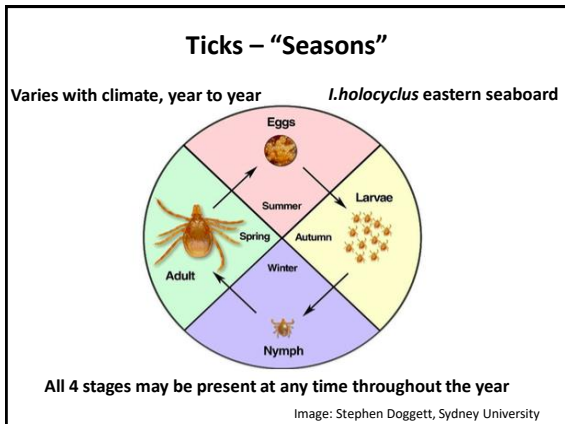
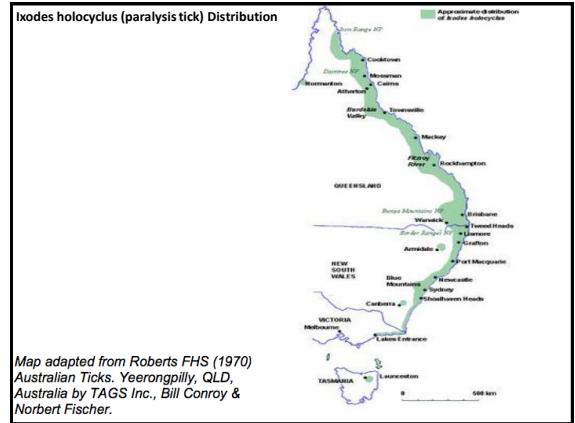
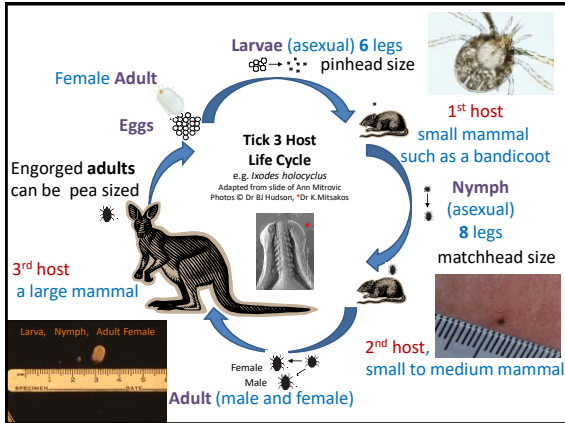
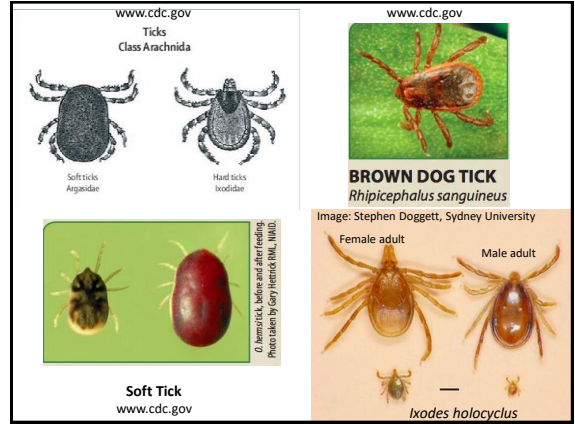


Tickborne Diseases

Dr Bernie Hudson
 Microbiology & Infectious Diseases, Royal North Shore Hospital, Sydney
 A/Professor, James Cook University, Townsville



Narrative review doi: 10.5694/mja17.00090

Tick-borne infectious diseases in Australia

Stephen R Graves, John Stenos

The incidence of tick-related medical problems in Australia is largely unknown. Appropriate diagnostic tests are not always available and, of all tick-related diseases, only Q fever is notifiable. Anecdotally, however, many patients present to their doctor after a tick bite. This narrative review focuses on tick-borne infections but also touches briefly on other medical problems caused by tick bites.

Summary

- Tick bites in Australia can lead to a variety of illnesses in patients. These include infection, allergic reactions, autoimmune disease, post-infection fatigue and Australian multisystem disorder.
- Rickettsial (*Rickettsia* spp.) infections (Queensland tick typhus, Flinders Island spotted fever and Australian spotted fever) and Q fever (*Coxiella burnetii*) are the only systemic bacterial infections that are known to be transmitted by tick bites in Australia.

