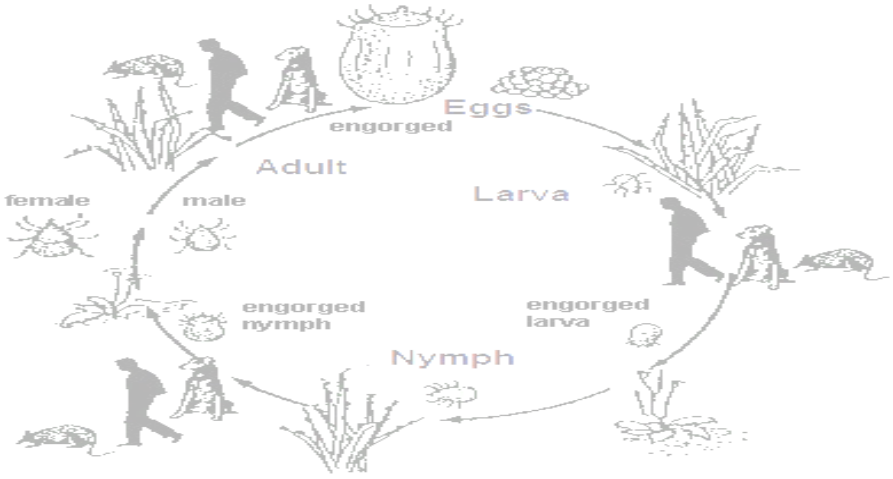
Lyme Disease:
A Counter Argument of the Australian Government's Denial



By
Karen Smith
B. Psych (Hons)

ISBN: 978-0-9923925-4-3

This document is to be credited as: “Lyme Disease: A Counter Argument of the Australian Government’s Denial”

Copyright notice

This work is licensed under the Creative Commons Attribution 3.0 Australia License (CC BY 3.0).
To view a copy of this license, visit http://creativecommons.org/licenses/by/3.0/au/

Please note: The majority of the information in this counterpoint argument has been displayed on the Lyme Australia Recognition and Awareness (LARA) website since July 2012, and was copyrighted at that time. The Creative Commons copyright notice is due to distribution of this research in pdf format in 2013. As noted on the website: The information is intended to be disseminated in order to promote awareness and further research in Australia; though I do ask that the source (myself) of the information is referenced appropriately. Information may not be used, distributed, or reproduced for any commercial purpose. Thank you. Karen Smith, B Psych (Hons)

Contact:

Enquires about this document are welcome at:

Lyme Australia Recognition and Awareness

Email: lymeaustralia@live.com.au

Postal: PO Box 822, Sarina QLD 4737

About Lyme Australia Recognition and Awareness (LARA)

Lyme Australia Recognition and Awareness (LARA) was founded by independent researcher, Karen Smith, B Psych (Hons). As well as her research work, Karen provides support and advocacy to patients and families living with Lyme disease through patient support forums and raising awareness of Lyme through organising and participating in awareness and protest events, both in the national and international arena.

Acknowledgments

Thank you to Brendan D for his input on the first draft of this counter-argument. Brendan was a Victorian Lyme patient who sadly lost his battle and died in August 2011. (Research, including this counter-argument was started in early 2011, though has been stopped and started many times due to the author’s health and treatment needs). Despite being so unwell, Brendan was always there to help, and will be forever remembered. Thank you also to the numerous other people who have read and offered helpful suggestions throughout this lengthy research process, in particular with the final draft; Tania Perich, Tony James, Amber Smith, Sherryn Jackson, Janice Foster and David Trathen.

References

As noted on the website: Any information with regards to Lyme disease that is freely available at numerous locations on the internet has not been referenced. For specific facts/arguments, see the reference list.

A further note for this hard copy format: As this research was originally started with the intention of expanding and viewing on a website platform, the reference section is separated into segments (content headings) for ease of updating information, and whilst not conventional referencing style, links have also been provided to where the journals/information can be accessed on-line.

Executive Summary

Lyme disease (LD) is a disease caused by an infection from the borrelia species of bacteria. As there are numerous species of borrelia underlying Lyme disease, it is also known as borreliosis, and in continents such as Europe and Asia, where the species responsible for neurological symptoms are more common, neuroborreliosis. In the initial stages Lyme may simply present with flu-like symptoms, however as the length of time of the infection increases, the disease may appear as something more chronic and difficult to treat due to the bacteria disseminating throughout the body's tissues and organs. In order to maximise the potential for early detection, treatment and full recovery, the recognition of the possibility of Lyme as a differential diagnosis is essential.

Lyme is the fastest growing vector borne disease in the world. In the United States of America (USA), the Centre for Disease Control (CDC) recently released figures of around 300,000 new cases of Lyme disease each year in America alone. Although there is no official collection of data, various sources reveal that the number of cases for the other continents, ie: Europe, Africa and Asia range from around 200,000 to 300,000 cases per year also. According to the Australian Health departments, Australia is the one continent exempt from this disease that affects over half a million people around the world each year. The 'No Lyme in Australia' stance is maintained, despite thousands of clinically suspected cases that date back as far as the 1980's. This position stems from research that was conducted on ticks and animals collected from New South Wales (NSW) over twenty years ago. The NSW Health department appears to be the only State with an official "policy" on the existence, or lack there-of, with regards to Lyme disease, though despite the lack of research in any other State or Territory, the 'No Lyme' position is cited by health departments Australia wide.

This counter-argument examines the research from the Department of Medical Entomology (DME), Westmead Hospital, NSW, that underlies the denial in Australia, the majority of which was published in a paper by Russel et al., in 1994, "Lyme disease: search for a causative agent in ticks in south-eastern Australia". As the research paper is also the basis of the information with regards to LD that is on the Department of Medical Entomology (DME), Westmead, website, various information from this site is also briefly looked at. The aim is simply to highlight that the research methodology has a number of flaws, and that the researchers, who were new to Lyme testing and analysis methods, utilised procedures for the first time. Procedures, which even specialist laboratories in other countries, have found difficulties with. There will always be problems encountered within research; it is the learning from these difficulties that allow the moving forward in science and knowledge. However, whilst most studies address the problems that arise and offer possible alternative viewpoints or conclusions, the disturbing factor in Westmead's investigations is that despite being new to the field of Lyme research, the Westmead team assessed its own investigations as "expert" and ignored all other research studies that revealed there is a high probability that Lyme is in Australia.

In reference to the information on Lyme disease, the DME website notes that the 1994 study was a result of "a multidisciplinary investigation" that began in 1988 to investigate the existence of Lyme disease in "coastal New South Wales". The individual components - clinical and serological studies, reservoir host, and vector study – are explored in detail in this counter-argument.

Clinical & Serological Studies: Despite over 1000 suspected cases per year at the time of the study, the conclusions reached were that patients were not positive according to international criteria. This section examines the blood tests performed, outlining the fact that they are not appropriate for Australian patients. The international criteria that the DME and Russel et al., refer to was established for surveillance purposes in the USA. The Western Blot (WB) criteria were developed in order to monitor the activity of the burgdorferi sensu stricto species of borrelia, which is the most common species of borrelia underlying Lyme in the USA. There is alternative European WB criterion that is recommended for use outside of the USA where other species from the borrelia sensu lato class, such as afzelii, garinii, and valaisiana are more prevalent than B burgdorferi ss.

Due to known variations and the differences in the immunological response to various borrelia species, the criteria for a positive WB test is vastly different in Europe. These differences have been known since the early 1990's, though for some reason the literature on the diversity of borrelia, and even the advice from the CDC, that the USA criteria should not be used outside of America, seems to be totally ignored by Westmead and Australian pathology laboratories. With the bird migratory pathways (it has been known since the 1980's that migrating seabirds, and the ticks they carry play a role in spreading borrelia), and the knowledge that many animals in Australia were imported from Europe and Asia, it would be more appropriate to utilise the European guidelines with regards to what is considered a positive WB test for Lyme in Australia.

Reservoir Host Studies: Seventeen (17) animals were examined by the Westmead team. As various species of borrelia are found within organs, rather than contained to the skin, ear punch biopsy of animals is not sufficient. The identification of reservoir hosts within the environment is crucial to identifying the pathogens present. Indeed, borrelia was found in the blood of Australian mammals, including rodents, cattle, kangaroos and bandicoots, in a Commonwealth Scientific and Industrial Research Organisation (CSIRO) study by Mackerras in 1959. Curiously this information is given little regard by DME. Seventeen animals can only be described as extremely limited in scope.

The conclusion and the statement on the DME Website (NSW Health) is: "None of the mammal species identified as reservoir hosts in the northern hemisphere are present in Australia". This is simply not correct. The primary reservoir host for borrelia in America is the white footed mouse; it is a mammal, belonging to the rodentia species of the *Muridae* family. Whilst we do not have any whitefooted mouse in Australia, over 20% of the mammal species belong to the *Muridae*, rat and mouse family. This includes the Australian Long-haired Rat, which in 1962 were the subject of a study in Richmond, north-west Queensland in which a new species of borrelia was identified and subsequently called Borrelia Queenslandica. This section also briefly looks at four mammal species that have in fact been shown to be reservoir hosts in the northern hemisphere and have been introduced and are established in Australia. These include: Black Rats, Brown Rats, House Mouse, and European Hares. Many other mammal species are known reservoir hosts for borrelia, including foxes, dogs, cats, horses and cattle. Other animal species such as birds, which include the European blackbird, mallard duck and turkeys that have been introduced into Australia, are also known reservoir hosts of the borrelia bacteria underlying Lyme disease.

Vector (Tick) Studies: The result of the research conducted on the ticks collected from the NSW coastline between 1990 and 1992 continues to be the primary basis of denial in Australia today. Of the 12, 000 ticks utilised in the study over half of them were larvae, leaving less than 6000 ticks that would have had a blood meal and have potentially been infected. No other study in the world uses larvae to ascertain the continents infection rates of borrelia. While 6000 ticks may still seem to be a relatively large number, it is not so when you consider that infection rates of ticks from different environmental areas and locations can vary anywhere from zero to ninety percent. The ticks in this study were collected from a small ecological niche of the NSW coastline that accounts for less than one eighth of Australia's entire coastline. It ignored not only other ecological areas such as pasture or mountain areas in NSW, but also the seven other States and Territories of Australia.

This section looks at the various methods used to ascertain infection within the ticks collected and argues that there were numerous problems contained within the study. Specialist laboratories all over the world have issues with the medium and culturing of spirochetes, and yet the researchers at Westmead hospital attempted this procedure for the first time for the purposes of this 1994 study. This counter-argument also presents the likelihood that what the study referred to as spirochete-like objects (SLO's), were indeed spirochetes, rather than contaminants of the culture as they concluded. The tick species that the SLO's were cultured from included the Paralysis tick (*Ixodes holocyclus*), Wallaby tick (*Haemaphysalis bancrofti*), Bush/Scrub tick (*Haemaphysalis longicornis*) and Snake tick (*Amblyomma morelia*).

A few points as to why these findings should have encouraged further research, rather than simply dismissed the existence of Lyme include: *I holocyclus* - As well as SLO's cultured from this species in this study, spirochetes were also cultured from *I holocyclus* ticks collected from the Hunter Valley and Manning River district of NSW in research by Wills and Barry in 1991 ; *H bancrofti* - In Wills and Barry's research, spirochetes were also cultured from the *Haemaphysalis* species. The *H bancrofti* tick not only attaches to wallaby's, its hosts also include kangaroos. In 1959, Mackerras reported the presence of borrelia in Australian animals, including kangaroos. ; *H longicornis* - is a vector of borrelia in China. It is also the tick species infesting a herd of cattle in which positive serology for borrelia was reported in a cow in Camden NSW in 1989. ; *A morelia* - Snakes are capable reservoir hosts of the borrelia species B. lusitaniae. This is a species of borrelia that might be expected along the coastline as it is carried by migrating seabirds. ; The Seabird tick (*I Uriae*) is a known vector of borrelia, it is found world wide – including Australia. ; Whilst it was originally presumed that only a small number of tick species were capable vectors of borrelia, it is now known that over two dozen species of ticks are involved in the borrelia cycle. This includes various species of ticks from the *Ixodidae* family, including *Ixode, Haemaphysalis* and *Amblyomma* species.

With increasing Lyme awareness in the last two years in Australia, the number of Lyme patients being diagnosed is rising at a rapid rate. Sadly, due to the denial of its existence in Australia, many of these people have been sick for a number of years. A prompt diagnosis is the best scenario for a rapid and full recovery from the Lyme bacteria. It is hoped that this counter-argument logically presents the real and potential problems and inconsistencies with the research by Russel et al., and that it highlights the fact that the twenty year "freeze" on government research of Lyme in Australia needs to be addressed.

