Ixodes scapularis</em> (deer tick, field mice and deer), other <em>Ixodes</em> spp, <em>Dermacentor andersoni</em></td>
</tr>
<tr>
<td><strong>INCUBATION PERIOD:</strong></td>
<td>~ 1 week</td>
</tr>
</tbody>
</table>

*Counts with reported Colorado tick fever cases* 
*Approximate geographic distribution of Dermacentor andersoni*
<table>
<thead>
<tr>
<th><strong>AGENT:</strong></th>
<th>Colorado Tick Fever Virus</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>EPIDEMIOLOGY:</strong></td>
<td><em>Ixodes scapularis</em> (deer tick, field mice and deer), other <em>Ixodes</em> spp, <em>Dermacentor andersoni</em></td>
</tr>
<tr>
<td><strong>INCUBATION PERIOD:</strong></td>
<td>~ 1 week</td>
</tr>
</tbody>
</table>
| **SYNDROME:** | *Most Colorado Tick Virus infections are subclinical* but when symptomatic are *rarely severe*, neurocognitive residual or fatalities are rare.  
*Fever with biphasic pattern, headache*, myalgias, weakness, nausea and vomiting, 15% have rash  
*Lab: Leukopenia, mild thrombocytopenia, CSF lymphocytic pleocytosis* |
<table>
<thead>
<tr>
<th><strong>AGENT:</strong></th>
<th>Colorado Tick Fever Virus</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>EPIDEMIOLOGY:</strong></td>
<td><em>Ixodes scapularis</em> (deer tick, field mice and deer), other <em>Ixodes</em> spp, <em>Dermacentor andersoni</em></td>
</tr>
<tr>
<td><strong>INCUBATION PERIOD:</strong></td>
<td>~ 1 week</td>
</tr>
</tbody>
</table>
| **SYNDROME:** | *Most Colorado Tick Virus infections are subclinical* but when symptomatic are rarely severe, neurocognitive residual or fatalities are rare.  
*Fever with biphasic pattern, headache*, myalgias, weakness, nausea and vomiting, 15% have rash  
*Lab: Leukopenia, mild thrombocytopenia, CSF lymphocytic pleocytosis* |
| **DIAGNOSIS:** | *SeroLogic* (IgM in serum or CSF, 4-fold seroconversion, confirmation by PRNT)  
*PCR* (CSF) |
## COLORADO TICK FEVER

<table>
<thead>
<tr>
<th><strong>AGENT:</strong></th>
<th>Colorado Tick Fever Virus</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>EPIDEMIOLOGY:</strong></td>
<td><em>Ixodes scapularis</em> (deer tick, field mice and deer), other <em>Ixodes</em> spp, <em>Dermacentor andersoni</em></td>
</tr>
<tr>
<td><strong>INCUBATION PERIOD:</strong></td>
<td>~ 1 week</td>
</tr>
</tbody>
</table>
| **SYNDROME:** | *Most Colorado Tick Virus infections are subclinical* but when symptomatic are rarely severe, neurocognitive residual or fatalities are rare.  
*Fever with biphasic pattern, headache*, myalgias, weakness, nausea and vomiting, 15% have rash  
*Lab: Leukopenia, mild thrombocytopenia, CSF lymphocytic pleocytosis* |
| **TREATMENT:** | *Supportive, only* |

---

**Serologic** (IgM in serum or CSF, 4-fold seroconversion, confirmation by PRNT)  
**PCR** (CSF)
SUDDEN FLACCID PARALYSIS

A previously well 19-year-old girl who resided in rural southern Wisconsin was taken to the ED because of a 2-day history of unsteady gait, difficulty standing, and inability to walk.

On physical examination, she was afebrile, alert, and normotensive but could stand only briefly before requiring assistance. Cranial nerves were intact. However, she exhibited marked extremity and mild truncal ataxia, DTRs were absent.