Australian ticks	Tick species	Common name	Known human pathogen	Disease	Possible human pathogen
<i>Ixodes holocyclus</i>	Paralysis tick (scrub tick in Queensland)	<i>Rickettsia australis</i>	Queensland tick typhus	<i>Candidatus Neoehrlichia</i> spp.	
<i>Ixodes tasmani</i>	Common marsupial tick	<i>Coxiella burnetii</i>	Q fever	<i>Bartonella henselae</i> ; <i>Ehrlichia</i> spp.	
<i>Ixodes cornuatus</i>	Southern paralysis tick	<i>R. honei</i> subsp. <i>marmorii</i>	Queensland tick typhus	<i>Candidatus R. tasmanensis</i>	
<i>Amblyomma triguttatum</i>	Ornate kangaroo tick	<i>R. australis</i>	Australian spotted fever	<i>Bartonella</i> spp.	
<i>Bothriocroton hydrosauri</i>	Southern reptile tick	<i>C. burnetii</i>	Q fever	<i>R. gravesii</i> ; <i>Anaplasma</i> sp.; <i>Ehrlichia</i> sp.	
<i>Haemaphysalis novaeguinae</i>	No common name	<i>R. honei</i> subsp. <i>marmorii</i>	Queensland tick typhus	Flinders Island spotted fever	
<i>Haemaphysalis longicoms</i>	Bush tick (introduced, not native to Australia)	<i>R. honei</i> subsp. <i>marmorii</i>	Australian spotted fever	<i>Babesia</i> sp.	
<i>Omithodorus capensis</i>	Seabird soft tick			Virus	

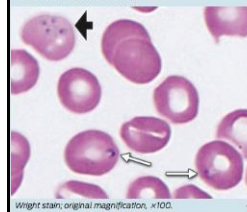
Tickbite - Sequelae

- Local reaction
 - With or without local lymph gland enlargement
- Allergic reactions
 - Generalised, Local, Red Meat Allergy
- Tickbite paralysis
 - Generalised, Local
- Agreed
 - Spotted Fever (Rickettsia), Q fever, Cellulitis
- Not agreed
 - Babesiosis, Borreliosis (incl. Lyme Disease), Ehrlichiosis
- NOTHING ? Other/Undetected

First report of human babesiosis in Australia

We report the first human case of babesiosis in Australia, thought to be locally acquired. MJA 2012; 196: 350-352
doi:10.5694/mja111378

1 Blood film from a 56-year-old man infected with *Babesia microti*, showing a tetrad form (black arrow) and single ovoid forms (white arrows)



Wright stain, original magnification, x100.

2 Diagnosing human babesiosis in Australia

When to suspect babesiosis
Clinicians should suspect babesiosis in patients in Australia who have haemolytic anaemia, thrombocytopenia, fever, an influenza-like illness and a history of at least one of the following:

- tick bites
- outdoor activities putting one at risk of tick bites
- transfusion of blood products
- overseas travel to a region where babesiosis is endemic.

How to proceed with the diagnosis
Thick and thin blood films should be examined for intraerythrocytic parasites (three sets of films should be taken, 8–12 hours apart). If the results of blood films are negative but the diagnosis is still suspected, antibody testing of serum and molecular testing of blood (by polymerase chain reaction) can be done.

Australian Spotted Fever (ASF)

Australian Spotted Fever

- All along the eastern seaboard
 - very **distinctive** clinical presentation
- Main species is *Rickettsia australis*
 - *rickettsia* are **obligate intracellular bacteria**
- Emerging species
 - *Rickettsia honei*
 - Others – *R. marmionii* (??CFS)
 - Strains or species?
- Clinical Manifestations (infected endothelial cells > **vasculitis**)
 - Similar to most spotted fever group (SFG) rickettsioses
 - Rash often **vesicular** with *R. australis*
 - Rash may be absent but **eschar is common**
 - Hospitalisation usually = lack of recognition
 - Few recorded deaths (~3)

Australian Spotted Fever

- 1946 **Andrew** (MJA) North Queensland
 - cluster of 12 cases (military)
 - first isolate of *Rickettsia australis*
 - QTT – **Queensland Tick Typhus**
- 1991 **Stewart** (MJA) Flinders Island in Bass Strait
 - febrile illness on the island related to tickbite
 - Eventual first isolate of *Rickettsia honei* (description Stenos et al 1998)
 - FISF – **Flinders Island Spotted Fever**
- 1991 **Sexton** (CID) - reviewed all ASF
 - FISF, QTT (*R. australis*)
- CMR update articles
 - 1997 Raoult
 - 2005 & 2013 Parola

CID – Clinical Infectious Diseases; CMR – Clinical Microbiology Reviews; MJA – Medical Journal of Australia

Table 6. Diagnostic criteria for **Mediterranean spotted fever** caused by *Rickettsia conorii* **Adaptable for ASF**