Contents

A Counter-Argument of the Australian Governments' Denial of Lyme

Introduction

Lyme is a disease that is the result of an infection from spirochete bacteria of the Borrelia genus. In the initial stages Lyme disease may simply present as flu like illness, however as the length of time of infection increases it can disseminate to become a multi-systemic inflammatory illness that may involve all organs, as well as the musculoskeletal, the peripheral and central nervous systems. When the infection is detected and treated early, the prognosis for a full recovery is excellent. Unfortunately, due to the various ways the illness can manifest, the lack of definitive laboratory tests for diagnosis and more importantly the overall lack of awareness surrounding Lyme disease, many people may go undiagnosed for long periods of time, rendering the treatment and recovery process much more complicated.

Lyme disease may also be called Lyme borreliosis (to account for the numerous species of borrelia associated with the disease) or neuroborreliosis, due to the more neurological manifestations associated with some borrelia species, such as B. garinni in Europe. A more detailed explanation of Lyme disease can be seen in this counter-arguments complimentary report, 'Lyme Disease / Borreliosis: An overview of Lyme and direction for further research required in Australia' (1).

Australian Health Department Information Regarding Lyme

Since as far back as the 1980's in Australia, there have been over one thousand patients tested each year for suspected Lyme disease. Despite the enormous number of clinically suspected cases, as well as hundreds of patients diagnosed in Australia, whose experiences were highlighted in a report released by the Lyme Disease Association of Australia (LDAA) in 2012 (2), the current position of the Australian Health Department continues to be that there is no evidence that Lyme disease is a threat to public health. This stance is based on one study by Russel et al., 1994 (3) and ignores all other research that was done prior to, or around the same time as the Russel et al research, and whose conclusions were highly suggestive of the fact that the borrelia bacteria that underlies Lyme is in Australia (eg: 4-14).

That the denial continues to be based on one study - *"*Lyme disease: search for a causative agent in ticks in south-eastern Australia*"* (Russel et al., 1994) - can be seen in the response that an Australian Lyme disease patient received in 2010 from then (2009-2011) Health Minister, Carmel Tebbutt. Her response explained that the relevant species *"have not been isolated in surveys of ticks collected in south-eastern Australia. Until there is solid evidence to indicate that locally acquired Lyme disease is a significant public health matter in Australia, specific measures to educate the general public or clinicians are difficult to justify."*

The Russel et al., 1994 study was also cited as evidence against Lyme disease in Australia by the "Lyme disease expert panel" (15) that convened in April 2011 (information about the expert review panel and its findings was not released until late 2012). One of these expert panel members, Dr Jeremy McAnulty of the NSW Health Department, consistently references this study in the "not a lot of evidence" stance with regards to Lyme being acquired in Australia. In addition, the results of this study are also the basis of what appears on the New South Wales (NSW) Department of Medical Entomology (DME) website (16).

The following report addresses both the information on the DME website, and the details of the Russel et al., 1994 study, providing further details and alternative viewpoints to the information and conclusions that they have provided. The information examined below will be identified as either being from the Department of Medical Entomology (DME) Website, or from Russel et al., study (with the page number). Quotations or information from these sites, article are italicised. Please note, Russel et al., has been published in numerous journals. In the following information, the page numbers referenced are those from:

Russell RC, Doggett SL, Munro R, Ellis J, Avery D, Hunt C, Dickeson D. (1994) Lyme disease: search for a causative agent in ticks in south-eastern Australia. Epidemiology and Infection 112: 375-384 (3).

DME Website : acknowledges that Lyme disease is the "*most frequently reported human tick-borne infection worldwide"*, it goes on to say that **"***it has been reported from every continent (except Antarctic), although doubt remains as to whether it occurs in the southern hemisphere in general, and in Australia in particular".*

This doubt is based on the fact that - *"In 1988 at Westmead Hospital, a multidisciplinary investigation of putative LD in coastal New South Wales began, encompassing clinical, serological, vector and reservoir host studies."*

The individual components - clinical and serological studies, reservoir host, and vector study – of these multidisciplinary investigations are examined in the following sections.

Clinical & Serological Studies

DME website: *"Despite clinical cases being reported from the early 1980's, there has been no confirmation that the disease occurs in Australia."*

Russel et al 1994 (Pg 376): *"The first Australian cases of a syndrome consistent with Lyme disease were reported from the Hunter Valley region of New South Wales in 1982. Further clinical cases were reported in 1986 from the south and central coast of NSW. In Queensland, in 1986-1989, the State Health Laboratories tested 1,247 patients for antibody response to B. burgdorferi, using an indirect fluorescent antibody test (IFAT), and reported 186 with positive (>64) titres". In 1988 a serological diagnostic service for Lyme disease was started at Westmead Hospital. Enzyme linked immunosorbent assay (ELISA) for IgG and IFAT for IgG and IgM, were used with antigens derived from a North American strain (B31) of B. burgdorferi. From 1988 to 1992, specimens were tested from 2,446 patients referred with suspected clinical Lyme disease; only 66 (2.7%) showed positive results by both methods indicating possible Lyme disease". These figures include seven patients infected outside Australia. More recent data from one of us (DD) indicate that to August 1993, 75 (2.2%) of 3458 local patients were positive for IgG by both methods. Less than 1% of the patients referred with suspected Lyme disease conformed with the United States national surveillance case definition for Lyme disease."*

DME webpage: The number of suspected cases referred to Westmead for testing by 1994 rose to 4,372. *"From 1988 to 1994 at Westmead Hospital, 78 (1.8%) of 4,372 from local patients with suspected LD were positive for IgG by ELISA and IFAT. All 78 were tested by WB, using North American and European strains of Borrelia; 46 sera showed one or more bands. None, including those with putative late stage disease, showed more than 4 specific bands and thus were all negative by international criteria."*

When you look at these test results, only a little over two percent of the patients tested were positive, and less than one percent of the patients were positive by international criteria - Though it must be noted that the DME Website differs to what was published in the 1994 study, as it notes that "all were negative by international criteria".

So what is the problem? Why do people insist Lyme is in Australia with so few patients testing positive to International Criteria?

- Lyme disease is a clinical diagnosis: Suspected clinical cases averaging around 1,000 patients per year (From as far back as the 1980's) indicates that something is going on and further research is urgently needed.
- CDC International criteria is surveillance, not clinical criteria.
- Due to species variations of the borrelia bacteria responsible for Lyme disease/borreliosis, the "international criteria" is not recommended for use outside of America (where B. burgdorferi ss is not the primary species underlying Lyme). Whilst the European, US-CDC surveillance criteria requires two tier testing – ELISA, then Western Blot (WB) – the interpretation of a positive WB is vastly different in each continent.

Lyme disease IS a CLINICAL Diagnosis

Clinical diagnosis is not a new phenomenon to the medical world. Diseases such as Parkinson's, Alzheimer's, Multiple Sclerosis, and Motor Neurone all rely on the clinician's interpretation of medical history, symptoms and response to treatment for diagnosis. As the underlying cause for most of these diseases is unknown, they do not have an available diagnostic test to definitively rule in or out the diagnosis. Whilst the cause of Lyme disease is known, the current available testing methods are inadequate due to a number of reasons, including the diversity of the borrelia bacteria that can cause Lyme disease and the lack of standardisation of testing methods, which renders the diagnosis of Lyme primarily as a clinical diagnosis that "may be" supported by blood tests.

In Australia, between 1988 and 1992, there were 2,446 patients suspected of Lyme disease that were tested at Westmead Hospital, NSW. By 1993 the number of suspected cases and tests had risen to 3,458 and again to 4,372 by 1994. Add to these figures the Queensland patients that a 1994 study of Russell et al., mentions, and these show that there was at least 6000 suspected cases of Lyme disease in Australia in 1994. There is no further information publicly available after this date to indicate whether or not that the growth of new suspected cases continued to average approximately 1,000 per annum as per the previous couple of years. With so many suspected clinical cases each year, it is baffling how it can be logically asserted that Lyme disease does not exist in Australia.

CDC International Criteria is Surveillance, NOT Clinical Criteria.

The International Criteria that Russell and others refer to is that of the CDC in the United States. It was developed for surveillance, and not clinical purposes. The CDC Morbidity and Mortality Weekly Report (1) states "This surveillance case definition was developed for national reporting of Lyme disease; it is NOT appropriate for clinical diagnosis**"** (pg 20). (Emphasis not added) The case definition includes serological results, "For the purposes of surveillance, the definition of a qualified laboratory assay…" (2).

Testimony by Paul Mead, Medical Epidemiologist with the CDC, given to the Connecticut Department of Public Health and the Connecticut Attorney General's Office at a hearing regarding CDC's Lyme Disease Prevention and Control Activities in 2004 notes "A clinical diagnosis is made for the purpose of treating an individual patient and should consider the many details associated with that patient's illness. Surveillance case definitions are created for the purpose of standardization, not patient care**"** (3).

"No surveillance case definition is 100% accurate. There will always be some patients with Lyme disease whose illness does not meet the national surveillance case definition. For this reason, CDC has stated repeatedly that the surveillance case definition is not a substitute for sound clinical judgment. Given other compelling evidence, a physician may choose to treat a patient for Lyme disease when their condition does not meet the case definition" (3).

CDC Western Blot Criteria was developed for use in America (B. burgdorferi ss species)

The problem of testing and species variations of borrelia has been long documented. Just a few of the known problems are:

- "The presence of at least 3 different species in Europe renders the diagnosis of Lyme borreliosis by serological testing complicated and difficult" (4)
- "The antibody response is more limited in European borrelia species; with these lower responses leaving the specificity and sensitivity of serodiagnostic tests lower" (4)
- "….it is clear from all accumulated studies on Lyme borreliosis serology that serological testing should be used as a support of clinical diagnosis rather than a confirmation" (5: Pg S195).

Despite the well known problems with testing due to numerous borrelia species worldwide, the first line of testing in Australia still utilises the B. burgdorferi ss species antigens. NSW Health does not acknowledge the differences in the European and United States Western Blot (WB) criteria, and they continue to ignore the fact that Lyme disease is a clinical diagnosis that is supported, rather than confirmed or denied by blood tests.

Advice for testing, and the wording in the Fact Sheet, 'Lyme Disease - Testing Advice for NSW clinicians' (6) and the 'Lyme Disease Fact Sheet' by the Institute of Clinical Pathology and Medical Research (ICPMR), Centre for Infectious Diseases and Microbiology Laboratory Services, Westmead (7), is very ambiguous. The 'Lyme Disease - Testing Advice for NSW clinicians' (6) implies that antigens from all three species are utilised in testing, yet based on the ICPMR fact sheet (7), it appears that it is only the blood/samples that are ELISA positive with B. burgdorferi species/antigens, will then go on to be further tested on the WB (and therefore with the inclusion of European antigens).

The 'Lyme Disease - Testing Advice for NSW clinicians' fact sheet notes*: "The recommended testing strategy follows European and US-CDC guidelines for two-step serological testing with a screening immunoassay and a confirmatory immunoblot for antigens from Borrelia burgdorferi sensu lato genospecies (including B. afzelii, B. garinii)".*

The Institute of Clinical Pathology and Medical Research (ICPMR), Centre for Infectious Diseases and Microbiology Laboratory Services , Westmead, Fact Sheet on Lyme Disease states : *"The screening test is an ELISA to detect combined B. burgdorferi IgG and IgM. The sensitivity of this kit is as high as 100% but specificity may be only 68% (unpublished data). False positive results may occur when the patient has other spirochaete diseases such as syphilis, leptospirosis and relapsing fever or has mononucleosis, lupus erythematosis or rheumatoid arthritis.*

All sera with positive or equivocal results on screening are tested by the Western immunoblot technique to determine specific IgG antibodies to particular proteins of B. burgdorferi (USA strain) and B. afzelii (European strain). At least five specific IgG immunoblot bands are required to confirm true Lyme disease after the first few weeks of infection (F.Dressler et al. J Infect Dis 1993;167:392-400) as recommended by the Second National Conference on Serologic Diagnosis of Lyme Disease, Centers for Disease Control,USA, 1994."

The ICPMR fact sheet notes, *"The screening test is an ELISA...The sensitivity of this kit is as high as 100% but specificity may be only 68% (unpublished data)".* What it doesn't say is that the ELISA as a screening test is only as sensitive as to the antigen/species of borrelia tested for and the sensitivity of this test can range from as low as 30% depending on length of infection and what testing kits are used (8). Ang and others (2011) note "ELISAs and immunoblots for detecting anti-Borrelia antibodies have widely divergent sensitivity and specificity, and immunoblots for detecting anti-Borrelia antibodies have only limited agreement" (9).

The statement in the ICPMR Fact Sheet "*At least five specific IgG immunoblot bands are required to confirm true Lyme disease after the first few weeks of infection (F.Dressler et al. J Infect Dis 1993;167:392-400)",* is also a little confusing for a various reasons: Its reliance on American (rather than European) WB interpretation; the time frame for testing positive; the reference to "true" Lyme disease.

