SUBMISSION TO THE SENATE INQURY
"The growing evidence of an emerging tick-borne disease that causes a Lyme-like illness for many Australian patients"

SUBMISSION FROM A MEMBER OF THE PUBLIC
TECHNICAL AND PERSONAL SUBMISSION

REGARDING CONGENITAL BORRELIA INFECTION AND AUTISM

This submission:

Section A – Congenital Borrelia Infection and Autism (Page 2)

This section has been prepared to explain how congenital borrelia infection is causative in the development of autism, a position denied by the Government/Australian Medical Authorities.

Section B – Personal Accounts – Borrelia Infection (Page 28)

This section provides an overview of the personal impacts associated with undiagnosed and untreated chronic borrelia, bartonella and babesia infection resulting in congenital infection of two children who are now disabled and know nothing but a life of ill health. This personal account supports the Technical Aspects presented in Section A. Please take the time to read this submission; it is the human side of the technical debate.

SUBMISSION BY:

Names & Age: [redacted] Age [redacted] years
Submission on behalf of: [redacted] Age [redacted] years
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THIS SUBMISSION IS TO BE NAME WITHHELD
I want my submission to be able to be read and downloaded from the Senate website. Do not publish my name, the names of my family, doctor's names or anybody's contact details including address or email details in the submission body as well as in the attachments.

Please contact me if any further supporting evidence / information is required.

SECTION A

CONGENITAL BORRELIA INFECTION AND AUTISM

SECTION B PERSONAL ACCOUNTS - BORRELIA INFECTION starts on page 26

SUBMISSION CONTENTS:

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Terms of Reference c - the process for diagnosis of patients with a Lyme-like illness, with a specific focus on the laboratory testing procedures and associated quality assurance processes, including recognition of accredited international laboratory testing.

Terms of Reference f - the signs and symptoms Australians with Lyme-like illness are enduring, and the treatment they receive from medical professionals; and

Terms of Reference g - any other related matters.

SECTION A – CONGENITAL BORRELIA INFECTION AND AUTISM

1.0 SUMMARY OF ISSUES - BORRELIA DIAGNOSIS IN AUSTRALIA

The arguments surrounding the denial of borrelia in Australia (overseas acquired, locally acquired congenitally acquired) firstly concern the ability of the existing system to identify, diagnose and treat those persons presenting with a borrelia infection, and secondly concern the existence of an Australian endemic Lyme-like illness (an illness that greatly resembles an infection with borrelia). Pertinent issues are summarised:

1. Acceptance of borrelia diagnosis is restricted to acceptance of results produced by the NATA/RCPA certified laboratories in Australia. These laboratories can detect up to 3 of the 39/40 strains of borrelia known to be human infective. In the event a patient presents with a strain of infection other than these 3, they will not be diagnosed in Australia.

2. A diagnosis from any other laboratory is rejected in Australia. The reason provided to the public, is that other laboratories are not NATA accredited. NATA (National Association of Testing Authorities) is an Australian organisation and primarily relates to accreditation of testing facilities in Australia.

   Australia can now legitimately recognise the results from many international laboratories, as on the 6th January 2016 after seeking membership, NATA was granted membership to the International Laboratory Accreditation Cooperation (ILAC). This means that ISO/IEC 15189:2012 and ISO/IEC 17043:2010 accreditation granted by NATA to Medical Testing laboratories and Proficiency Testing Providers respectively, is internationally recognised and ensures the results of activities (pathology tests) conducted by these facilities can be accepted worldwide. Likewise, the test results from other members of the ILAC are now able to be recognised and accepted by NATA labs. However Australia continues to reject laboratory results from ISO/IEC 15189:2012 and ISO/IEC 17043:2010 accredited labs without explanation.

3. The NATA/RCPA laboratories use a two-step test, being an ELISA followed by a Western Blot. This two-step system effectively misses over half of patients infected with borrelia. These two step system is not a direct identification of borrelia infection (direct test), it is an indirect test that can at best prove an immune response to the infection. The indirect testing is not proof of active infection and can be easily dismissed as a false positive or as evidence of a past infection. This is the position used to deny the presence of positive diagnosis of borrelia from the NATA/RCPA laboratories.