WBC 5.2, CRP 0.
# TICK PARALYSIS

| AGENT: | Dermacentor andersoni (Rocky Mountain wood tick)  
|        | Dermacentor varabilis (Dog tick)  
|        | Amblyomma americanum (Lone Star tick)  
|        | Ixodes scapularis (Wisconsin Deer tick)  
|        | Ixodes pacificus |

| EPIDEMIOLOGY: | Sustained feeding by one of above species |

| INCUBATION PERIOD: | ~ 4 - 7 days of continuous feeding by a female tick |

| SYNDROME: | Produced by *tick salivary neurotoxin*  
|           | *Paresthesia, myalgias and weakness followed by rapidly progressive local or ascending total body paralysis, including cranial nerves.*  
|           | *No* fever, leukocytosis, CSF, EEG or MRI abnormalities |

*Picture courtesy of Dr. Jodi Hibdon, University of Florida*
# TICK PARALYSIS

| **AGENT:** | *Dermacentor andersoni* (Rocky Mountain wood tick)  
*Dermacentor varabilis* (Dog tick)  
*Amblyomma americanum* (Lone Star tick)  
*Ixodes scapularis* (Wisconsin Deer tick)  
*Ixodes pacificus* |
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>EPIDEMIOLOGY:</strong></td>
<td>Sustained feeding by one of above species</td>
</tr>
<tr>
<td><strong>INCUBATION PERIOD:</strong></td>
<td>~ 4 - 7 days of continuous feeding by a female tick</td>
</tr>
</tbody>
</table>
| **SYNDROME:** | Produced by *tick salivary neurotoxin*  
*Paresthesia, myalgias and weakness followed by rapidly progressive local or ascending total body paralysis, including cranial nerves.*  
No fever, leukocytosis, CSF, EEG or MRI abnormalities |
| | **Differential Diagnosis:**  
GBS, myasthenia gravis, botulism, poliomyelitis, shellfish poisoning, insecticide poisoning, viral encephalomyelitis |
# TICK PARALYSIS

| AGENT: | Dermacentor andersoni (Rocky Mountain wood tick)  
|        | Dermacentor varabilis (Dog tick)  
|        | Amblyomma americanum (Lone Star tick)  
|        | Ixodes scapularis (Wisconsin Deer tick)  
|        | Ixodes pacificus |
| EPIDEMIOLOGY: | Sustained feeding by one of above species |
| INCUBATION PERIOD: | ~ 4 - 7 days of continuous feeding by a female tick |
| SYNDROME: | Produced by *tick salivary neurotoxin* |

- *Paresthesia, myalgias and weakness followed by rapidly progressive local or ascending total body paralysis, including cranial nerves.*

- *No* fever, leukocytosis, CSF, EEG or MRI abnormalities

- **Differential Diagnosis:** GBS, myasthenia gravis, botulism, poliomyelitis, shellfish poisoning, insecticide poisoning, viral encephalomyelitis

| DIAGNOSIS: | Rule out other etiologies  
|            | FIND THE FEEDING TICK |
# TICK PARALYSIS

## AGENT:
- *Dermacentor andersoni* (Rocky Mountain wood tick)
- *Dermacentor variabilis* (Dog tick)
- *Amblyomma americanum* (Lone Star tick)
- *Ixodes scapularis* (Wisconsin Deer tick)
- *Ixodes pacificus*

## EPIDEMIOLOGY:
Sustained feeding by one of above species

## INCUBATION PERIOD:
~ 4 - 7 days of continuous feeding by a female tick

## SYNDROME:
Produced by *tick salivary neurotoxin*

- Paresthesia, myalgias and weakness followed by rapidly progressive local or ascending total body paralysis, including cranial nerves.
- No fever, leukocytosis, CSF, EEG or MRI abnormalities

## Differential Diagnosis:
GBS, myasthenia gravis, botulism, poliomyelitis, shellfish poisoning, insecticide poisoning, viral encephalomyelitis

## DIAGNOSIS:
Rule out other etiologies

**FIND THE FEEDING TICK**

## TREATMENT:
Remove the feeding tick + Supportive
RED MEAT INTOLERANCE


The patient, a previously well 47 year-old farmer with mild asthma had enjoyed them all for most of his life, with no discomfort, only great pleasure.