The European and US-CDC guidelines do recommend two-step testing – that is correct. However, despite being known since the 1990's, what is not mentioned, and indeed what is not acknowledged or followed through anywhere in Australian testing laboratories is the fact that the European Western Blot (WB) criteria is very different. Whilst the US surveillance criteria (10, 11) requires five bands (IgG) for a test to be considered positive, the WB criteria in Europe acknowledges that the immune response is lower in borrelia species other than B. burgdorferi ss, and the requirement for a test to be considered positive is for one or two - depending on what species being tested for - bands only (12).

Regardless of whether using American or European Western Blot interpretation, the length of time of IgG responses is not 'set' to the "first few weeks". A few excerpts on this:

Craft, Fischer, Shimamoto and Steere (1986), whose article is referenced in the introduction in the Dressler et al paper that the ICPMR fact sheet refers to: "In 12 patients with early disease alone, both the IgM and IgG responses were restricted primarily to a 41-kD antigen. This limited response disappeared within several months...The IgG response in these patients appeared in a characteristic sequential pattern over months to years.." (13;pg 934).

Strle et al., (1996) "Our work also highlighted the continuing problems associated with use of serological methods for patients with early disease. Fewer than 50% of cases demonstrated seropositivity at any time within the first 2months" (14; pg 64).

Aguero-Rosenfeld et al., (1996) report on the serological results from Culture-Confirmed cases of Lyme: "Although 89% of the patients developed IgG antibodies as determined at a follow-up examination, only 22% were positive by the IgG IB criteria of the Centers for Disease Control and Prevention-Association of State and Territorial Public Health Laboratory Directors. (15; pg 1).

The question that also needs to be answered is, what exactly does the Centre for Infectious Diseases and Microbiology at Westmead define as, or refer to, when they say "true" Lyme disease. At the time of their study it was known that there were various species of borrelia responsible for Lyme, for example, in 1994, Steere (who first 'recognised' Lyme disease) described "Lyme disease or Lyme borreliosis", as the result of an infection from B.burgdorferi ss, B azfelli or B garinii (16). It has also been long known that there are numerous other borrelia species in the burgdorferi sensu lato complex that may cause Lyme disease (eg: 17-22), which the testing advice provided by Westmead does not appear to acknowledge, or take into account in their testing procedures.

In an interview with Doctor Jeremy McAnulty, the Director of Health Protection with the New South Wales Department of Health in July 2010, Bronwyn Herbert asked whether the testing methods for Lyme disease in Australia were adequate. The reply from Jeremy McAnulty *"Look they do seem to be and again we need to put in context who needs to be tested and when and the doctor's decision and advice about that. But there is a specialist laboratory at Westmead that's very expert in the range of tests that need to be done and can be done and they of course keep in contact with the experts around the world"* (23).

To Dr McAnulty I would say: If Westmeads specialist laboratory is in contact with experts around the world, why does their advice with regards to Lyme disease and testing procedures ignore over half the literature in the world ; Why is the Australian laboratories first line of testing looking for burgdorferi ss species, especially when outside of America, B. Burgdorferi ss is not the most commonly found species of borrelia. For example, in a 2005 meta analysis of studies in Europe, it was noted that the afzelii, garinii, and valaisiana were more common then sensu stricto (21). In Asia, the presence of B. burgorferi ss was not found (and then only in animals) until 2011 (22); With so many species of borrelia being found worldwide, why are Westmead officials adamant a species of borrelia underlying Lyme cannot possibly be in Australia, denying any further government research for the last twenty years.

Reservoir Host Studies

DME Website: *"A small number (17) of native vertebrate animals were sampled by ear punch biopsy for culture and PCR investigation but there was no evidence of borreliae".*

Counterpoint: The reservoir host studies limitations speak for themselves with only **seventeen** (17) animals tested. Ear punch biopsy for culture and PCR investigation of only 17 vertebrate animals is also very limited and restrictive when considering that different species of borrelia have different reservoir host preferences. For example, the B. burgdorferi ss species appears to have preferential preference for rodent hosts, whilst B. valaisiana has not been found in rodents, rather the preferred reservoir host is birds (1-3). Differences in borrelia species can also extend to where in the host the bacteria can be detected, for example, in one study it was noted that "B. garinii infections were not detected in the skin of the rodents, but were confined to internal organs, particularly the brain" (3). Ear punch biopsy of **17** animals cannot be used to ascertain the presence, or lack thereof, of a borrelia infection/species in Australian animals.

DME Website: *"None of the mammal species identified as reservoir hosts in the northern hemisphere are present in Australia".*

Counterpoint: This statement is simply not correct. Mammals that have been identified as reservoir hosts in the northern hemisphere include, rats, mice, hares, rabbits, foxes, cats, dogs and many other animals.

The primary reservoir host in America is the white-footed mouse (*Peromyscus leucopus*). It is a mammal, belonging to the rodentia species of the *Muridae* family. Whilst we do not have any white-footed mouse in Australia, "22 % of Australian mammal species are all in the rat and mouse family, *Muridae*" (4).

This includes the Australian Long-haired Rat (*Rattus villosissimus: Muridae family*). In 1962 these rats were the subject of a study in north-west Queensland in which a new species of borrelia was identified and subsequently called borrelia Queenslandica (5). As well as this study, a 1959 CSIRO study of Australian animals reported that borrelia was found in the blood of cattle, kangaroos, bandicoots and rodents (6).

The DME website brushes over these two studies**,** *"There are reports of spirochaetes in Australian native animals, and a local mammal could be a reservoir host for an indigenous spirochaete…" ,* With no indication of these studies in the further readings/reference section they do not give the implications of this research enough respect or consideration. Which is, if there is borrelia in the animals in the Australian environment, then there must be a capable vector maintaining/spreading the bacteria.

The following identifies just a few of the mammal species that are present in Australia, and have been found to be reservoir hosts for borrelia in the northern hemisphere:

Black Rat (*Rattus rattus*): This species of rats was introduced into Australia and is "spread throughout much of coastal Australia and is most commonly seen in urban environments" (7). Black rats have been shown to be competent reservoir hosts in Bulgaria and Germany (8,9).

Brown Rat (*Rattus norvegicus*) : The brown rat, was introduced into Australia around the same time as black rats (and mice). Whilst not as abundant as the black rats, "the Norway or brown rat is found in or near human habitation, especially in coastal towns (10)". Brown rats are also competent reservoir hosts of borrelia (11,12).

House Mouse (*Mus musculus* : sub species *mus m musculus, mus m domesticus*) : House mouse have a worldwide distribution. Early zooarchaeological evidence (13) suggests they were introduced into Australia late in the 18th century, around the time of the first European settlers. They are currently spread throughout Australia (14). *Mus musculus* was shown to be a competent reservoir/maintenance host of borrelia within the environment in Bulgaria (8). *Mus musculus* are also frequently used in laboratory experiments due to their susceptibility to the borrelia bacteria (15).

The Brown/ European Hare (*Lepus europaeus*) : This hare species have been shown to be a competent reservoir host in Sweden (16,17) "European Hares were successfully introduced to mainland Australia in the 1860s. They were first introduced to Westernport Bay, Victoria, in 1862. Hares transported in the 1930s became established in pockets at Townsville, Ayr and Mackay, Queensland." "They are currently established in a crescent extending from near Ceduna in South Australia, through Victoria and most of New South Wales to as far north as Cairns in north-eastern Queensland" (18).

As mentioned and referenced extensively in this counter-arguments complimentary report, 'Lyme Disease / Borreliosis: An overview of Lyme and direction for further research required in Australia' (19), there are numerous other mammal species in Australia that are reservoir hosts for the borrelia bacteria. These include domestic animals such as dogs and cats, as well as other wild and farm animals such as foxes, sheep, deer, horses and cattle.

The Department of Medical Entomology's information also overlooks the fact that other animal classes, such as reptiles and birds, can be reservoir hosts for borrelia species. Whilst some borrelia species, such as B. burgdorferi ss cannot survive in reptile blood, the preferred host for the borrelia species Lusitaniae (which has been associated with Lyme disease/borreliosis in humans in Asia and Europe), is lizards (20). Bird species that are reservoir hosts of borrelia, and have been introduced into Australia, include; song thrushes and common black birds, wild turkeys, pheasants, quails and mallard ducks (21-29).

Borrelia has been found in the blood of Australian animals (5, 6). The examination of seventeen animals to rule out the existence of borrelia bacteria responsible for Lyme disease cannot seriously be considered 'an encompassing reservoir host study'. As it couldn't be put it any clearer, this section ends with two quotes on how it is necessary to examine reservoir hosts in order to ascertain the presence of pathogens in the environment:

- "Since the abundance of reservoir hosts in a habitat is crucial to the establishment of infected tick populations it is important to identify both the presence of particular reservoir hosts in a habitat and also their role in generating infected ticks" (30: pg 256)

- "To predict and prevent human risk of exposure to vector-borne diseases, it is vital to identify the reservoir hosts of the pathogens" (31: pg 535).

Vector Studies

DME Website: *"There are reports of spirochaetes in Australian native animals, and a local mammal could be a reservoir host for an indigenous spirochaete that occasionally infects humans through a tick vector and produces a clinical syndrome similar to LD; however, no spirochaete was detected in the 12,000 ticks or animals processed".*

As discussed in the reservoir host section, the total number of 'animals processed' (processed by, capturing, and taking an 'ear punch biopsy') was seventeen. Ear punch biopsy of 17 vertebrate animals cannot be used to ascertain the presence, or lack thereof, of the borrelia species responsible for Lyme disease in Australia.

The 12,000 ticks processed, is the primary research performed in 1994 by Russel et al., of which the denial of the existence of Lyme in Australia is still based on today. This research is examined in detail below.

Lyme disease: search for a causative agent in ticks in south-eastern Australia. Epidemiology and Infection. Russell RC, Doggett SL, Munro R, Ellis J, Avery D, Hunt C, Dickeson D. 1994. Epidemiology and Infection 112: 375-384.

Abstract: *"Attempts were made to identify the causative organism of Lyme disease in Australia from possible tick vectors. Ticks were collected in coastal areas of New South Wales, Australia, from localities associated with putative human infections. The ticks were dissected; a portion of the gut contents was examined for spirochaetes by microscopy, the remaining portion inoculated into culture media. The detection of spirochaetes in culture was performed using microscopy, and immunochemical and molecular (PCR) techniques. Additionally, whole ticks were tested with PCR for spirochaetes. From 1990 to 1992, approximately 12,000 ticks were processed for spirochaetes. No evidence of Borrelia burgdorferi or any other spirochaete was recovered from or detected in likely tick vectors. Some spirochaete-like objects detected in the cultures were shown to be artifacts, probably aggregates of bacterial flagellae. There is no definitive evidence for the existence in Australia of B. burgdorferi the causative agent of true Lyme disease, or for any other tick-borne spirochaete that may be responsible for a local syndrome being reported as Lyme disease".*

*Please note: The spelling of 'spirochaete' in the abstract, and when referencing Russel et al below is as it was printed in the article. It is acknowledged that throughout the world, it is typically spelt 'spirochete'.

Russel et al Pge 377: *"The study area comprised the coastal strip of NSW, from the Queensland border in the north to the Victorian border in the south".*

Pge 378: *"From January 1990 and December 1992, > 20,000 ticks were collected"*

Pge 375: *"From 1990 to 1992, approximately 12,000 ticks were processed for spirochaetes".*

Counterpoints**:** There was over 20,000 ticks collected, and approximately 12,000 of these were examined in the study. There is no explanation as to what happened to the other 8,000 or more ticks that were collected, whether they died in storage, or how it was determined which ticks should be utilised.

Of the 12,000 ticks processed, 6,235 of them were questing (looking for a blood meal) larvae. From the numerous journal articles read in relation to this topic, it is typically only the nymph and adult ticks that are utilised in studies examining borrelia rates in the environment as the transoveral infection rate of larvae is less than 1%. "In general, less than 1% of host-seeking larvae are infected, compared with between 10% and 30% of the nymphs and between 15% and 40% of adults" (1: pg13). Indeed, Russell et al., do note in their introduction, "*Transmission to humans will only occur from ticks that feed first on infected reservoir hosts and then on humans"* (pg 376).

From the 12,000 ticks tested, this leaves approximately 5,770 ticks that would have definitely had a chance to acquire borrelia infection/spirochetes via a host/blood meal. Whilst nearly 6,000 ticks 'may be' considered a relatively large number, the figure "12,000" is always brought up in relation to this study, and the denial of Lyme in Australia. On saying 6,000 'may be' considered a relatively large number, what needs to be taken into account is that infection rates of ticks from the same country can vary dependant on, species of tick, stage of tick (ie: larval, nymph, adult), region and environmental area (ie: pastures, mountains, forests, coastal) they are collected from. Differences in infection rates can be tremendous, varying from 0 to <90% (1-6). Therefore, when you take into consideration that the ticks examined in the Russel et al., study were collected from a 2,000km section of Australia's 35,000+ km coastline (one region/state of Australia and one environmental location), and that this tick collection examination/study is the primary basis of denial of Lyme, not just in the State of New South Wales they were collected from, but the whole of Australia, then 6,000 ticks is not a large number by any standard.