4. As there has been no endemic strain of borrelia identified, the official government advice is that diagnosis cannot be done clinically, but should rely on pathology. Refer to page 2 of the Federal Health Minister's Letter in Attachment A. This is at odds with accepted world wide practice that borrelia is a clinical diagnosis.

5. The Australian Health Practitioner Regulation Agency has taken action against medical professionals that treat borrelia, discouraging doctors from treating borrelia infection.

6. The majority of doctors encountered by the borreliosis patient can not / will not provide effective treatment to the borreliosis patient.

7. Patients suffering Lyme-like-illness or borreliosis in Australia are frequently subject to unprofessional treatment by the medical profession.

8. The medical profession is not disseminating accurate information to practitioners regarding the prevalence, symptoms, or treatment for tick borne infections.

9. Government funding towards identifying the cause of the endemic lyme like illness is scant. Research is hampered by funding, which has to be sourced through charity donations or university research.

10. Borrelia is not reportable, giving the government no way of monitoring an infection that is epidemic elsewhere in the world.

11. Congenital infection of borrelia is being denied despite vast evidence to the contrary.

12. The scientifically established relationships between chronic Bacterial and Viral Infection and Neurodegenerative and Neurobehavioral Diseases are being ignored, despite significant published evidence. Diseases include: Autism Spectrum Disorder, Multiple sclerosis, Amyotrophic lateral sclerosis, Alzheimer's Disease, Parkinson's disease Chronic Fatigue Syndrome, Schizophrenia. Australia's medical treatment is orientated around symptom management not identification and treatment of infection.

2.0 AUTISM & BORRELIA INFECTION

2.1 Incidence of Autism

Autism Spectrum Disorder (ASD) in Australia is increasing rapidly (Figure 2), and comprises approximately 2% of the population and represents 1 in 49 school aged children between 5 and 15 years of age (Table 1). The number of reported cases of severe and profound autism is also rapidly increasing (Figure 3) so it cannot be argued that the increase is due to over diagnosis as profound autism is just as easy to diagnose today as it was 10 year ago. The rise in autism follows that seen in the United States, where in 2014 the autism diagnosis rate in USA was 2.24% or 1 in 45 (National Health Statistic Report No 37 Nov 13, 2015). Incidence increase is not dissimilar to that seen in epidemic.

Table 1 – Autism Prevalence Rates determined from Carer Allowance (child) payment recipients 2015.

Child Age	Number Children with ASD Diagnosis	Australian Population	Prevalence (% of Population)		Prevalence (1 in x)	
0 - 4	5 748	1 538 952	0.37%	Note	268	Note
5 - 9	29 027	1 522 192	1.91%		52	
10 - 14	31 840	1 415 903	2.25%	2.06%	44	49
15	5 569	287 190	1.94%		52	
Total Age 5 to 15	**66 436**	**3 225 285**				

Note: due to difficulties associated with assessment and diagnosis of children below the age of 4, the data is for this age bracket is unreliable and has been excluded from calculations.