Until 6 months ago, that is. He had begun to experience nausea, cramps and diarrhea with hives accompanied by a ferocious itching “four times worse than poison ivy” appearing 3-4 hours after he ate red meat but never with fish or chicken.
**ALPHA-GAL (RED MEAT) ALLERGY**

**AGENT:**

- *Amblyomma americanum* (Lone Star tick)
- *Ixodes holocyclus* (Australian paralysis tick)

![Tick Images](images.png)

**Geographical range of *Amblyomma americanum* population**

Data from CDC website, accessed 9/2012
## ALPHA-GAL (RED MEAT) ALLERGY

| AGENT: | *Amblyomma americanum* (Lone Star tick)  
*Ixodes holocyclus* (Australian paralysis tick) |
|---|---|
| EPIDEMIOLOGY AND PATHOGENESIS: | Alpha-gal allergy, also known as Mammalian Meat Allergy, an IgE-mediated reaction to *galactose-alpha-1,3-galactose* (alpha-gal), found in all mammals except humans and apes.  
Bites from the **lone star tick** in the US can induce this delayed allergic response triggered by the consumption of red meat products (but NOT fowl or fish). The allergy is most often seen in the central and southern United States, which corresponds to the distribution of the lone star tick. |
# ALPHA-GAL (RED MEAT) ALLERGY

<table>
<thead>
<tr>
<th><strong>AGENT:</strong></th>
<th></th>
</tr>
</thead>
</table>
|  | *Amblyomma americanum* (Lone Star tick)  
*Ixodes holocyclus* (Australian paralysis tick) |

<table>
<thead>
<tr>
<th><strong>EPIDEMIOLOGY AND PATHOGENESIS:</strong></th>
<th></th>
</tr>
</thead>
</table>
|  | Alpha-gal allergy, also known as Mammalian Meat Allergy, an IgE-mediated reaction to *galactose-alpha-1,3-galactose* (alpha-gal), found in all mammals except humans and apes.  
Bites from the *lone star tick* in the US can induce this delayed allergic response triggered by the consumption of red meat products (but NOT fowl or fish). The allergy is most often seen in the central and southern United States, which corresponds to the distribution of the lone star tick. |

<table>
<thead>
<tr>
<th><strong>CLINICAL FEATURES:</strong></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>A typical allergic reaction to alpha-gal occurs 3–8 hours after the consumption of mammalian meat products with severe whole-body itching, hives and GI upset, and even anaphylaxis commonly with respiratory distress. Unlike most food allergies, it may be possible for the alpha-gal allergy to recede with time,</td>
</tr>
</tbody>
</table>
| **AGENT:** | *Amblyomma americanum* (Lone Star tick)  
*Ixodes holocyclus* (Australian paralysis tick) |
| **EPIDEMIOLOGY AND PATHOGENESIS:** | Alpha-gal allergy, also known as Mammalian Meat Allergy, an IgE-mediated reaction to *galactose-alpha-1,3-galactose* (alpha-gal), found in all mammals except humans and apes.  
Bites from the *lone star tick* in the US can induce this delayed allergic response triggered by the consumption of red meat products (but NOT fowl or fish). The allergy is most often seen in the central and southern United States, which corresponds to the distribution of the lone star tick. |
| **CLINICAL FEATURES:** | A typical allergic reaction to alpha-gal occurs 3–8 hours after the consumption of mammalian meat products with severe whole-body itching, hives and GI upset, and even anaphylaxis commonly with respiratory distress. Unlike most food allergies, it may be possible for the alpha-gal allergy to recede with time, |
| **DIAGNOSIS:** | A traditional skin prick allergy test for allergy to meat or detection of IgE antibody to alpha-gal |
# ALPHA-GAL (RED MEAT) ALLERGY

| **AGENT:** | *Amblyomma americanum* (Lone Star tick)  
*Ixodes holocyclus* (Australian paralysis tick) |
| --- | --- |
| **EPIDEMIOLOGY AND PATHOGENESIS:** | Alpha-gal allergy, also known as Mammalian Meat Allergy, an IgE-mediated reaction to *galactose-alpha-1,3-galactose* (alpha-gal), found in all mammals except humans and apes.  