PCR testing

Russell et al Pge 378-9: *"1038 ticks tested using PCR, no amplification products which would suggest the presence of borrelia were detected".*

Russel and others note their own limitations of the PCR testing of ticks in this study: *"It is possible that the monoclonal antibodies and PCR primers used in this study may not have been appropriate to identify indigenous Australian spirochaetes. However, the tick gut contents were also negative by culturing and dark field microscopy" (pg 381).*

The problems relating to the techniques used and conclusions drawn from the culturing and dark field microscopy examinations is the subject of the next section.

Spirochaete detection and isolation: Darkfield Microscopy and Culture.

Russel et al Pge 378-379 : *"Between January 1990 and December 1992, > 20,000 ticks were collected. Approximately 11,000, including all stages of four species, Ixodes holocyclus, I. tasmani, Haemaphysalis bancrofti and H. longicornis were dissected for spirochaete isolation. With the additional 1,038 ticks tested using PCR, no amplification products which would suggest the presence of borrelia were detected. "*

Russell et al Pge 378: *"No spirochaetes were detected by dark field microscopy of the gut contents of the unfed ticks…"*

Counterpoints**:** The methods section of Russel et al, explains that the ticks were stored live until processed (pg 377), though doesn't explain the length of time the ticks were actually stored for (ie: were they all stored from 1990, until processed in 1992?), or give any understanding (ie; had 8,000 ticks died while being stored?) as to why only 12,000 of the 20,000+ ticks collected were subsequently processed in the study.

The time and method of tick storage is very relevant as to whether or not spirochetes may or may not be able to be observed in the gut contents of ticks. In studies that examine poor environmental conditions, such as starvation, it has been observed that motile spirochetes convert into non-motile cyst forms; until such time that their environment is more conducive to their requirements (7-9). The lack of detection of spirochetes in the gut contents of ticks that had been stored live, rather than immediately frozen or stored in ethanol to preserve their contents (10) for an indefinite period of time, cannot rule out the presence of cystic forms of borrelia. Other detection methods, such as indirect fluorescent antibody (IFA), have been shown to be more sensitive in detecting the presence of spirochetes (11).

Culture: Spirochaete like Objects (SLO's)

Russell et al Pge 379: *Spirochaete-like objects (SLO's)… were revealed by dark field microscopy in 92 cultures.." "Purified SLO's were obtained with 0.45um filters, but it was not possible to subculture them in the absence of bacterial contaminants…"*

Background of culture medium and counterpoints: BSKII medium is a specialized growth medium that may be used for culturing spirochetes, though as the quality of the medium is variable due to variations of medium components such as bovine serum albumin, rabbit serum and yeast extract and each batch mixed requires special care in preparation, filtering, and screening for its ability to support the growth of borrelia (12). Considering this variability, specialist laboratories examine each batch of medium prepared to assess its viability to maintain spirochetal growth. There is no indication that the batch of medium prepared by Russel et al., (a laboratory with no prior experience in culturing borrelia spirochetes) was tested for its ability to maintain viable spirochetal growth prior to use in this study.

The use of 0.45um filter paper is ideal to obtain purified spirochetes as unlike most other bacteria, leptospires and treponemes are able to migrate through filter papers (13). Whilst Russell et al., concluded that what they obtained from the cultures were SLO's, the fact that the bacteria that was isolated was able to migrate through the filter paper is highly suggestive of the fact that they were indeed spirochetes.

As purified SLO's were obtained via filtration methods, it is feasible to assume that the bacterial contaminants in the subculture were more than likely due to the BSK II medium, rather than contaminants from the ticks bloodmeal.

Whilst the use of 0.45um filter paper has been found to be one way of culturing purified spirochetes, another method known to rid the culture of contaminants is the addition of antibiotics such as Rifampin, Phosphomycin, Amphotercin B (12,14) that borrelia are resistant to. It has also been found that BSK medium containing Co-trimoxazole (15) or Rifampin, is "more efficient for spirochete isolation than unsupplemented BSK medium" (11). It is not possible to determine from the methods section of the Russel et al., paper whether the use of antibiotics such as those previously mentioned was employed. The methods section does however mention the use Skirrows supplement, which is an antibiotic supplement recommended for selective isolation of campylobacter species and contains three antibiotics, Vancomycin, Polymixin and Trimethoprim (16), Spirochetes are susceptible (killed) to both Vancomycin (17,18) and Trimethoprim (19), rendering the choice of Skirrows supplement a less than ideal additive, considering the aim was to culture/grow (rather than kill) spirochetes.

Molecular identification & description of culture products

Russel et al Pge 379 : *"While a few positive results were obtained by IFAT using polyclonal antibodies, the results were both variable and inconsistent for the 18 SLO's tested."*

Counterpoint: Variability of positive IFAT results should be cause for further investigation: The quality of medium has been found to, alter gene expression patterns (20), effect the morphology (length and number of coils) and motility of spirochetes as well as alter the results of IFAT tests (21).

Russel et al Pge 380: *"PCR … successfully amplified a 950bp fragment in 92 of 92 SLO cultures, however the fragments amplified produced characteristic enzyme digestive products of a Bacillus sp. and not a Borrelia sp."*

Russel et al Pge 380: *".. the SLO's appeared straight, rigid and uniformly coiled, varied in length (10-300um)* and had 2-40 complete coils; all appeared to be non-motile."*

*It is assumed that 300um is a typographical error and should read *30um. This would be in line with the picture (pg 380) showing a 50um bar for comparison.

Counterpoints:The 950bp fragments amplified by 16S rDNA cannot be interpreted as being able to rule out Borrelia species as enzyme digestive products with a 950bp have also been identified for B. burgdorferi ss (22). "…heterogeneities between 16S rRNA genes seems to be a common phenomenon and, that for species identification, 16S rDNA analysis has to be interpreted with care" (23: Pg 2246).

Bacterial species cannot be defined by DNA similarities alone, (24), and what is more descriptive here is the appearance of the SLO's. Bacillus species are rod like and 5-10um in length. Borrelia species are spiral shaped and 10-30um. The pictures provided in the journal article (pg 380) and the description of the SLO's are more representative of the appearance of the Borrelia species rather than bacillus.

Russel et al Pge 381 : *"Electron micrographs showed that these SLO's had no distinct cellular structure but were composed of fibre-like subunits, and were not spirochaetes."*

Counterpoint: Spirochetes do not have a distinct cellular structure and are composed of axial filaments which have one or more fibrials. The three brief quotations below expand on this:

"The outer sheaths of S. plicatilis, all Borrelia species, and T. phagedenis strains so far examined are characterized by a lack of structural detail" (25 :pg 118).

"Ultrastructural examination of spirochetes has established their procaryotic nature and the one ultrastructural feature-the axial fibril-that sets them apart from other prokaryotes" (25: pg 152).

"Spirochetes consist of three main structures: aprotoplasmic cylinder, an axial filament (consisting of one or more fibrils), and an outer envelope..." (26 pg: 1087).

Whilst the conclusion was drawn by Russel and others that the 'objects' cultured from some of the ticks were spirochete-like objects (SLO's), the following section is based on the assumption that they were in fact more than likely spirochetes and examines the tick species they were cultured from briefly below.

Tick Species Spirochete-Like Objects (SLO's) Cultured from

Russel et al Pg 379: *"The tick species yielding these SLO's were I.holocyclus, H.bancrofti, H.longicornis and Amblyomma morelia."*

Paralysis Tick (*Ixodes holocyclus*)

I. holocyclus is more commonly known as the paralysis tick as bites from this tick can cause paralysis in animals and humans. It is found in Queensland, New South Wales, Victoria and Tasmania. The *holocyclus* range of hosts is extremely wide and includes both native and introduced animals, including birds and reptiles. The mammalian hosts range from rodents, to animals in the wild such as kangaroos, koalas, bandicoots, to domesticated and farm animals such as dogs, cats, cattle, horses, pigs and sheep. Humans may occasionally become accidental hosts (27-29).

The *I. holocyclus* is the tick "presumed" most likely to be the vector for borrelia in Australia, and as such is the only tick species to have been examined in relation to its capability of transmitting borrelia. In 1991 Piesman and Stone (30) conducted a study that examined the ability of *I. holocyclus* to acquire, maintain and transfer the borrelia burgdorferi ss species. It was found that while larval *I. holocyclus* could ingest the spirochetes, the infectivity was not maintained once the tick had "moulted" to its next cycle, the nymphal stage. The conclusion was, "These experiments should be repeated with Australian strains of spirochetes" (30). However, in the 21 years since; no further studies have been performed. Whilst there has been no Australian spirochete identified (due to no ongoing research), studies to ascertain the ability of transmission with European species of borrelia, would have been possible to conduct.

Taking into consideration the knowledge that certain tick species may only transmit species of borrelia (eg 31) common to their country of origin, it is not appropriate to rely on one study (30) that examined an indigenous Australian tick species ability to transmit a borrelia species most common to America. As Piesman and Stone (30) concluded, additional research should be performed. Along with the fact that SLO's were cultured from this species of tick, further information adding to the argument for additional research on this species ability as a vector is that borrelia-like spirochetes were also cultured from *I.holocyclus* ticks collected from the Manning River district of NSW in research conducted by Wills and Barry in 1991 (32). Additionally, very many of the animal hosts of *I. holocyclus* are capable reservoir hosts for borrelia, for example mice, rats, cats, dogs, cows, horses and birds, adding even more reason to conduct further research with regards to what pathogens the ticks may carry, and the ability of this tick to carry/transmit borrelia species that are more common in Europe and Asia.

Wallaby Tick *(Haemaphysalis bancrofti)*

H. bancrofti is informally known as the wallaby tick as their principle hosts are wallabies. This species has also been collected from kangaroos, bandicoots and other mammals and livestock including cattle and sheep. *H. bancrofti* is found in Queensland, New South Wales and on Kangaroo Island, off South Australia (33, 34).

As *H. bancrofti* is only found in Australia and New Guinea, countries that have not typically been associated with borrelia, there does not appear to have been any research to determine its capabilities as a vector. What is known is that *H. bancrofti* is a vector of Theileria (piroplasm) (35-37), and this tick species is thought to be involved in the transmission of severe outbreaks of the disease which resulted in the death of over 800 in cattle on NSW farms in 2008 (37, 38). In research across the world there has been found to be an association between ticks that transmit piroplasms and borrelia (eg:39, 40). Considering this association, as well as the fact that SLO's were cultured from this tick species, it would seem apparent that further research on this tick species vector capabilities would be appropriate. This is especially so when you also add in the information that in 1991 Wills and Barry cultured borrelia-like spirochetes from *Haemaphylsalis* species of ticks, and many of the animal hosts of *H. bancrofti* are capable reservoir hosts for borrelia, including cattle, kangaroos, bandicoots and rodents in which a 1962 study (41) reported borrelia in the blood of these Australian animals.

Scrub/Bush Tick *(Haemaphysalis longicornis)*

The *H. longicornis* is more commonly known as the scrub or bush tick. It was introduced into Australia on cattle from Northern Japan and was first recognised in 1901 in north eastern New South Wales. This tick species is now established along coastal areas in Queensland, New South Wales, and through north eastern Victoria (esp Murray Valley) and Western Australia (42-44).

The hosts of the *H. longicornis* tick (45) include numerous animals that have been found to be reservoir hosts for borrelia and have been introduced or imported into Australia from countries that are endemic for Lyme disease (45,46). These animals include the smaller reservoir hosts listed previously in this counter-argument, ie: mice, rats and hares, as well as domestic animals such as dogs and cats (47-52) and medium to large animals such as foxes (53,54), cattle, horses (55-62), sheep and deer (63-65) that have been introduced into Australia and have varying levels of reservoir competence for borrelia.

Examination of *H. longicornis* as a possible vector of Lyme in Australia is warranted for numerous reasons. This includes knowledge about its role in the borrelia cycle in China (66-70). Combine this information with the fact that SLO's were cultured from this tick species, and the knowledge that this tick species was the one infesting cattle in cases of suspected Lyme disease in cattle at Camden NSW in 1989, in which positive IFAT serology for Borrelia burgdorferi was reported: "the herd from which these cases came was heavily infested with the Bush tick, *Haemophysalis longicornis*, at the times of presentation..." (71: pg 298), then it would seem apparent that research on this tick species role in the borrelia cycle in Australia is long overdue.

NB: Due to its known role of in China, the *H. longicornis* tick is covered in more depth in this counter-arguments complimentary report, Lyme Disease / Borreliosis: An overview of Lyme and direction for further research required in Australia.