Criteria	Score ^a
Epidemiological criteria	
Stay in endemic area	2
Occurrence in May–October	2
Contact (certain or possible) with ticks	2
Clinical criteria	
Fever > 39°C	5
Eschar	5
Maculopapular or purpuric rash	5
Two of the above criteria	3
All three of the above criteria	5
Non-specific laboratory findings	
Platelets < 150 C/L	1
SCOT or SGPT > 50 U/L	1
Bacteriological criteria	
Blood culture positive for <i>Rickettsia conorii</i>	25
Detection of <i>Rickettsia conorii</i> in a skin biopsy	25
Serological criteria	
Single serum and IgG > 1/128	5
Single serum and IgG > 1/128 and IgM > 1/64	10
Four-fold increase in two sera obtained within a 2-week interval	20

SCOT, serum glutamate-oxaloacetate transaminase; SGPT, serum glutamate-pyruvate transaminase.

^aA positive diagnosis is made when the overall score is ≥ 25.

■ Fever + eschar + rash + tick bite or likely tick exposure = likely ASF

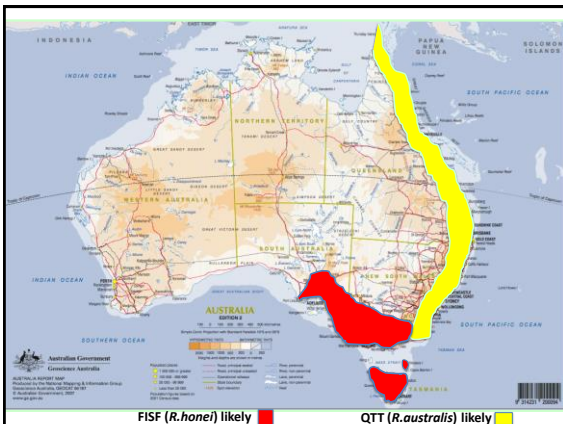
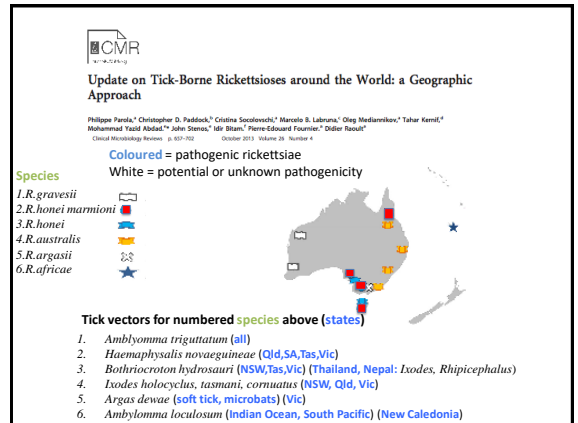
Some areas notorious – residents often know more than the new doctor!!



**Other Clinical Features
in ASF (NSW, Qld)**

FEATURE	FREQUENCY	N
Headache	88 %	58
Myalgias	82 %	62
Local nodes	79 %	53
Arthralgias	61 %	59
Neck stiffness	54 %	56
Anorexia	43 %	44
Photophobia	29 %	51
Gen. nodes	28 %	46
Sore throat	26 %	47
Nausea/ vomit	27 %	44
Confusion	22 %	55
Cough	19 %	43
Conjunctivitis	14 %	44
Splenomegaly	14 %	35*
Diarrhea	7 %	44
Jaundice	2 %	43

Often diagnosed with "viral illness", including meningitis, CSF normal



Spotted Fever

Diagnosis

Blood - Rickettsemia
 Short-lived
 Low titre
 Antibiotic affected
 Storage affected

Serology
 Can take 28 days to be (+)
 Cut-off 64 or 128
 IgM & IgG
 Rarely get a 2nd specimen

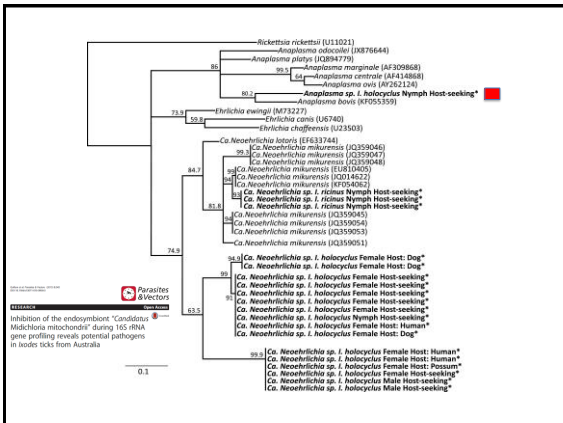
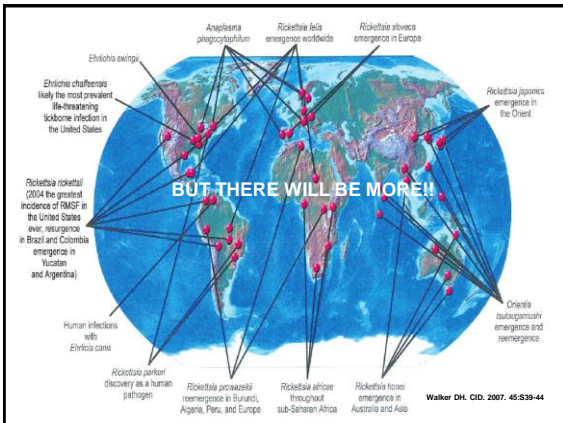
PCR of eschar (viral) swab is best
 Eschar is where the bugs are:

- Rickettsiae persist in eschar
- For weeks at high titre
- Not affected (?much) by antibiotics
- Original advice to biopsy or remove eschar unpractical and unacceptable

Other Rickettsial Illnesses

- Cross-reactive serology – with SFG and each other
 - Murine typhus - *R. typhi* (flea-borne)*^
 - Rickettsialpox - *R. akari* (mouse mite-borne) – rash vesicular^
 - Cat-flea typhus - *R. felis* (flea-borne)*^
 - Epidemic typhus - *R. prowazekii* (louse-borne)*^
 - Unclassified rickettsia?
- Usually **not** cross-reactive serology
 - *Orientia tsutsugamushi* (mite-borne)*^
 - *Orientia chuto* (mite-borne)*^

*Rash absent or if present not vesicular
 ^Eschar may be less obvious or absent
Bolded species occur in Australia



DISPATCHES

Francisella tularensis subsp. holarctica in Ringtail Possums, Australia

John-Sebastian Eden,¹ Karrie Rosa,¹ Jimmy Ng, Mang Shi, Qimning Wang, Vitali Sinchenko, Edward C. Holmes

The occurrence of *Francisella tularensis* outside of endemic areas, such as North America and Eurasia, has been enigmatic. We report the metagenomic discovery and isolation of *F. tularensis* ssp. *holarctica* biovar *japonica* from diseased ringtail possums in Sydney, Australia. This finding confirms the presence of *F. tularensis* in the Southern Hemisphere.

Tularemia is a highly infectious zoonotic disease caused by the bacterium *Francisella tularensis* that affects humans and other animals (1,2). Globally, tularemia has been identified in a wide range of animal hosts; rabbits and ro-

been largely considered tularemia-free. In 2003, a divergent *Francisella* spp. was isolated from a human foot wound in the Northern Territory, Australia (6), and has since been reclassified as *F. hispaniensis* (7). However, in 2011, a case of ulceroglandular tularemia was reported in an adult bitten by a wild ringtail possum (*Pseudocheirus peregrinus*) in western Tasmania, Australia (8). No isolate was obtained in this case, although infection by *F. tularensis* was suggested by both 16S rRNA sequencing and serology (8). Two additional cases of suspected human tularemia were reported in Tasmania in 2011, close to the site of the original infection; 1 of these involved exposure to ringtail possums (9). Together, these cases suggest a possible wider distribution of *F. tularensis* in the Southern Hemisphere and a potential reservoir in ringtail possums in Australia.

Emerging Infectious Diseases • www.cdc.gov/eid • Vol. 23, No. 7, July 2017

Tularemia

Gram negative coccobacillus transmitted by:

- Contact – rabbits/hares, possums (Tasmania possum bites)
- Tick bite
- Biting flies

Febrile illness, inoculation lesion, "plague", bioterrorism

- Gentamicin, streptomycin, ciprofloxacin

Francisella tularensis sub-species

- *Francisella tularensis* – most virulent
- *Francisella holarctica* – intermediate virulence
- *Francisella novicida* – low virulence
- *F. hispaniensis*
- *F. philomiragia*



FIGURE 229-3 Examples of primary lesions seen in ulceroglandular tularemia. **A**, Large cervical and submandibular lymph nodes in a young child; an ulcer was found under the hairline on her forehead at the site of a tick bite. **B**, Papule undergoing central necrosis with degeneration on the thigh of a middle-aged man. **C**, Inguinal adenopathy and suppurative mass in a young hunter who had carried a dead hare at his side. **D**, Nodule ulcer that was suspected of being syphilis or another sexually transmitted disease until the history of a recent tick bite was obtained by the infectious diseases consultant. **E**, Central ulcer and nodular adenopathy with a spirochoidal appearance in a 6-year-old girl who had a tick removed from the area of the ulcer 2 weeks before presentation. The nodes coalesced, suppurated, and required drainage after 3 days of gentamicin therapy. Cultures of the ulcer and nodes drainage both grew *A. tularensis*. **A** and **C** courtesy Dr. Joseph A. Borczyk, Louisiana State University Health Sciences Center, Shreveport, LA; **D** courtesy Dr. John W. King, Louisiana State University Health Sciences Center, Shreveport, LA; and **E** courtesy Dr. Robin Freeman, CovHealth Infectious Diseases Specialty Clinic, Springfield, MO.