Bites from the **lone star tick** in the US can induce this delayed allergic response triggered by the consumption of red meat products (but NOT fowl or fish). The allergy is most often seen in the central and southern United States, which corresponds to the distribution of the lone star tick. |
| **CLINICAL FEATURES:** | A typical allergic reaction to alpha-gal occurs 3–8 hours after the consumption of mammalian meat products with severe whole-body itching, hives and GI upset, and even anaphylaxis commonly with respiratory distress. Unlike most food allergies, it may be possible for the alpha-gal allergy to recede with time, |
| **DIAGNOSIS:** | A traditional skin prick allergy test for allergy to meat or detection of IgE antibody to alpha-gal |
| **TREATMENT:** | **AVOIDANCE OF RED MEAT IF CONFIRMED** + Supportive |

THE VIRULENT TICKS OF WISCONSIN

16 known species of ticks in Wisconsin, 5 species vector most of the tick-borne disease seen in the upper Midwest:

• *Ixodes scapularis* (the Deer tick)
  - *Borrelia burgdorferi*, *Borrelia miyamotoi*
  - *Ehrlichia muris*-like
  - *Anaplasmosis phagocytophilum*
  - *Babesia microti*
  - Powassan Fever Virus

• *Dermacentor variabilis* (Wood tick, American Dog tick)
  - Rocky Mt Spotted Fever
  - *Francicella tularensis*
  - Tick paralysis

• *Rhipicephalus sanguineus* (Brown Dog tick)
  - *Ehrlichia chafeensis*
  - Rocky Mt Spotted Fever
  - *Bartonella henselae*

• *Amblyomma americanum* (Lone Star tick)
  - *Ehrlichia chafeensis*
  - *Francicella tularensis*
  - *Coxiella burnetti* (Q Fever)

• *Ixodes marxii* (Squirrel tick)
  - Powassan Fever Virus
NEW TICK-BORNE PATHOGENS IN NORTH AMERICA

Borrelia miyamotoi
Borrelia mayonii
Heartland Virus
Rickettsia parkeri
STARI agent
Conclusion:

1. There are several hundred species and trillions of ticks throughout North America, tick-borne disease has been identified in every state and tick-borne disease are on the rise world-wide.
Conclusion:

1. There are several hundred species and trillions of ticks throughout North America, tick-borne disease has been identified in every state and *tick-borne disease are on the rise world-wide.*

2. Wisconsin and Minnesota have been particularly blessed.
Conclusion:

1. There are several hundred species and trillions of ticks throughout North America, tick-borne disease has been identified in every state and tick-borne disease are on the rise world-wide.

2. Wisconsin has been particularly blessed.

3. *Five species cause over 99% of identified disease* in the subarctic upper midwest, *Ixodes scapularis* (the Deer tick) reigns supreme in Wisconsin and Minnesota.
1. There are several hundred species and trillions of ticks throughout North America, tick-borne disease has been identified in every state and tick-borne disease are on the rise world-wide.

2. Wisconsin has been particularly blessed.

3. Five species cause over 99% of identified disease in the subarctic upper midwest, *Ixodes scapularis* (the Deer tick) reigns supreme in Wisconsin.

4. Health preservation starts with targeted efforts to prevent tick-borne disease.
TICK-BORNE DISEASES OF NORTH AMERICA

**Conclusion:**

1. There are several hundred species and trillions of ticks throughout North America, tick-borne disease has been identified in every state and *tick-borne disease are on the rise world-wide.*

2. Wisconsin has been particularly blessed.

3. *Five species cause over 99% of identified disease* in the subarctic upper midwest, *Ixodes scapularis* (the Deer tick) reigns supreme in Wisconsin.

4. Health preservation starts with *targeted efforts to prevent* tick-borne disease.

5. *Excellent diagnostic tests are available* for all of the tickborne diseases but take time, days to a week.
1. There are several hundred species and trillions of ticks throughout North America, tick-borne disease has been identified in every state and tick-borne disease are on the rise world-wide.

