Borrelia burgdorferi has not to my knowledge been grown or shown by PCR to be present in Australia in ticks, animals or people using a NATA/RCPA accredited laboratory and using NATA approved methods for the testing. Other Borrelia are present in Australia however. These are in the same genus/family as burgdorferi but are different to Borrelia burgdorferi.

The implications of other Borrelia species being present in Australia is they can look similar when initially isolated, they will look very similar if I looked under a microscope and more importantly there be there maybe cross-reactions with Lyme disease if testing is done by serology (antibodies because of cross-reactions). All bacteria that are similar will have some similar structures in their cell wall (e.g. some proteins). Therefore if people have been infected with any of these are bacteria they will have make antibodies against these proteins that may cause a false positive result on any blood test looking at antibodies for a very specific bacterial infection e.g. with Borrelia burgdorferi .

A good example of this is Rickettsia which are often in ticks. There are many different species of Rickettsia but if someone is infected with one species they will often have antibodies present to many of the other species (although usually in lower titre levels).

If Borrelia are found by PCR it is important that one knows the makeup of the PCR primer sequence. Then with the product that is amplified by PCR it is important that that product is able to be completely sequenced to make sure that what has been found is in fact Borrelia burgdorferi rather than another Borrelia or other bacteria.

A point has been made about long term antibiotics being used in acne. The major bacteria involved in acne are strains of Propionibacterium acnes. I have been involved in research with GP's on this issue over a number of years. There is no doubt that in people on antibiotics for acne that a proportion will develop antibiotic resistant strains. The proportion developing carrying resistant bacteria is usually over time proportionate to the numbers of people being treated with antibiotics. When the organism is resistant to the antibiotic the acne does not respond very well when the antibiotic is ingested. More importantly when resistant strains of Propionibacterium acnes develop, if then another family member develops acne they are more likely to have a the same resistant strain and also have a relatively poor response to antibody therapy.

I understand also that many people involved with advocacy for Lyme-Like disease have discussed pleomorphism, the ready acquisition of genetic material and biofilm formation and why these issues makes Lyme disease different to other infections. Biofilm formation, plasmid acquisition and pleomorphism are not unique to bacteria in the genus Borrelia. Pleomorphism is readily seen in routine laboratory practice especially when a cell-wall activate antibacterial agent is present. Such forms (often called L forms) do not necessarily make the bacteria any more successful in vivo and often can make them less likely to survive. It is seen for instance with Streptococcal species. The rigid cell wall for many bacteria is very important. Most bacteria have high internal osmotic pressures. The rigid cell wall then effectively stops them from bursting and dying if in a normal osmotic environment (e.g. in most area of the human body). Beta-lactam antibiotics (e.g. penicillin and ceftriaxone) work by disrupting the rigid cell wall and then effectively kill bacteria by letting

them “explode”. Hence these pleomorphic bacterial L- forms for most situations are a disadvantage to the bacteria being able to cause active infection.

Biofilms are important and especially develop with foreign material. Catheters used for long term antimicrobial administration are a common example when they become colonised with bacteria and which occurs frequently after they have been in place for 3 days or more. This is an important reason for avoiding the use of long term intravenous antibiotics in patients presenting with this chronic debilitating illness of unknown ætiology.

The acquisition of new genetic material occurs with many bacteria. Enterococcus and E.coli are examples where this is very frequent. It involved both the acquisitions of antibiotic resistance genes (often as plasmids) and also genes that can increase their ability to cause disease (virulence). This is therefore not unique to bacteria in the genus Borrelia and occurs more commonly in many very common bacteria causing large numbers of infection in people (e.g. E.coli). If there are claims to the contrary that show Borrelia are unique for these issues compared to other bacteria I would be interested in seeing any peer reviewed literature that supports that theory.

Dr Derham and Dr Nuttall from their testimony in Perth seem to believe that antibiotic resistance is not an issue if people are treated with ceftriaxone and for Lyme disease like symptoms. I think this is patently not true. In hospitals we currently have programs to decrease the use of ceftriaxone because of the known resistance rates that rise with the use of this drug in hospitals. In food animals overseas it has also been clearly shown that when you use the 3rd generation cephalosporins similar to ceftriaxone (e.g. ceftiofur) that

resistant bacteria develop in those animal populations. More importantly in The Netherlands and elsewhere these Superbugs (ESBL E.coli and salmonella) have been shown to spread to people directly and through the food chain and via water. In hospitals in Australia and elsewhere we wouldn't recommend the use of prolonged ceftriaxone. More than 1 or 2 weeks of therapy would be very unusual for any patient to receive. We would almost never use the length of therapy or high doses that is being requested in some people with Lyme like disease in Australia by some medical practitioners (e.g. more than 6 months).

What I think is beyond doubt with ceftriaxone or any other antibiotic is that antibiotic resistance is associated with resistance and the larger the volumes and length of time the antibiotics are used then the more antibiotic resistance develops and spreads.

If you have any other queries you would like me to respond to I'm more than happy to supply whatever information you need.

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