Snake Tick *(Amblyomma morelia)*

Amblyomma morelia is more commonly known as the snake tick. Whilst its preferential host is snakes, they are also found on various reptiles such as lizards and monitors (72). In Australia it is found in Queensland, New South Wales, Victoria, and the Northern Territory (73).

Whilst snakes and lizards were initially thought to be incompetent reservoir hosts of borrelia, one species, *B. lusitaniae* has been associated with lizards in several studies (74,75). Further examination of *A. morelia* is warranted, especially considering that even though the number of this tick species in this study was limited to 14 (4 nymphs and 10 adults), SLO's were cultured. Also of interest would be the examination of smaller rodents such as mice and rats, which due to their close natural environmental coexistence, the larvae may have initially fed on.

As well as the above mentioned ticks, there are also numerous other species from the *Ixodidae* genera in Australia. This includes the Seabird tick (*Ixodes uriae)* and a Bird tick (*Ixodes auritulus)* that are known vectors of borrelia (76-80). The *I. uriae tick* is found worldwide, including Australia and its offshore islands (81). The role that migrating seabirds and the *I. uriae* tick play in spreading borrelia has been known of since the early 1990's (82). In a 1993 study by Olsen et al., Borrelia DNA was found in *I. Uriae* ticks from Crozet and Campbell Islands, off the New Zealand coast suggesting Lyme is indeed in the Southern hemisphere (83). The *I. auritulus* is a native tick of Tasmania (84-85). The first reports of borrelia being found in this tick species were from Canada in 2005 (80). *I. auritulus* attach to bird hosts such as the European blackbird and song thrushes. Both of these bird species have been introduced into Australia (86-88), and both are known reservoir hosts of borrelia (89-92). These two tick species as well as others from the *Ixodidae* genera, are covered in more detail in, 'Lyme Disease / Borreliosis: An overview of Lyme and direction for further research required in Australia'.

Conclusion

In concluding this examination of the various components - clinical and serological studies, reservoir host, and vector – of the multidisciplinary investigations performed at the Department of Medical Entomology, Westmead Hospital, hopefully it has been made apparent that the NSW Health Department has relied for far too long on the 'not a lot of evidence' rhetoric about the absence of Lyme in Australia.

The major issues of this counterpoint argument can be summarised as follows:

(1) Clinical and serological studies: There have been over a thousand clinically suspected cases each year since the 80's & 90's in Australia, along with the hundreds of diagnosed cases reported in the Lyme Disease Association of Australia's survey. Despite the fact that Lyme is primarily a clinical diagnosis, supported by blood tests, the denial of Lyme disease continues. This denial is based on interpretation of tests that are not recommended outside of the United States of America, and are vastly different to European recommendations. The continued use of outdated and incorrect serology techniques to continue to deny the existence of Lyme can only be described as highly inappropriate and negligent.

(2) Reservoir host studies: As noted, the examination of seventeen (17) animals cannot be considered anything but limited. The denial of the existence of mammal reservoir hosts of borrelia in Australia is also incorrect, with mammals such as mice, rats, and hares, which have been shown in the Northern hemisphere to be capable reservoir hosts, being present in Australia. Larger mammals that have been imported into Australia, as well as other animal species, such as snakes and birds are also known reservoir hosts of borrelia.

(3) Vector: Suffice to say, ticks collected from a 2,000 kilometre section of Australia's 35,000+ km coastline, over 20years ago (Whilst the journal article date was 1994 – the ticks were collected between the period of January 1990-December 1992), really should not be the basis for the denial of Lyme in Australia. A study with many inconsistencies, including variable IFAT results and the culturing of "spirochete-like objects", should not have been used to deny the existence of Lyme in Australia, not twenty years ago, and certainly not now. As noted there are many species of ticks, including one in which Russel et al., cultured 'spirochete like objects' from that have been shown to be a vector of borrelia in the Northern Hemisphere. Further research, including the examination of numerous tick species from right around Australia need to be conducted in order to ascertain what pathogens they carry.

Despite over a half a dozen other publications around the 1980's and 1990's that were highly suggestive of the fact that Lyme disease is in Australia, the 1994 study by the researchers at the Department of Medical Entomology (DME), Westmead is the only study that is acknowledged by the Australian Government. As this counter-argument highlights, numerous erroneous conclusions seem to have been drawn and rather than concede there was some inconsistencies and utilise the study as the basis for further research, its authors, the DME at Westmead, and Government health departments have continued to defer to this study to put a stranglehold on any further investigations and government research with regards to Lyme disease in Australia for the last twenty years.

The lack of advice - or worse still, the vehement insistence that Lyme is not in Australia - to both clinicians, and patients with regards to this disease by the health department of Australia means that rather than having access to all information that would allow recognition and short term treatment in the initial stages of Lyme disease, people are left undiagnosed for many years which then leads to disseminated long term infections that are much harder to treat. Up to date research needs to be urgently undertaken with regards to the pathogen underlying Lyme in Australia.

References

A Counter-Argument of the Australian Governments' Denial of Lyme

(1) Lyme Disease / Borreliosis: An overview of Lyme and direction for further research required in Australia. Karen Smith, May 2013. ISBN: 978-0-646-90428-3. http://www.lymeaustralia.com/k-smith-lara-research.html

(2) Lyme disease: Australian patient experience in 2012. Lyme Disease Association of Australia. November 2012. ISBN: 978-0-646-59269-5. http://www.lymedisease.org.au/wp-content/uploads/2012/11/ldaa-lyme-disease-_australian-patient-experience-in-2012-22nov12.pdf

(3) Russell RC, Doggett SL, Munro R, Ellis J, Avery D, Hunt C, Dickeson D. (1994) Lyme disease: search for a causative agent in ticks in south-eastern Australia. Epidemiology and Infection 112:375-384. http://www.ncbi.nlm.nih.gov/pubmed/8150011

(4) Mackerras MJ (1959) The haematozoa of Australian mammals. Australian Journal of Zoology, 7(2)105 – 135. http://www.publish.csiro.au/paper/ZO9590105.htm

(5) Carly JG and Pope JH (1962) A new species of Borrelia (B. queenslandica) from Rattus villosissimus in Queensland. Aust J Exp Biol Med Sci; 40:255-61. http://www.ncbi.nlm.nih.gov/pubmed/13876596

(6) Stewart A, Glass J, Patel A, Watt G, Cipps A and Clancy R (1982) Lyme arthritis in the Hunter Valley. Med J Aust; 1(3):139. http://www.ncbi.nlm.nih.gov/pubmed/7132855

(7) McCrossin I (1986) Lyme disease on the NSW South Coast (letter). Med J Aust; 144(13):724-5. http://www.ncbi.nlm.nih.gov/pubmed/3724608

(8) Lawrence RH, Bradbury R, Cullen JS (1986) Lyme disease on the NSW Central Coast (letter). Med J Aust; 145(7):364. http://www.ncbi.nlm.nih.gov/pubmed/3762468

(9) Rothwell JT, Christie BM, Williams C and Walker KH (1989) Suspected Lyme disease in a cow. Aust Vet J; 66(9):296-8. http://www.ncbi.nlm.nih.gov/pubmed/2684126

(10) Wills MC and Barry RD (1991) Detecting the cause of Lyme Disease in Australia. Med J Aust; 155(4):275. http://www.ncbi.nlm.nih.gov/pubmed/1875848

(11) Hudson BJ, Barry RD, Shafren DR, Wills MC, Caves SF and Lennox VA (1994). Does Lyme borreliosis exist in Australia? J Spirochetal and Tick-Borne Dis; 1(2): 46-51. http://www.lyme.org/journal/journal/vol1no2/v1n2-Exist.pdf

(12) McGoll GJ, Frauman AG, Dowling JP and Variagos GA (1994) A report of Lyme disease in Victoria. Aust NZJ Med; 24(3)324-325. Published online March 2008: http://onlinelibrary.wiley.com/doi/10.1111/j.1445-5994.1994.tb02189.x/abstract

(13) Hudson BJ, Stewart M, Lennox VA, Fukunaga M, Yabuki M, Macorison H, Kitchener-Smith J (1998) Culture-positive Lyme borreliosis. Med J Aust; 16(10):500-2. http://www.ncbi.nlm.nih.gov/pubmed/9631675

(14) Cestnick L (1998) Lyme disease in Australia. Aust NZJ Public Health; 22(5):524. http://www.ncbi.nlm.nih.gov/pubmed/9744201

(15) Lyme Disease Expert Panel - 2324 Parliament of New South Wales. Question asked by Dr Kaye to the Minister for Police and Emergency Services, Minister for the Hunter, and Vice-President of the Executive Council representing the Minister for Health, and Minister for Medical Research. http://www.parliament.nsw.gov.au/prod/lc/qalc.nsf/c63f637ee30ce3beca2578c300122a54/0abdb574b102f8d4ca257a7d00264142?OpenDocument

(16) Lyme Disease. The Department of Medical Entomology, Westmead Hospital NSW. http://medent.usyd.edu.au/fact/lyme%20disease.htm

Clinical & Serological Studies

(1) CDC Morbidity and Mortality Weekly Report (1990) Case definitions for public Health Surveillance. (39) No. RR-13:20-22. Accessed July 2011, http://www.cdc.gov/mmwr/PDF/rr/rr3913.pdf

(2) CDC 2011 Lyme Disease (Borrelia Burgdorferi) Case Definition. Accessed July 2011, http://www.cdc.gov/osels/ph_surveillance/nndss/casedef/lyme_disease_current.htm

(3) Testimony by Paul Mead, Medical Epidemiologist with the CDC, given to the Connecticut Department of Public Health and the Connecticut Attorney General's Office at a hearing regarding CDC's Lyme Disease Prevention and Control Activities in 2004 notes. http://www.hhs.gov/asl/testify/t040129.html

(4) Dressler F, Ackermann R and Steere AC (1994). Antibody Responses to the Three Genomic Groups of Borrelia burgdorferi in European Lyme Borreliosis. The Journal of Infectious Diseases;169:313-8. http://www.ncbi.nlm.nih.gov/pubmed/8106763

(5) Piesman J and Gern L (2004) Lyme borreliosis in Europe and North America. Parasitology, 129, S191-S220. DOI: 10.1017/S0031182003004694 http://www.ncbi.nlm.nih.gov/pubmed/15938512 http://www.cbpv.com.br/artigos/CBPV_artigo_012.pdf

(6) Lyme Disease - Testing Advice for NSW clinicians : NSW Health. Accessed April, 2013 http://www0.health.nsw.gov.au/resources/publichealth/infectious/diseases/pdf/lyme_disease_testing_advice.pdf

(7) Lyme Disease Fact Sheet. The Institute of Clinical Pathology and Medical Research (ICPMR), Centre for Infectious Diseases and Microbiology Laboratory Services , Westmead. Sydney West Area Health Service. (Document no longer seems to be accessible on line)

(8) Marangoni A, Sparacino M, Cavrini F, Storni E, Mondardin V, Sambri V and Cevenini R (2005) Comparative evaluation of three different ELISA methods for the diagnosis of early culture-confirmed Lyme disease in Italy. J Med Microbiol; 54 (Pt 4): 361-7. http://www.ncbi.nlm.nih.gov/pubmed/15770021

(9) Ang CW, Notermans DW, Hommes M, Simoons-Smit AM & Herremans (2011) Large differences between test strategies for the detection of anti-Borrelia antibodies are revealed by comparing eight ELISAs and five immunoblots. Eur J Clin Microbiol Infect Dis. 30 (8) 1027-32. Epub Jan 27 2011 http://www.ncbi.nlm.nih.gov/pubmed/21271270#

(10) Dressler F, Whelan JA, Reinhart BN, Steere AC (1993) Western blotting in the serodiagnosis of Lyme disease. J Infect Dis,167, 392-400. http://www.ncbi.nlm.nih.gov/pubmed/8380611

(11) Engstrom SM, Shoop E, Johnson RC (1995) Immunoblot interpretation criteria for serodiagnosis of early Lyme disease. J Clin Microbiol, 33, 419-22. http://www.ncbi.nlm.nih.gov/pubmed/7714202

(12) Hauser U, Lehnert G, Lobentanzer R and Wilske B (1997) Interpretation criteria for standardized Western blots for three European species of Borrelia burgdorferi sensu lato. J Clin Microbiol; 35(6):1433-44. http://www.ncbi.nlm.nih.gov/pubmed/9163458

(13) Craft JE, Fischer DK, Shimamoto GT and Steere, AC (1986) Antigens of Borrelia burgdorferi Recognized during Lyme Disease. Appearance of a new immunoglobulin M response and expansion of the immunoglobulin G response late in the illness. J Clin Invest. 1986 October; 78(4): 934–939. http://www.ncbi.nlm.nih.gov/pmc/articles/PMC423723/