2. Wisconsin has been particularly blessed.

3. Five species cause over 99% of identified disease in the subarctic upper midwest, *Ixodes scapularis* (the Deer tick) reigns supreme in Wisconsin.

4. Health preservation starts with targeted efforts to prevent tick-borne disease.

5. Excellent diagnostic tests are available for all of the tickborne diseases but take time, days to a week.

6. Initial treatment in most cases is empiric and relies on a good history (a documented or plausible tick exposure, not necessarily a bite) AND a compatible syndrome.
TICK-BORNE DISEASES OF NORTH AMERICA

Conclusion:

1. There are several hundred species and trillions of ticks throughout North America, tick-borne disease has been identified in every state and *tick-borne disease are on the rise world-wide*.
2. Wisconsin has been particularly blessed.
3. *Five species cause over 99% of identified disease in the subarctic upper midwest, Ixodes scapularis* (the Deer tick) reigns supreme in Wisconsin.
4. Health preservation starts with *targeted efforts to prevent* tick-borne disease.
5. *Excellent diagnostic tests are available* for all of the tickborne diseases but take time, days to a week.
6. *Initial treatment in most cases is empiric and relies on a good history* (a documented or plausible tick exposure, not necessarily a bite) AND a *compatable syndrome*.
7. *Treatments are highly effective for all of the tick-borne disease, thank you God for DOXYCYCLINE.*
IS DOXYCYCLLINE SAFE IN SMALL CHILDREN?

Tetracycline staining of mandibular teeth caused by the ingestion of tetracycline when the patient was aged 3 years.
IS DOXYCYCLINE SAFE IN SMALL CHILDREN?
Absolutely YES


IS DOXYCYCLLINE SAFE IN SMALL CHILDREN?
Absolutely YES
(espec for RMSF, Neuroborreliosis, Anaplasmosis)


TICK-BORNE DISEASES OF NORTH AMERICA

Conclusion:

1. There are several hundred species and trillions of ticks throughout North America, tick-borne disease has been identified in every state and tick-borne disease are on the rise world-wide.
2. Wisconsin has been particularly blessed.
3. Five species cause over 99% of identified disease in the subarctic upper midwest, *Ixodes scapularis* (the Deer tick) reigns supreme in Wisconsin.
4. Health preservation starts with targeted efforts to prevent tick-borne disease.
5. Excellent diagnostic tests are available for all of the tickborne diseases but take time, days to a week.
6. Initial treatment in most cases is empiric and relies on a good history (a documented or plausible tick exposure, not necessarily a bite) AND a compatible syndrome.
7. Treatments are highly effective for all of the tick-borne disease, thank you God for DOXYCYCLINE.
8. We need to bring back *Lyme Vaccine*. 
TICK-BORNE DISEASES OF NORTH AMERICA

Conclusion:

1. There are several hundred species and trillions of ticks throughout North America, tick-borne disease has been identified in every state and all tick-borne disease are on the rise world-wide.

2. Wisconsin has been particularly blessed.

3. Five species cause over 99% of identified disease in the subarctic upper midwest, *Ixodes scapularis* (the Deer tick) reigns supreme in Wisconsin.

4. Health preservation starts with targeted efforts to prevent tick-borne disease.

5. Excellent diagnostic tests are available for all of the tickborne diseases but take time, days to a week.

6. Initial treatment in most cases is empiric and relies on a good history (a documented or plausible tick exposure, not necessarily a bite) AND a compatible syndrome.

7. Treatments are highly effective for all of the tick-borne disease, thank you God for DOXYCYCLINE.

8. We need to bring back Lyme Vaccine.
DO WE NEED A NEW WISCONSIN STATE INSECT?
(Other than *Apis mellifera*, the honey bee)
DO WE NEED A NEW WISCONSIN STATE INSECT?
(Other than *Apis mellifera*, the honey bee)

The *Mosquito* or the *Deer Tick*?
Thank You for coming to this year’s Wisconsin Chapter ACP Meeting.