Figure 1 Carer Allowance for ASD Australian Children

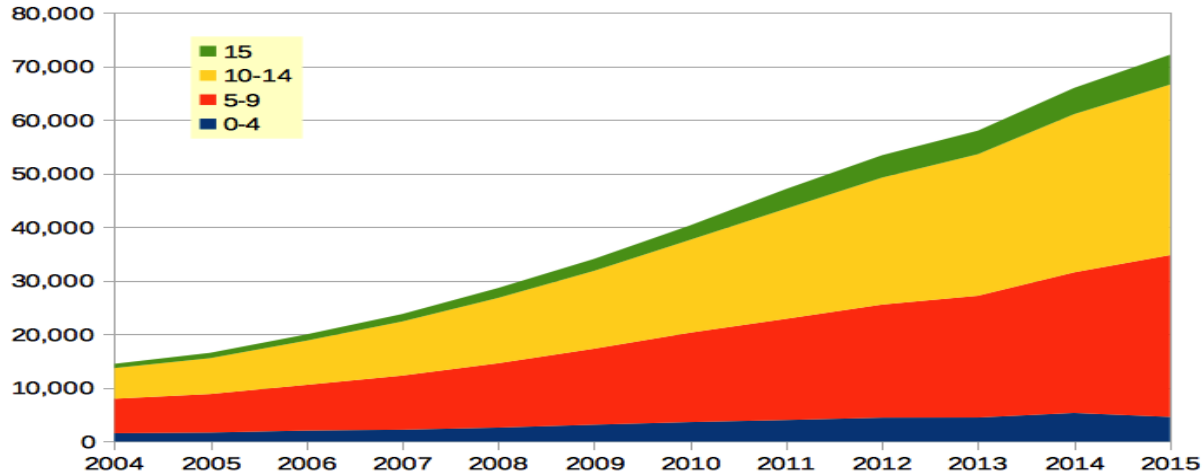

Figure 2 Autism by Type

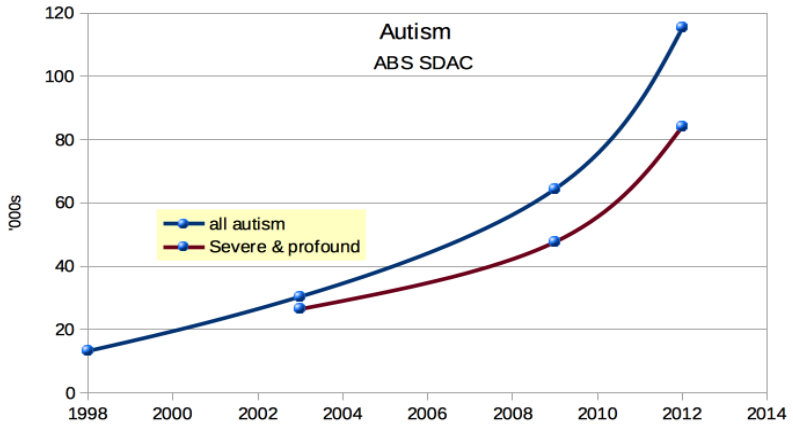

Table and Graphs produced by Autism Aspergers Advocacy Australia (http://www.a4.org.au/prevalence2015)

2.2 The Autistic Condition

Autism Spectrum Disorder (ASD) is diagnosed under the Diagnostic and Statistical Manual of Mental Disorders. The diagnosis is one of brain difference, made on the basis of neuropsychological presentation and does not take into account the physical conditions associated with autism or explain the underlying etiology of Autism which presents as significant immune system dysfunction and nervous system abnormality.

The presentation of Autism involves nutritional deficiencies, environmental toxins, chronic infections, autoimmune immunological responses, allergies, gastrointestinal tract problems, changes in neurotransmitter concentrations and biochemical changes related to oxidative stress. Autistic children have a higher incidence of health issues such as tuberous sclerosis, epilepsy, ear infections and many other physical conditions than their developmentally normal peers. They also develop a higher incidence of additional psychiatric diagnosis. Mothers of those with autism present with increased incidence of autoimmunity and related conditions.

Autism is a condition arising from interplay between genetic and environmental factors. Environmental factors are highlighted in yellow in **Figure 3** and relate to toxins, stress, maternal immune response (including autoimmune response), and maternal infection. Environment also modifies genetic expression (relationship is not depicted in Figure 3).