(14) Strle F, Nelson JA, Ruzic-Sabljic E, Cimperman J, Maraspin V, Lotric-Furlan S, Cheng Y, Picken MM, Trenholme GM and Picken RN (1996) European Lyme Borreliosis: 231 Culture-Confirmed Cases Involving Patients with Erythema Migrans. Clin Infect Dis; 23(1):61-65. http://cid.oxfordjournals.org/content/23/1/61.short

(15) Aguero-Rosenfeld ME, Nowakowski J, Bittker S, Cooper D, Nadelman RB and Wormser GP (1996) Evolution of the serologic response to Borrelia burgdorferi in treated patients with culture-confirmed erythema migrans. J Clin Microbiol; 34 (1): 1-9. http://www.ncbi.nlm.nih.gov/pubmed/8748261

(16) Steere AC (1994) Lyme disease: A growing threat to urban populations. Proc Natl Acad Sci; 91(7): 2378-*2383.* http://www.ncbi.nlm.nih.gov/pmc/articles/PMC43375/

(17) Collares-Pereira M, Couceiro S, Franca I, Kurtenback K, Schafer SM, Vitorino L, Goncalves L, Bapista S, Vieira ML and Cunha C (2004) First isolation of Borrelia lusitaniae from a human patient. J Clin Microbiol;42(3):1316-8. http://www.ncbi.nlm.nih.gov/pubmed/15004107

(18) Margos G, Vollmer SA, Cornet M, Garnier M, Fingerle V, Wilske B, Bormane A, Vitorino L, Collares-Pereira M, Drancourt M and Kurtenbach K (2009) A new Borrelia species defined by multilocus sequence analysis of housekeeping genes. Appl Environ Microbiol;75(16):5410-6. Epub 2009 Jun 19. http://www.ncbi.nlm.nih.gov/pubmed/19542332

(19) Stanek G and Reiter M (2011) The expanding Lyme Borrelia complex – Clinical significance of genomic species? Clin Microbiol Infect;17(4):487-93 http://www.ncbi.nlm.nih.gov/pubmed/21414082

(20) Kampen H, Rotzel DC, Kurtenbach K, Maier WA and Seitz HM (2004) Substantial Rise in the Prevalence of Lyme Borreliosis Spirochetes in a Region of Western Germany over a 10-Year Period. Appl Environ Microbiol ; 70(3):1576-82. http://www.ncbi.nlm.nih.gov/pubmed/15006781

(21) Rauter C and Hartung T (2005) Prevalence of Borrelia burgdorferi Sensu Lato Genospecies in Ixodes ricinus Ticks in Europe: a Metaanalysis. Appl. Environ. Microbiol ; 71 (11) 7203-7216. http://aem.asm.org/content/71/11/7203.full

(22) Hao Q, Hou X, Geng Z and Wan K (2011) Distribution of Borrelia burgdorferi Sensu Lato in China. J Clin Microbiol; 49(2): 647-650. http://www.ncbi.nlm.nih.gov/pubmed/21106783

(23) Bronwyn Herbert (ABC) Report on Lyme Green Australia Blog Australian's Lyme Disease Testing Inadequate - ABC

Reservoir Host Studies

(1) Dubska L, Literak I, Kocianova E, Taragelova V, Sverakova V, Sychra O and Hromadko M (2011) Synanthropic birds influence the distribution of Borrelia species: analysis of Ixodes ricinus ticks feeding on passerine birds. Appl Environ Microbiol;77(3):1115-7. Epub 2010 Dec 10. http://www.ncbi.nlm.nih.gov/pubmed/21148704

(2) Marie-Angele P, Lommano, E, Humair PF, Douet V, Rais O, Schaad M, Jenni L and Gern L (2006) Prevalence of Borrelia burgdorferi Sensu Lato in Ticks Collected from Migratory Birds in Switzerland. Appl Environ Microbiol;72(1):976-9. http://www.ncbi.nlm.nih.gov/pubmed/16391149

(3) Kurtenbach K, Peacey M, Rijpkema SG, Hoodless AN, Nuttall PA and Randolph SE (1998) Differential transmission of the genospecies of Borrelia burgdorferi sensu lato by game birds and small rodents in England. Appl Environ Microbiol;64(4):1169-74. http://www.ncbi.nlm.nih.gov/pubmed/9546150

(4) Wildlife Tourism Australia. Accessed August 2011, http://wildlifetourism.org.au/experiencing-our-wildlife/australian-wildlife-types/

(5) Carly JG and Pope JH (1962) A new species of Borrelia (B. queenslandica) from Rattus villosissimus in Queensland. Aust J Exp Biol Med Sci; 40:255-61. http://www.ncbi.nlm.nih.gov/pubmed/13876596

(6) Mackerras MJ (1959) The haematozoa of Australian mammals. Australian Journal of Zoology, 7(2)105 – 135. http://www.publish.csiro.au/paper/ZO9590105.htm

(7) Australian Museum, Nature, Culture, Discover. "Black Rat" Accessed August 2011, http://australianmuseum.net.au/Black-Rat

(8) Christova I and Gladnishka T (2005) Prevalence of Infection with Francisella Tularensis, Borrelia Burgdorferi Sensu Lato and Anaplasma Phagocytophilum in Rodents from an Endemic Focus of Tularemia in Bulgaria. Ann Agric Environ Med, 12, 149–152. http://www.ncbi.nlm.nih.gov/pubmed/16028881

(9) Matuschka F-R, Endepolis A, Richter D and Spielman A (1997) Competence of urban rats as reservoir hosts for Lyme disease spirochetes. J Med Entomol. 34, 489-493. http://www.ncbi.nlm.nih.gov/pubmed/9220684

(10) Rats and rodents: Black Rat, Norway Rat and the House Mouse. Environmental Health Organisation. Greg McAvoy, EHO, Indigenous Communities Environmental Health. A National Workforce Capacity Building Program. Accessed August 2011. http://iceh.uws.edu.au/docs/rats_rodents.html

(11) Smith, R. P., P. W. Rand, E. H. Lacombe, S. R. Telford, S. M. Rich, J. Piesman, and A. Spielman. 1993. Norway rats as reservoir hosts for Lyme disease spirochetes on Monhegan Island, Maine. J. Infect. Dis. 168:687–691. http://www.ncbi.nlm.nih.gov/pubmed/8354910

(12) Shih CM, Chang HM, Chen SL and Chao LL (1998) Genospecies Identification and Characterization of Lyme Disease Spirochetes of Genospecies Borrelia burgdorferi Sensu Lato Isolated from Rodents in Taiwan. Journ Clin Microbiol, 36 (11) 3127-3132. http://jcm.asm.org/cgi/content/full/36/11/3127

(13) Davies P and Garvey J (2011) Early Zooarchaeological Evidence for Mus musculus in Australia. International Journal of Osteoarchaeology. http://onlinelibrary.wiley.com/doi/10.1002/oa.1244/abstract

(14) Rats, mice and people: rodent biology and management. Symposium 5: Population Ecology and Modeling. Australian Centre for International Agricultural Research 2003. ISBN 1 86320 357 5 http://aciar.gov.au/files/node/451/mn96chapter3.pdf

(15) Stevenson B, El-Hage N, Hines MA, Miller JC and Babb K (2002) Differential Binding of Host Complement Inhibitor Factor H by Borrelia burgdoreri Erp Surface Proteins: a Possible Mechanism Underlying the Expansive Host Range of Lyme Disease Spirochetes. Infect Immun, 70 (2) 491-497. http://iai.asm.org/content/70/2/491.full

(16) Talleklint L, Jaenson TG (1993) Maintenance by hares of European Borrelia burgdorferi in ecosystems without rodents. J Med Entomol, 30(1):273-6. http://www.ncbi.nlm.nih.gov/pubmed/8433337

(17) Talleklint L and Jaenson TG (1994) Transmission of Borrelia burgdorferi s.l. from mammal reservoirs to the primary vector of Lyme borreliosis, Ixodes ricinus (Acari: Ixodidae), in Sweden. J Med Entomol, 31(6):880-6. http://www.ncbi.nlm.nih.gov/pubmed/7815401

(18) World Association of Zoos and Aquariums http://www.waza.org/en/zoo/visit-the-zoo/rodents-and-hares/lepus-capensiseuropaeus

(19) Lyme Disease / Borreliosis: An overview of Lyme and direction for further research required in Australia. Karen Smith, May 2013. ISBN: 978-0-646-90428-3. http://www.lymeaustralia.com/k-smith-lara-research.html

(20) Richter, D. and Matuschka, F.R. (2006) Perpetuation of the Lyme Disease Spirochete Borrelia lusitaniae by Lizards, Applied and Environmental Microbiology, 72(7) 4627-4632. http://www.cfsph.iastate.edu/Factsheets/pdfs/lyme_disease.pdf

(21) Comstedt P, Bergstrom S, Olsen B, Garpmo U, Marjavaara L, Meilon H, Barbour AG and Bunikis J (2006) Migratory passerine birds as reservoirs of Lyme borreliosis in Europe. Emerg Infect Dis;12(7):1087-95. http://www.ncbi.nlm.nih.gov/pubmed/16836825

(22) Dubska L, Literak I, Kocianova E, Taragelova V, Sverakova V, Sychra O and Hromadko M (2011) Synanthropic birds influence the distribution of Borrelia species: analysis of Ixodes ricinus ticks feeding on passerine birds. Appl Environ Microbiol;77(3):1115-7. Epub 2010 Dec 10. http://www.ncbi.nlm.nih.gov/pubmed/21148704

(23) Dubska L, Literak I, Kocianova E, Taragelova V and Sychra O (2009) Differential role of passerine birds in distribution of Borrelia spirochetes, based on data from ticks collected from birds during the postbreeding migration period in Central Europe. Appl Environ Microbiol;75(3):596-602. Epub 2008 Dec 5. http://www.ncbi.nlm.nih.gov/pubmed/19060160

(24) Taragel'ova V, Koci J, Hanincova K, Kurtenbach K, Derdakova M, Ogden NH, Literak I, Kocianova E and Labuda M (2008) Blackbirds and song thrushes constitute a key reservoir of Borrelia garinii, the causative agent of borreliosis in Central Europe. Appl Environ Microbiol;74(4):1289-93. Epub 2007 Dec 21. http://www.ncbi.nlm.nih.gov/pubmed/18156328

(25) Jordan BE, Onks KR, Hamilton SW, Hayslette SE and Wright SM (2009) Detection of Borrelia burgdorferi and Borrelia lonestari in birds in Tennessee. J Med Entomol; 46(1):131-8. http://www.ncbi.nlm.nih.gov/pubmed/19198527

(26) Kurtenbach K, Peacey M, Rijpkema SG, Hoodless AN, Nuttall PA and Randolph SE (1998) Differential transmission of the genospecies of Borrelia burgdorferi sensu lato by game birds and small rodents in England. Appl Environ Microbiol;64(4):1169-74. http://www.ncbi.nlm.nih.gov/pubmed/9546150

(27) Kurtenbach K, Carey D, Hoodless AN, Nuttall PA and Randolph SE (1998) Competence of pheasants as reservoirs for Lyme disease spirochetes. J Med Entomol; 35(1):77-81. http://www.ncbi.nlm.nih.gov/pubmed/9542349

(28) E Isogai, S Tanaka, I S Braga, 3rd, C Itakura, H Isogai, K Kimura, and N Fujii (1994) Experimental Borrelia garinii infection of Japanese quail. Infect Immun; 62 (8): 3580-3582. http://www.ncbi.nlm.nih.gov/pmc/articles/PMC302998/

(29) Burgess EC (1989) Experimental inoculation of mallard ducks (Anas platyrhynchos platyrhynchos) with Borrelia burgdorferi. J Wildl Dis; 25(1):99–102 http://www.ncbi.nlm.nih.gov/pubmed/2644453

(30) Gray JS (1998) Review, The Ecology of ticks transmitting Lyme borreliosis. J Experimental & Applied Acarology, 22 (5), 249-258. http://www.springerlink.com/content/t244893832806488/fulltext.pdf

(31) Daniel J. Salkeld DJ, Leonhard S, Girard YA, Hahn N, Mun J, Padgett KA, and Lane RS (2008) Identifying the Reservoir Hosts of the Lyme Disease Spirochete Borrelia burgdorferi in California: The Role of the Western Gray Squirrel (Sciurus griseus Am J Trop Med Hyg, 79 (4), 535-540. http://www.ncbi.nlm.nih.gov/pubmed/18840740

Vector Studies

(1) Lyme borreliosis in Europe: influences of climate and climate change, epidemiology, ecology and adaptation Measures By: Elisabet Lindgren, Thomas G.T. Jaenson, 2004. World Health Organisation, Europe. http://www.euro.who.int/__data/assets/pdf_file/0006/96819/E89522.pdf