Lyme disease: the tick-born epidemic the experts can't agree on

HOW IT'S TRANSMITTED

- Dogs, cats and bandicoots are the main carriers of *Borrelia burgdorferi* bacteria
- Infected ticks pass on the disease while sucking blood

SYMPTOMS

- Rash up to 100mm around tick bite
- Flu-like fever and aches
- Inability to concentrate
- Long term: Meningitis

doi:10.5694/mja16.00824 • Online first 31/10/16 Narrative review

Does Lyme disease exist in Australia?

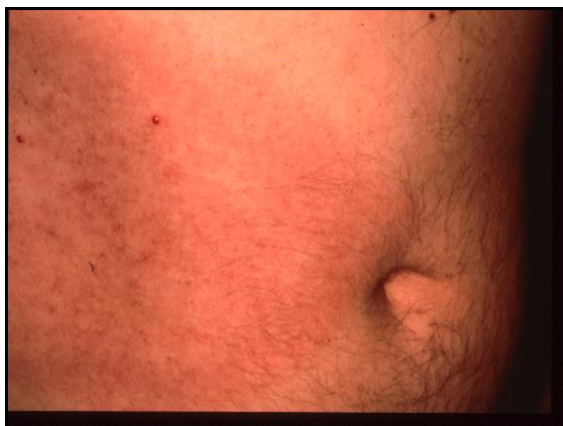
Peter J Collignon^{1,2}, Gary D Lum^{2,3}, Jennifer MB Robson⁴

Despite a number of reports of putative cases and a discussion across several decades,¹⁻¹⁰ locally acquired classic Lyme disease has not been identified in Australia. Despite intensive efforts, the bacteria that cause Lyme disease, *Borrelia* species collectively termed the *Borrelia burgdorferi sensu lato* (*B. burgdorferi* s.l.) complex, have not been cultured from any definite locally acquired cases of the disease. Further, Australia does not appear to have a competent tick vector for these species.^{11,12} Finally, bacterial DNA has not been definitively detected in patients for whom acquisition in a country where *B. burgdorferi* is known to be endemic could be excluded.^{13,14}

The controversy is not restricted to whether *B. burgdorferi* s.l. and a competent tick vector exist in Australia. We also need to consider whether chronic Lyme disease exists here. This concept does not require the aetiological agent to be metabolically active beyond maintaining a resting metabolism; it need only be present in the patient and viable. Further, the term "chronic Lyme disease" is not consistently defined; it has been applied to patients who present with active, previously untreated *B. burgdorferi* s.l. infections, to those who have persistent symptoms after being treated for Lyme borreliosis, to people who have had Lyme borreliosis in the past but whose current illness is unrelated to that infection, and to patients without any history of borreliosis. In Australia, substantial

Summary

- There is no convincing evidence that classic Lyme disease occurs in Australia, nor is there evidence that the causative agent, *Borrelia burgdorferi*, is found in Australian animals or ticks.
- Lyme disease, however, can be acquired overseas but diagnosed in Australia; most people presenting with laboratory-confirmed Lyme disease in Australia were infected in Europe.
- Despite the lack of evidence that Lyme disease can be acquired in Australia, growing numbers of patients, their supporters, and some politicians demand diagnoses and treatment according to the protocols of the "chronic Lyme disease" school of thought.
- Antibiotic therapy for chronic "Lyme disease-like illness" can cause harm to both the individual (eg, carnula-related intra-venous sepsis) and the broader community (increased antimicrobial resistance rates).
- Until there is strong evidence from well performed clinical studies that bacteria present in Australia cause a chronic debilitating illness that responds to prolonged antibiotics, treating patients with "Lyme disease-like illness" with prolonged antibiotic therapy is unjustified, and is likely to do much more harm than good.