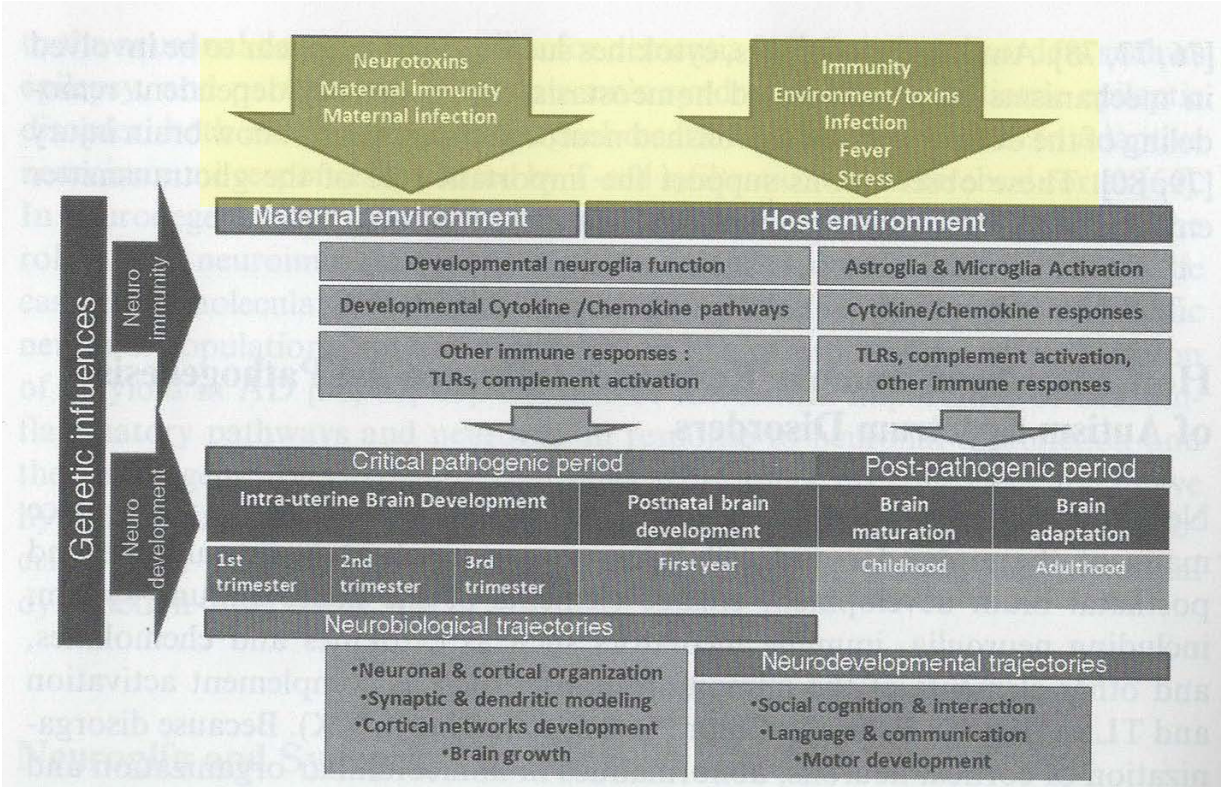

Fig. 15.3 Mechanisms involved in the pathogenesis of autism spectrum disorders (ASD) likely affect brain development during the critical pathogenic period of intrauterine life and the first postnatal year. Genetic, as well as maternal and environmental, factors likely influence neuroimmune pathways and eventually the normal progression of neurobiological trajectories that determine neuronal and cortical organization, synaptic modeling, cortical networking, and brain growth. The aberrant development of neurobiological trajectories modifies neurodevelopmental trajectories of social interaction, language, and motor development, which trigger the onset of stereotypic behaviors that characterize the clinical profile of ASD. Neuroimmune activation may persist during the postpathogenic period and continue to affect brain maturation and adaptation in children and adults with ASD (*see* Color Plate 5)

Figure 3 Mechanisms in Development of ASD

Extract from Page 338 of the medical text “Autism Current Theories and Evidence, edited by A. Zimmerman”, Part IV (Immunology, Maternal – Fetal Interaction, and Neuroinflammation), 2008, Humana Press

2.3 Maternal Infection

The developmental outcome of an unborn child is highly dependent on maternal health and maternal response to infection. Two examples illustrating this connection are provided below.

Research earlier this year uncovered the physical link between maternal infection and the development of autism. When infected, the body's T helper (Th) cells release cytokines that amplify the immune response when necessary and help suppress or regulate it to prevent over activity. A study found that a cytokine named Interleukin 17a (IL-17a) when present in pregnant mice, resulted in the development of autism like traits (behavioral abnormalities and brain cortical changes) in the mice pups[1]. This groundbreaking research is presented in Example 1. IL-17a is an immune response typically associated with viral infection, and chronic Lyme borreliosis.