(2) Wielinga PR, Gaasenbeek C, Fonville M, de Boer A, de Vries A, Dimmers W, Akkerhuis Op Jagers G, Schouls LM, Borgsteede F and van der Giessen JW (2006) Longitudinal Analysis of Tick Densities and Borrelia, Anaplasma, and Ehrlichia Infections of Ixodes ricinus Ticks in Different Habitat Areas in The Netherlands. Appl Environ Microbiol; 72(12):7594-601. http://www.ncbi.nlm.nih.gov/pubmed/17028227

(3) Strube C, Montenegro VM, Epe C, Eckelt E and Schnieder T (2010) Establishment of a minor groove binder-probe based quantitative real time PCR to detect Borrelia burgdorferi sensu lato and differentiation of Borrelia spielmanii by ospA-specific conventional PCR. Parasites & Vectors; 3:69. http://www.parasitesandvectors.com/content/pdf/1756-3305-3-69.pdf

(4) Cisak E, Wojcik-Fatla A, Stoiek N, Chmielewska-Badora J, Zwolinski J, Buczek A and Dutkiewicz J (2006) Prevalence of Borrelia burgdorferi genospecies in Ixodes ricinus ticks from Lublin region (eastern Poland). Ann Agric Environ Med;13(2):301-6. http://www.ncbi.nlm.nih.gov/pubmed/17196005

(5) Kirstein F, Rijpkema S, Molkenboer M and Gray JS (1997) Local variations in the distribution and prevalence of Borrelia burgdorferi sensu lato genomospecies in Ixodes ricinus ticks. Appl Environ Microbiol ; 63(3):1102-6. http://www.ncbi.nlm.nih.gov/pubmed/9055424

(6) Kampen H, Rotzel, D, Kurtenbach K, Maier WA and Seitz HM (2004) Substantial Rise in the Prevalence of Lyme Borreliosis Spirochetes in a Region of Western Germany over a 10-Year Period. Applied and Environmental Microbiology. Vol 70 (3) 1576-1582. http://www.ncbi.nlm.nih.gov/pubmed/15006781

(7) Alban PS, Johnson PW, and Nelson DR. (2000) Serum-starvation induced changes in protein synthesis and morphology of Borrelia burgorferi. Microbiology, 146, 119-127. http://www.ncbi.nlm.nih.gov/pubmed/10658658

(8) Brorson O, Brorson SH (1997) Transformation of cystic forms of Borrelia burgdorferi to normal, mobile spirochetes. Infection, 25 (4) 240-246. http://www.ncbi.nlm.nih.gov/pubmed/9266264

(9) Burgdorfer W (1999) . Lyme Disease, Survival in Adverse Conditions. The Strategy of Morphological Variation in Borrelia burgdorferi & Other Spirochetes 1900-2001. Keynote Address - The Complexity of Vector-borne Spirochetes. 12th International Conference on Lyme Disease and Other Spirochetal and Tick-Borne Disorders. http://www.lymeinfo.net/medical/LDAdverseConditions.pdf

(10) Scott JD, Lee MK, Fernando K, Jorgensen DR, Durden LA and Morshed MG (2008) Rapid introduction of Lyme disease spirochete, Borrelia burgdorferi sensu stricto, in Ixodes scapularis (Acari: Ixodidae) established at Turkey Point Provincial Park, Ontario, Canada. Journal of Vector Ecology. Vol 33 (1) 64-69. http://www.ncbi.nlm.nih.gov/pubmed/18697308

(11) Nelson JA, Bouseman JK, Kitron U,Callister SM, Harrison B, Bankowski MJ,Peeples ME,Newton BJ and Anderson JF (1991) Isolation and Characterization of Borrelia burgdorferi from Illinois Ixodes dammini. Journal of Clinical Microbiology, 29 (8) 1732-1734. http://www.ncbi.nlm.nih.gov/pmc/articles/PMC270194/pdf/jcm00044-0198.pdf

(12) Pollack RJ, Telford III SR and Spielman A. (1993). Standardization of Medium for Culturing Lyme Disease Spirochetes. Journal of Clinical Microbiology, May, 1251-1255. http://www.ncbi.nlm.nih.gov/pubmed/8501226

(13) Jobe DA, Callister SM, Schell RF (1993). Recovery of Borrelia burgdorferi by Filtration. Journal of Clinical Microbiology, July, 1896-1898. http://jcm.asm.org/content/31/7/1896.abstract

(14) Barbour, AG (1984) Isolation and Cultivation of Lyme Disease Spirochetes. The Yale Journal of Biology and Medicine, 57, 521-525. http://www.ncbi.nlm.nih.gov/pubmed/6393604

(15) Preac-Mursic V, Wilske B and Schierz G (1986) European Borrelia burgdorferi isolated from humans and ticks culture conditions and antibiotic susceptibility. Zentralbl Bakteriol Mikrobiol Hyg A; 263(1-2):112-8. http://www.ncbi.nlm.nih.gov/pubmed/3577473

(16) Campylobacter Selective Supplement, Skirrow Formula. Pro-Lab Diagnostics. http://www.pro-lab.com/inserts/PL501_460.pdf

(17) Dever LL, Jorgensen JH and Barbour AG (1993) In vitro activity of vancomycin against the spirochete Borrelia burgdorferi. Antimicrob Agents Chemother; 37(5):1115-21. http://www.ncbi.nlm.nih.gov/pubmed/8517700

(18) Kazragis RJ, Dever LL, Jorgensen JH and Barbour AG (1996) In vivo activities of ceftriaxone and vancomycin against Borrelia spp. in the mouse brain and other sites.Antimicrob Agents Chemother; 40(11):2632-6. http://www.ncbi.nlm.nih.gov/pubmed/8913478

(19) Reisinger EC, Wendelin L, and Gasser R (1997) In vitro activity of trimethoprim against Borrelia burgdorferi. Eur J Clin Microbiol Infect Dis; 16(6):458-60. http://www.ncbi.nlm.nih.gov/pubmed/9248750

(20) Yang X, Popova TG, Goldberg MS and Norgard MV (2001). Influence of Cultivation Media on Genetic Regulatory Patterns in Borrelia burgdorferi. Infection and Immunity, June, 4159-4163. http://www.ncbi.nlm.nih.gov/pubmed/11349092

(21) Callister SM,Case KL, Agger WA, Schell RF, Johnson RC, Elllingson JLE. (1990) Effects of Bovine Serum Albumin on the Ability of Barbour- Stoenner-Kelly Medium To Detect Borrelia burgdorferi. Journal Of Clinical Microbiology, Feb. 363-365. http://www.ncbi.nlm.nih.gov/pubmed/2179264

(22) Wodecka B, Leon´ ska A and Skotarczak B (2010) A comparative analysis of molecular markers for the detection and identification of Borrelia spirochaetes in Ixodes ricinus. Journal of Medical Microbiology, 59, 309–314. http://www.ncbi.nlm.nih.gov/pubmed/20007765

(23) Bosshard PP, Zbinden R and Altwegg M. (2002) Paenibacillus turicensis sp. nov., a novel bacterium harbouring heterogeneities between 16S rRNA genes. International Journal of Systematic and Evolutionary Microbiology, 52, 2241–2249. http://www.ncbi.nlm.nih.gov/pubmed/12508893

(24) Joung, KB and Cote JC (2002) Evaluation of ribosomal RNA gene restriction patterns for the classification of Bacillus species and related genera. Journal of Applied Microbiology, 92, 97-108. http://www.ncbi.nlm.nih.gov/pubmed/11849333

(25) Holt SC (1978) Anatomy and Chemistry of Spirochetes. Microbiological Reviews; 42(1) 114-160. http://www.ncbi.nlm.nih.gov/pmc/articles/PMC281421/pdf/microrev00001-0120.pdf

(26) Listgiamten MA and Socransky SS (1964) Election Microscopy of Axial Fibrils, Outer Envelope, and Cell Division of Certain Oral Spirochetes. Journ of Bacteriol; 88 (4), 1087-1103. http://www.ncbi.nlm.nih.gov/pmc/articles/PMC314859/pdf/jbacter00593-0293.pdf

(27) Fauna of Ixodid Ticks of the World (GV Kolonin 2009). http://www.kolonin.org/13_6.html#r89

(28) Ixodes holocyclus Neumann. CSRIO. http://www.ces.csiro.au/aicn/system/c_121.htm

(29) Tick Paralysis in Domestic Animals in Australia caused by Ixodes holocyclus (the Paralysis Tick) by Robert Wylie B.V.Sc.,Q.D.A. Ulladulla Veterinary Hospital. http://www.ullavet.com.au/tick.html

(30) Piesman J and Stone B (1991) Vector competence of the Australian paralysis tick, Ixodes holocyclus, for the Lyme disease spirochete Borrelia burgdorferi. Int. J. Parasitol; 21: 109–11. http://www.ncbi.nlm.nih.gov/pubmed/2040556

(31) Mazuzawa T (2004) Terrestrial distribution of the Lyme borreliosis agent Borrelia burgdorferi sensu lato in East Asia. Jpn J Infect Dis ; 57(6):229-35. http://www.ncbi.nlm.nih.gov/pubmed/15623946

(32) Wills MC and Barry RD (1991) Detecting the cause of Lyme Disease in Australia. Med J Aust; 155(4):275. http://www.ncbi.nlm.nih.gov/pubmed/1875848

(33) Fauna of Ixodid Ticks of the World (GV Kolonin 2009). http://www.kolonin.org/11_1.html#r10

(34) Haemaphysalis bancrofti Nuttall & Warburton CSRIO http://www.ces.csiro.au/aicn/system/c_113.htm

(35) Stewart NP, de Vos AJ, Shiels IA and Jorgensen WK (1989) Transmission of Theileria buffeli to cattle by Haemaphysalis bancrofti fed on artificially infected mice. Vet Parasitol; 34(1-2):123-7. http://www.ncbi.nlm.nih.gov/pubmed/2588463

(36) Theileriosis in Australia – an emerging disease. Petulia,, A World of Petcare http://www.petalia.com.au/Templates/StoryTemplate_Process.cfm?specie=Beef&story_no=2161

(37) Benign Bovine Theileriosis – A Questionnaire of 64 Affected Properties. Flock and Herd Case Notes. http://www.flockandherd.net.au/cattle/reader/benign%20bovine%20theileriosis.html

(38) Theileriosis in NSW Cattle disease more serious. From the April 2009 edition of Agriculture Today. NSW Department Primary Industries http://www.dpi.nsw.gov.au/archive/agriculture-today-stories/april-2009/cattle-disease-more-serious

(39) Borreliosis and Associated Diseases Awareness UK. Babesiosis : http://www.bada-uk.org/babesiosis#.UnvaI9W4bIU

(39) Human Babesiosis. UCL International Institute of Cellular and Molecular Pathology http://www.icp.ucl.ac.be/~opperd/parasites/human_babesiosis.html

(40) Thompson C, Spielman A, Krause PJ (2001) Coinfecting Deer-Associated Zoonoses: Lyme Disease, Babesiosis, and Ehrlichiosis. Clin Infect Dis, Sep 1;33(5):676-85.Epub 2001 Aug 6. http://www.ncbi.nlm.nih.gov/pubmed/11486290

(41) Mackerras MJ (1959) The haematozoa of Australian mammals. Australian Journal of Zoology, 7(2)105 – 135. http://www.publish.csiro.au/paper/ZO9590105.htm

(42) Haemaphysalis longicornis http://www.kolonin.org/11_5.html#r81

(43) Haemaphysalis longicornis Neumann, CSRIO ; http://www.ces.csiro.au/aicn/system/c_116.htm

(44) Ticks, Bees, Fleas, Flies, Spiders, and other Gremlins. Ticks in Australia, Lowchens ens Australia; http://www.lowchensaustralia.com/pests/bites.htm

(45) New Zealand BioSecure Entomology Laboratory. Haemaphysalis longicornis profile. R Cane 2010. http://www.smsl.co.nz/site/southernmonitoring/files/NZB/Ha%20longicornis%20Profile.pdf

(46) Introduced / Pest Animals. Office of Environment and Heritage NSW Government http://www.environment.nsw.gov.au/pestsweeds/pestanimals.htm

(47) Skotarczak B (2002) Canine borreliosis--epidemiology and diagnostics. Ann Agric Environ Med; 9(2):137-40. http://www.ncbi.nlm.nih.gov/pubmed/12498579

(48) Mather TN, Fish D and Coughlin RT (1994) Competence of dogs as reservoirs for Lyme disease spirochetes (Borrelia burgdorferi). J Am Vet Med Assoc; 205(2):186–188. http://www.ncbi.nlm.nih.gov/pubmed/7928571

(49) Krupka I and Straubinger RK (2010) Lyme borreliosis in dogs and cats: background, diagnosis, treatment and prevention of infections with Borrelia burgdorferi sensu stricto. Vet Clin North Am Small Anim Pract; 40(6):1103-19. http://www.ncbi.nlm.nih.gov/pubmed/20933139