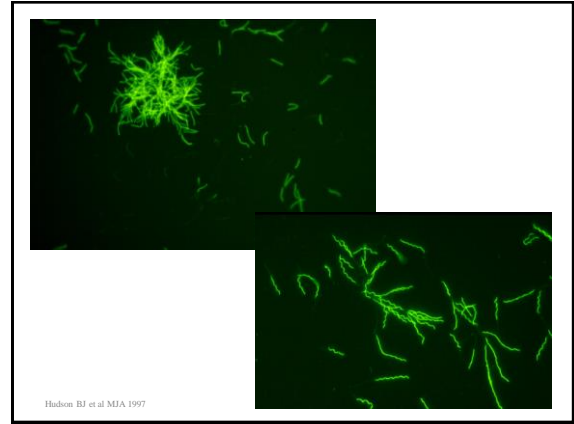


Lyme Disease

- LD is a tickborne infection that is caused by a spiral bacteria (spirochete)
- Spirochete that causes LD is a *Borrelia* species (**lyme borreliosis – LB**)
- The "hallmark" skin lesion of LD is called **erythema migrans**
 - Can appear at the tickbite site
 - Can appear elsewhere & be multiple
 - Tickbite (30-40%) or EM (10-25%) not always reported

Lyme - Clinical

- **Stage I - Skin, mild constitutional**
 - Erythema (chronicum) migrans (e.g. 2-30 days)
- **Stage II - Skin, CNS, Cardiac**
 - Multiple EM, aseptic meningitis, nerve roots & nerve pain, heart problems (e.g. wks to mths)
- **Stage III - Joints, CNS/PNS, Skin**
 - Arthritis (1/>1 jts) USA (80% if unRx) (days/wks)
 - Encephalomyelitis, Periph.neuropathy (mths/yrs)
 - Funny skin lesions (e.g. ACA years)



***Borrelia burgdorferi sensu lato* - Lyme**

- Eurasia associated
 - *B.afzelii*, *B.bavariensis*, *B.garinii*, *B.japonica*, *B.lusitaniae*, *B.sinica*, *B.spielmanii*, *B.tanukii*, *B.turdi*, *B.valaisiana*, *B.yangtze*
- USA only (initially thought so)
 - *B.americana*, *B.andersonii*, *B.californiensis*, *B.carolinensis*, *B.kurtenbachii*, *B.mayonii*
- Both Old & New World
 - *B.burgdorferi sensu stricto*, *B.bissettii*, *B.carolinensis*

Haemaphysalis sp. & Ixodes granulatus; all others Ixodid vector

Identification of a novel pathogenic *Borrelia* species causing Lyme borreliosis with unusually high spirochaetemia: a descriptive study. *Lancet Infect Dis* 2016; 16: 556-64

6 (5 blood & 1 synovial fluid) of 100,545 samples submitted to Mayo Clinic (Rochester MN) for Lyme PCR (over 11 years)

- *Borrelia mayonii*
- Illness is like relapsing fever
- Early negative serology
- Late positive serology but ...
- Only 1 positive IgG western blot

Figure 1. Diffuse macular rash in patient 1 and dark field microscopic visualization of a spirochete in patient 6. (A) Diffuse macular rash seen 4 days after onset of symptoms in patient 1. Rash was reported by patient's caregiver to involve the palm and sole, but this was not documented in the medical record. (B) Dark field microscopic visualization (400 \times magnification) of a single spirochete in diluted blood from patient 6.

Patient	Days from onset of illness to collection of specimen	<i>B burgdorferi</i> EA—whole cell	<i>B burgdorferi</i> EA-C6	<i>B burgdorferi</i> IgM immunoblot (number of bands detected/number of possible bands); specific antigens detected	<i>B burgdorferi</i> IgG immunoblot (number of bands detected/number of possible bands); specific antigens detected
Patient 1	6	Not available	Positive	Positive (2/3); 23, 41	Negative (1/3); 41
Patient 2	2	Not available	Equivocal	Negative (0/1)	Negative (0/3)
Patient 3	29	Not available	Positive	Positive (3/3); 23, 39, 41	Negative (2/3); 23, 41
Patient 4	104	Positive	Positive	Negative (0/1)	Negative (4/10); 18, 23, 39, 41
Patient 5	266	Positive	Positive	Negative (0/1); 23	Positive (5/20); 23, 39, 41, 45, 58
Patient 5 (plasma)	3	Negative	Positive	Negative (0/1)	Negative (0/3)
Patient 6	32	Positive	Positive	Positive (2/2); 23, 39	Negative (2/10); 23, 41
Patient 6 (plasma)	1	Negative	Negative	Negative (0/1)	Negative (1/10); 41

Specimens from patient 2 were not available for testing. IgM immunoblot was deemed second tier positive for *B burgdorferi* if two or more of a possible three bands (23–25 kDa) [OspC, 39 kDa (OspA), and 41 kDa (OspB)] are detected within 30 days of onset. IgG immunoblot was deemed second tier positive for *B burgdorferi* if two or more of a possible ten bands (28 kDa, 29 kDa (OspC), 29 kDa, 30 kDa, 33 kDa (OspA), 41 kDa (OspB), 45 kDa, 58 kDa (OspE), 66 kDa, and 93 kDa) were detected.

