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Lyme disease in Australia

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Some people still hold the view that Lyme disease does not exist in Australia, partly because the symptoms of people infected here have been found to be slightly different to the symptoms of those infected in North America,[1] where the spirochete, *Borrelia burgdorferi*, was first discovered and also because serological evidence has been inconclusive.

In European countries and Australia, there have been central nervous system (CNS) complaints of sharp, shooting pains and migraines in the absence of any rashes or arthritic pain,[2-4] whereas in North America there are more peripheral arthritic complaints than direct CNS complaints,[5,6] although people with both sets of complaints do exist in these areas of the world.

Clinical Lyme specialists (e.g. Bernie Hudson and Janet Kitchener-Smith) in NSW have seen strikingly similar/identical symptoms and response to treatment post tick bite from many patients bitten in the northern beaches area of Sydney as those with positive serology from other countries. In addition, different symptoms could be a result of different DNA strands of the spirochete within CNS and peripheral systems.[7,8]

Denial of the existence of Lyme in Australia based on different symptoms of patients here versus in America is not justifiable and may be hazardous from a treatment perspective, particularly given recent evidence suggesting that the spirochete may exist in Pittwater, NSW.[9] Scepticism lies in the fact that the patient who recently tested positive had spent time in Europe 17 months before the onset of symptoms so there is a chance that the infection was gained overseas. The patient was adamant, however, that he was not bitten by a tick in Europe, but was bitten in Pittwater.

Lyme researchers in Australia are of the strong opinion that the spirochete exists here, but behaviours of the spirochete and our immune systems make it difficult to detect serologically. In addition, the only laboratory every to perform Lyme serologies for patients from NSW (Infectious Disease Pathology laboratory, University of Newcastle) was only in operation for less than one year. Clearly, without adequate research, the task of identifying the spirochete in NSW has been difficult (for information on laboratories performing serologies throughout Australia, see Russell[10]). (Note: the Ixodes tick, which carries the disease, is endemic in the northern beaches of Sydney, where the largest number of Lyme symptom complaints originate from.)[11]

The etiology(ies) behind these symptoms is (are) not exactly clear, but could be a direct result of the micro-organism *Borrelia burgdorferi* which invades the body or the reactions of the immune system to the spirochete.

The causative agent of Lyme, the spirochete *B. burgdorferi*, has been detected in the cerebrospinal fluid of many persons with the symptoms of Lyme.[11] In most cases, however, symptoms of Lyme exist in the absence of positive Borrelia serology.[12] This pernicious and ingenious micro-organism has a long replication time, may undergo genetic variations once in the CNS,[7] can hide in intracellular locations[13,14] and in difficult to penetrate sites (e.g CNS, joints, anterior chamber of the eye).[15] These features make it very difficult to detect or kill, which is why treatment decisions must be made based on the symptomatology of the patients and exposure to endemic areas for Lyme disease as opposed to positive serology.

Lyme may persist for such a lengthy time after initial infection as a result of the *B. burgdorferi* synthesising large amounts of interleukin-1 necessary for T-cells to mature rapidly, as well as from the excitotoxicity of quinolinic acid and n-methyl-n-aspartate. It appears that both the spirochete and the immune system may interact to maintain the disease.

In summary, given exposure to a tick, particularly in a proposed endemic area, and even one symptom of Lyme, the logical decision appears to be to offer treatment and ask more detailed questions later.

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