(50) Shaw SE, Binns SH, Birtles RJ, Day MJ, Smithson R and Kenny MJ (2005) Molecular evidence of tick-transmitted infections in dogs and cats in the United Kingdom. Vet Rec;157(21):645-8. http://www.ncbi.nlm.nih.gov/pubmed/16299364

(51) Magnarelli LA, Anderson JF, Levine HR and Levy SA (1990) Tick parasitism and antibodies to Borrelia burgdorferi in cats. J Am Vet Med Assoc; 197(1):63-6. http://www.ncbi.nlm.nih.gov/pubmed/2196252

(52) Magnarelli LA, Bushmich SL, IJdo JW and Fikrig E (2005) Seroprevalence of antibodies against Borrelia burgdorferi and Anaplasma phagocytophilum in cats. Am J Vet Res; 66(11):1895-9. http://www.ncbi.nlm.nih.gov/pubmed/16334946

(53) Schoffel I, Schein E, Wittstadt U and Hentsche J (1991) Parasite fauna of red foxes in Berlin (West). Berl Munch Tierarztl Wochenschr;104(5):153-7. http://www.ncbi.nlm.nih.gov/pubmed/1872791

(54) Gern L (2008) Borrelia burgdorferi sensu lato, the agent of lyme borreliosis: life in the wilds. Parasite; 15(3):244-7. http://www.ncbi.nlm.nih.gov/pubmed/18814688 Full copy: http://parasite-journal.org/dwld/Parasite08-3_244-247_Gern.pdf

(55) Stefancikova A, Adaszek L, Pet'ko B, Winjarczyk S and Dudinak V (2008) Serological evidence of Borrelia burgdorferi sensu lato in horses and cattle from Poland and diagnostic problems of Lyme borreliosis. Ann Agric Environ Med; 15(1):37-43. http://www.ncbi.nlm.nih.gov/pubmed/18581977

(56) Takahashi K, Isogai E, Isogai H, Takagi T, Sasaki K, Fujii N and Kimura K (1993) Serological survey for Borrelia burgdorferi infection in cattle in southern Hokkaido. J Vet Med Sci; 55(6):921-4. http://www.ncbi.nlm.nih.gov/pubmed/8117816

(57) Carter SD, May C, Barnes A and Bennett D (1994) Borrelia burgdorferi infection in UK horses. Equine Vet J; 26(3):187-90. http://www.ncbi.nlm.nih.gov/pubmed/8542836

(58) Magnarelli LA, Anderson JF, Shaw E, Post JE and Palka FC (1998) Borreliosis in equids in northeastern United States. Am J Vet Res; 49(3):359-62. http://www.ncbi.nlm.nih.gov/pubmed/3282461

(59) Parker JL and White KK (1992) Lyme borreliosis in cattle and horses: a review of the literature. Cornell Vet; 82(3):253-74. http://www.ncbi.nlm.nih.gov/pubmed/1643876

(60) James FM, Engiles JB and Beech J (2010) Meningitis, cranial neuritis, and radiculoneuritis associated with Borrelia burgdorferi infection in a horse. J Am Vet Med Assoc; 237(10):1180-5. http://www.ncbi.nlm.nih.gov/pubmed/21073390

(61) Burgess EC, Mattison M: 1987, Encephalitis associated with Borrelia burgdorferi infection in a horse. J Am Vet Med Assoc 191:1457–1458. http://www.ncbi.nlm.nih.gov/pubmed/3692996

(62) Sorensen K, Neely DP, Grappell PM and Read W (1990) Lyme disease antibodies in Thoroughbred mares, correlation to early pregnancy failure. Equine Vet J; 10(3):166–168. http://www.sciencedirect.com/science/article/pii/S073708060680153X

(63) Fridriksdóttir V, Nesse LL, and Gudding R (1992) Seroepidemiological Studies of Borrelia burgdorferi Infection in Sheep in Norway. J Clin Microbiol. 1992 May; 30(5): 1271–1277. http://www.ncbi.nlm.nih.gov/pmc/articles/PMC265263/

(64) Hoymark A, Asbrink E, Schwan O, Hederstedt B and Christensson D (1986) Antibodies to Borrelia spirochetes in sera from Swedish cattle and sheep. Acta Vet Scand; 27(4):479-85. http://www.ncbi.nlm.nih.gov/pubmed/3604822

(65) Isogai E, Isogai H, Masuzawa T, Yanagihara Y, Sato N, Hayashi S, Maki T and Mori M (1991) Serological survey for Lyme disease in sika deer (Cervus nippon yesoensis) by enzyme-linked immunosorbent assay (ELISA). Microbiol Immunol; 35(9):695-703. http://www.ncbi.nlm.nih.gov/pubmed/1808467

(66) Wan K, Zhang Z, and Dou G (1998) Investigation on primary vectors of Borrelia burgdorferi in China. Chin J Epidemiol 19, 263–266 http://www.ncbi.nlm.nih.gov/pubmed/10322682

(67) Hao Q, Hou X, Geng Z and Wan K (2011) Distribution of Borrelia burgdorferi Sensu Lato in China. J Clin Microbiol; 49(2): 647-650. http://www.ncbi.nlm.nih.gov/pubmed/21106783

(68) Chu CY, Jiang BG, Liu W, Zhao QM, Wu XM, Zhang PH, Zhan H and Cao WC (2008). Presence of pathogenic Borrelia burgdorferi sensu lato in ticks and rodents in Zhejiang, south-east China. J Med Microbiol;57(8):980-5 http://www.ncbi.nlm.nih.gov/pubmed/18628499

(69) Chu CY, Liu W, Jiang BG, Wang DM, Jiang WJ, Zhao QM, Zhang PH, Wang ZX, Tang GP, Yang H and Cao WC (2008) Novel Genospecies of Borrelia burgdorferi Sensu Lato from Rodents and Ticks in Southwestern China. J Clin Microbiol; 46(9):3130-3 http://www.ncbi.nlm.nih.gov/pubmed/18614645

(70) Sun J, Liu Q, Lu L, Ding G, Guo J, Fu G, Zhang J, Meng F, Wu H, Song X, Ren D, Li D, Guo Y, Wang J, Li G, Liu J and Lin H (2008) Coinfection with four genera of bacteria (Borrelia, Bartonella, Anaplasma, and Ehrlichia) in Haemaphysalis longicornis and Ixodes sinensis ticks from China. Vector Borne Zoonotic Dis; 8(6): 791-5. http://www.ncbi.nlm.nih.gov/pubmed/18637722

(71) Rothwell JT, Christie BM, Williams C and Walker KH (1989) Suspected Lyme disease in a cow. Aust Vet J; 66(9):296-8. http://www.ncbi.nlm.nih.gov/pubmed/2684126

(72) Fauna of Ixodid Ticks of the World (GV Kolonin 2009). http://www.kolonin.org/4_4.html#r57

(73) Amblyomma moreliae (L. Koch). CSRIO http://www.ces.csiro.au/aicn/system/c_105.htm

(74) Richter, D. and Matuschka, F.R. (2006) Perpetuation of the Lyme Disease Spirochete *Borrelia lusitaniae* by Lizards, Applied and Environmental Microbiology, 72(7) 4627-4632. http://aem.asm.org/content/72/7/4627.full

(75) Ekner A, Dudek K, Sajkowska Z, Mailathova V, Mailath I and Tryjanowski P (2011) Anaplasmataceae and Borrelia burgdorferi sensu lato in the sand lizard Lacerta agilis and co-infection of these bacteria in hosted Ixodes ricinus ticks. Parasit Vectors; 20;4:182. doi: 10.1186/1756-3305-4-182. http://www.ncbi.nlm.nih.gov/pubmed/21933412

(76) Olsen B, Jaenson TG, Noppa L, Bunikis J, Bergstrom S (1993) A Lyme borreliosis cycle in seabirds and Ixodes uriae ticks. Nature; 362:340-342. http://www.ncbi.nlm.nih.gov/pubmed/8455718

(77) Olsen B, Duffy DC, Jaenson TG, Gylfe A, Bonnedahl J and Bergstrom S (1995) Transhemispheric exchange of Lyme disease spirochetes by seabirds. J Clin Microbiol; 33:3270-3274. http://www.ncbi.nlm.nih.gov/pubmed/8586715

(78) Scott JD, Anderson JF and Durden LA (2011) Widespread dispersal of Borrelia burgdorferi-infected ticks collected from songbirds across Canada. J Parasitol Aug 24. [Epub ahead of print] http://www.ncbi.nlm.nih.gov/pubmed/21864130

(79) Scott JD, Lee MK, Fernando K, Durden LA, Jorgensen DR, Mak S and Morshed MG (2010) Detection of Lyme disease spirochete, Borrelia burgdorferi sensu lato, including three novel genotypes in ticks (Acari: Ixodidae) collected from songbirds (Passeriformes) across Canada. J Vector Ecol;35(1):124-39. http://www.ncbi.nlm.nih.gov/pubmed/20618658

(80) Morshed MG, Scott JD, Fernando K, Beati L, Mazerolle DF, Geddes G and Durden LA (2005) Migratory songbirds disperse ticks across Canada, and first isolation of the Lyme disease spirochete, Borrelia burgdorferi, from the avian tick, Ixodes auritulus. J Parasitol;91(4):780-90. http://www.ncbi.nlm.nih.gov/pubmed/17089744

(81) CSRIO: Ixodes uriae White: http://www.ces.csiro.au/aicn/system/c_127.htm

(82) Olsen B, Jaenson TG, Noppa L, Bunikis J, Bergstrom S (1993) A Lyme borreliosis cycle in seabirds and Ixodes uriae ticks. Nature; 362:340-342. http://www.ncbi.nlm.nih.gov/pubmed/8455718

(83) Olsen B, Duffy DC, Jaenson TG, Gylfe A, Bonnedahl J and Bergstrom S (1995) Transhemispheric exchange of Lyme disease spirochetes by seabirds. J Clin Microbiol; 33:3270-3274. http://www.ncbi.nlm.nih.gov/pubmed/8586715

(84) Ixodes auritulus: Fauna of Ixodid Ticks of the World: GV Kolonin 2009: http://www.kolonin.org/13_1.html#r18

(85) Ticks of Australia: http://www.lowchensaustralia.com/pests/paralysis-tick/ticks-of-australia.htm

(86) Csurhes S and Markula A (2010) Pest Animal Risk Assessment: Blackbird (Turdus merula) The State of Queensland, Department of Employment, Economic Development and Innovation. Biosecurity Queensland. http://www.dpi.qld.gov.au/documents/Biosecurity_EnvironmentalPests/IPA-Blackbird-Risk-Assessment.pdf

(87) Warning: Common Blackbird (Turdus merula) Qld Government, Department of Primary industries and Fisheries: http://www.dpi.qld.gov.au/documents/Biosecurity_EnvironmentalPests/IPA-Common-Blackbird-Warning.pdf

(88) Mapped Occurance Records, Turdus: Atlas of Living Australia: http://bie.ala.org.au/species/urn:lsid:biodiversity.org.au:afd.taxon:56eb79bd-1f99-4040-850a-4cb21bfd5ce5;jsessionid=99B440CEC69F3C0D172174055775D50C

(89) Comstedt P, Bergstrom S, Olsen B, Garpmo U, Marjavaara L, Meilon H, Barbour AG and Bunikis J (2006) Migratory passerine birds as reservoirs of Lyme borreliosis in Europe. Emerg Infect Dis;12(7):1087-95. http://www.ncbi.nlm.nih.gov/pubmed/16836825

(90) Dubska L, Literak I, Kocianova E, Taragelova V, Sverakova V, Sychra O and Hromadko M (2011) Synanthropic birds influence the distribution of Borrelia species: analysis of Ixodes ricinus ticks feeding on passerine birds. Appl Environ Microbiol;77(3):1115-7. Epub 2010 Dec 10. http://www.ncbi.nlm.nih.gov/pubmed/21148704

(91) Dubska L, Literak I, Kocianova E, Taragelova V and Sychra O (2009) Differential role of passerine birds in distribution of Borrelia spirochetes, based on data from ticks collected from birds during the postbreeding migration period in Central Europe. Appl Environ Microbiol;75(3):596-602. Epub 2008 Dec 5. http://www.ncbi.nlm.nih.gov/pubmed/19060160

(92) Taragel'ova V, Koci J, Hanincova K, Kurtenbach K, Derdakova M, Ogden NH, Literak I, Kocianova E and Labuda M (2008) Blackbirds and song thrushes constitute a key reservoir of Borrelia garinii, the causative agent of borreliosis in Central Europe. Appl Environ Microbiol;74(4):1289-93. Epub 2007 Dec 21. http://www.ncbi.nlm.nih.gov/pubmed/18156328