Table 2. Serological test results from patients infected with the novel *B burgdorferi sensu lato* genospecies

Other Borrelia Infections

- Relapsing Fever – Ixodid vector*
 - *B.miyamotoi* (Europe, USA & Asia)
- Relapsing Fever – Soft (Argasid) ticks
 - *B.crocidurae*, *B.dugesii*, *B.duttoni*, *B.hermsii*, *B.hispanica*, *B.parkeri*, *B.persica*, *B.recurrens* (louse), *B.turicatae*
- More acute illness - usually
 - False (+) LB all tests: EIA, IFA
 - False (+) WB (even with CDC criteria for Lyme Dx)
 - **Cannot be distinguished by serology from Lyme**

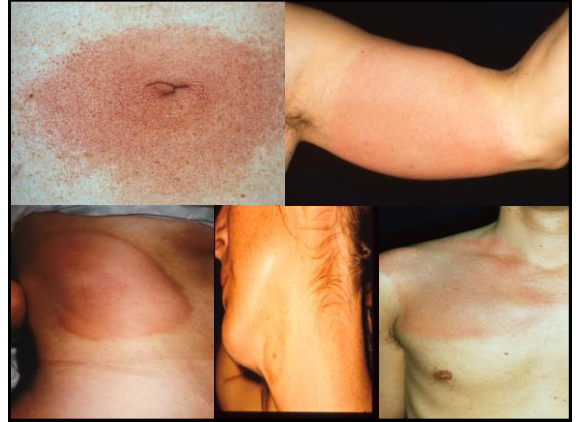
*TBRF & LB coinfection possible with same bite

Borreliosis

Australia?

Borrelia in Australia

- **Mackerras 1958**
 - *Borrelia* in bandicoots & kangaroos in Queensland
- **Carley & Pope 1962**
 - *Borrelia queenslandica* identified as cause of disease and death in a population of native rats in Queensland
- **Other borrelia**
 - *Borrelia anserina* (tick fever domestic birds)



Can you get LB in Australia ?

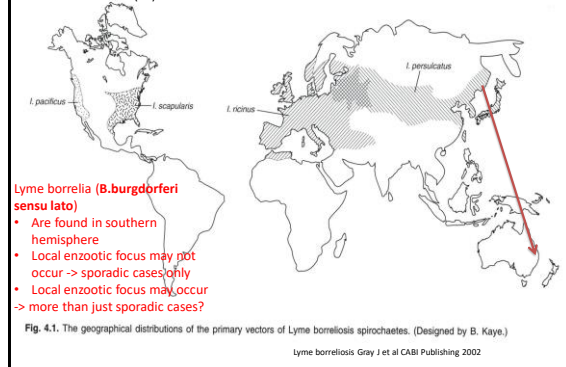
- **A body of clinical evidence**
 - But DDx EM from local reaction (?48 hr)
 - Not strong enough evidence by itself
- **Negative tick data**
 - Not necessarily mean absence
 - PCR with different primers & methods
- **Culture & PCR data**
 - Culture (+) case Pittwater v. Czech
 - Optimal methods & adequate specimens

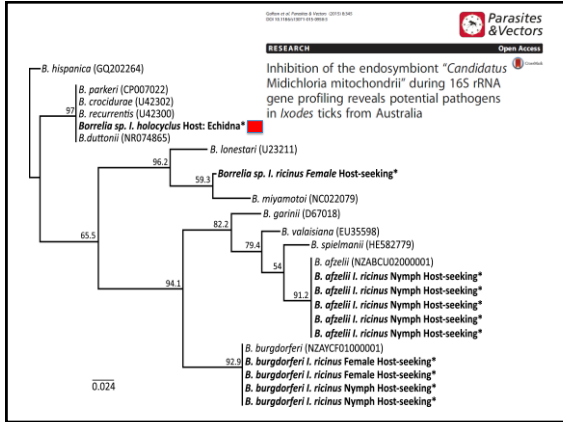
Can you get LB in Australia ?

- **Can you find anyone who has never travelled to a Lyme endemic area ?**
 - Not on Sydney's Northern Beaches
 - The organism can persist for years
- **What if patient & tick are both (+) PCR &/or culture ?**
 - Is that good enough even with travel ?
- **Combined Clinical, Animal, Vector**
 - Use better tools - PCR, culture, other
 - **Updated next gen. sequencing finding multiple potential pathogens but no *B.burgdorferi* s.l.**

What's It All Mean for Australia?

Bird migration - north-south spread of infected ticks - *B.burgdorferi* s.l. spirochetes (*B.garinii*)
Olsen B 1995 JCM 33(12) 3270-4





Lyme or Lyme-like or?

- There are a **lot** of organisms in Australian ticks
 - Including ticks that bite humans
 - Including a **relapsing fever borrelia** (RFB)
- Consider ***Borrelia miyamotoi*** (RFB) 1st found in ticks
 - Discovered **Japan 1995** –but “not a human pathogen”
 - 2011: first cases Europe (meningitis, fevers, rash)
 - 2013: illness in LB(-) and LB(+) patients USA LB areas
 - 2017: common in LB ticks in LB endemic areas
 - 2017: LB-like illness agreed, best test ?PCR
- History would indicate Oz RFB **will** be a pathogen

Lyme or Lyme-like or?

- ***Borrelia miyamotoi*** (RFB) illness
 - Suspected in USA 2006 (Fish D et al)
 - Research funding calls ignored...
 - ... until 2011 cases reported from Europe
- Australian RFB “could be a human pathogen”
 - **Research needed via GPs in tick areas**
 - **With laboratory support**
 - **to define illness, incidence, case management, preventive strategies**

Lyme Borreliosis

Diagnosis

Lyme “Classic” - Diagnosis

- **Still a clinical diagnosis**
 - Gardner P. JAMA. 2000; 283:658-9
 - Wang G et al. JCM. 1999; 12:633-53
- **Antibody tests – the usual test for diagnosis**
 - screening tests – EIA IgM and IgG
 - “confirmatory” Western Blot
 - False -ves & false +ves
 - C6 specific assays best (no WB needed)
 - **Uncertain how relevant overseas testing is to Australian tickborne illness**

Lyme “Classic” - Diagnosis

- **Culture (sensitivity)**
 - EM up to 80%
 - Blood (early) up to 40%
 - CSF and other specimens low yield
- **PCR (sensitivity)**
 - EM up to 60% (not homogeneously present)
 - CSF up to 25%
 - Joint fluid up to 85%
- **Histopathology**
 - Silver stain EM up to 40%

Lyme Borreliosis

Treatment

Lyme "Classic" - Treatment


- **Early treatment (?within 12 weeks)**
 - Most people recover fully
- **Oral antibiotics (mild or early disease)**
 - 14-28 days (doxy 200mg/d, amoxil 1.5g/d)
- **Intravenous antibiotics (neuroborreliosis, late disease)**
 - 14-28 days (ceftriaxone 2g/d) - then orals?
- **Antibiotics after tickbite**
 - ? **No** - 200mg Doxycycline (USA - Ct) in 72hrs
- **Delayed treatment may prevent cure**
 - **Controversial but true** – cystic v. spirochetal forms

Tickbite Survey

- How many attendees see **patients with tickbite**
- How many see **> 5 patients with tickbite annually**
- Participate in a GP surveillance network
 - Paid participation

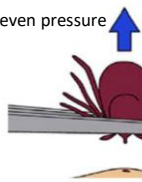
Prevention

Removing a Tick

 Avoid folklore remedies such as "painting" the tick with nail polish or petroleum jelly, or using heat to make the tick detach from the skin. Your goal is to remove the tick as quickly as possible—not waiting for it to detach.

- **Fine-tipped tweezers** - grasp tick as **close** to the skin's surface as possible

- Pull **upward** - steady, even pressure



DoH
Pyrethroid aerosol repellent **first** is OK

ASCI
Spray **first** is best:

- Aerostart
- Wart spray (ether)
- Liquid N2 (Doctor)

Graphic & method: www.cdc.gov

Removing a Tick

- **Don't twist or jerk the tick**
 - can cause mouth-parts to break off & remain in the skin
 - if this happens, remove mouth-parts with tweezers
 - if unable to remove mouth-parts easily with clean tweezers, **leave it alone** & let the skin heal
- **After removing the tick**
 - thoroughly clean the bite area & your hands
 - use rubbing alcohol, iodine scrub, or soap & water
 - save tick (in pot with alcohol or a blade of grass)
 - dispose of in tape, tissue, flush down toilet
 - **never** crush it with your bare hands

Avoiding Tickbite

- Wear **light-coloured** long-sleeved shirts & long trousers
 - ticks seen more easily on light-coloured clothing
- **Tuck in!**
 - tuck shirt into trousers & trousers into long socks
- Wear a wide-brimmed **hat** &/or **overalls**
 - can be treated with **permethrin**
- **Brush** clothing before coming indoors
 - removes ticks
- Using an effective insect **repellent**
 - DEET, icaridin, extract of lemon eucalyptus (Mosiguard®)
- Carefully check for ticks in “favoured” sites
 - neck, scalp, groin, axillae in the shower/bath (“**tick check**”)