Example 1

The human cytokine Interleukin 17a (IL-17a) is an immune response present in late state Lyme borreliosis, especially neuroborreliosis and Lyme arthritis[2, 3].

Dr. Dan Littman and his team of researchers have identified a subsection of immune cells that appear to cause certain behaviors linked to autism. They found that the cytokine Interleukin 17A (IL-17a) which is produced by the T helper cells called Th17 during infection, caused behavioral abnormalities showing distinct autism – like traits and triggered cortical changes in mouse pup's brains (evident on brain dissection) . Certain sections of the cortex, responsible for making sense of sights and sounds, were chaotically ordered. These types of cortical disorganization have previously been found in other autism models[1].

The team then trialed two distinct methods of blocking the effects of IL-17a in the pregnant mice; one used anti-IL17a antibodies, and the other blocked the receptor responsible for the maturation of T cells and their consequent production of IL-17a. Whichever method the team used to block IL-17a production, the resulting mice pups were now behaviorally normal. Blocking production of IL-17a in the mother resulted in normal mouse pups [1].

IL-17 is also heavily associated with other chronic infections (Klebsiella pneumonia Bacteroides fragilis, Mycobacterium turberculosis, and fungal species) and human autoimmune diseases such as such as multiple sclerosis rheumatoid arthritis, and inflammatory bowel disease[5].

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1. Choi GB, Yim YS, Wong H, Kim S, Kim H, Kim SV, Hoeffer CA, Littman DR, Huh JR. Choi GB, Yim YS, Wong H, The maternal interleukin-17a pathway in mice promotes autism-like phenotypes in offspring. Science. 2016;351(6276):933-9.

2. Bachmann M, Horn K, Rudloff I, Goren I, Holdener M, Christen U, et al. (2010) Early Production of IL-22 but Not IL-17 by Peripheral Blood Mononuclear Cells Exposed to live *Borrelia burgdorferi*: The Role of Monocytes and Interleukin-1. PLoS Pathog 6(10): e1001144. doi:10.1371/journal.ppat.1001144

3. Oosting M, Van de veerdonk FL, Kanneganti TD, et al. Borrelia species induce inflammasome activation and IL-17 production through a caspase-1-dependent mechanism. Eur J Immunol. 2011;41(1):172-81

4. Sambor Grygorczuk, Joanna Osada, Renata Świerzbińska, Anna Moniuszko, Joanna Zajkowska, Maciej Kondrusik, Piotr Czupryna, Justyna Dunaj, Milena Dąbrowska, Sławomir Pancewicz, "Synthesis of Th17 cytokines by peripheral blood mononuclear cells stimulated with Borrelia burgdorferi sensu lato.", ESCMID Online Poster, contact Sambor Grygorczuk PhD Department of Infectious Diseases and Neuroinfections Medical University in Bialystock Poland . 2016. P0158.pdf

5. Quesniaux V. Ryffel B., Padava,F. , 2009. "*Th 17 Cells: Role in Inflammation and Autoimmune Disease"* Berlin, Springer Science + Business Media, p 21

Example 2

A 2015 study of the Swedish nationwide register-based birth cohort born 1984-2007, comprising of 2,371,403 persons with 24,414 ASD cases revealed an there was approximately a 30% increase in ASD risk associated with any (hospital) in patient diagnosis of infection during pregnancy[1]. Timing of infection did not appear to influence risk in the total Swedish population, since elevated risk of ASD was associated with infection in all trimesters. In a subsample analysis, infections were associated with greater risk of ASD with intellectual disability than for ASD without intellectual disability. If this 30% increase in ASD incidence was applied to Australia, it would represent a 1 in 37 chance of having a child with Autism if the Mother had an inpatient diagnosis of infection during pregnancy. This study adds to the growing body of evidence, encompassing both animal and human studies, that supports immune-mediated mechanisms underlying the etiology of ASD[1].

Note that viral infection also causes IL-17a release.

Attachment 1 provides a selection of 26 papers regarding Maternal Infection and the development of Autism. **Figure 4** depicts the complex arrangements between the central nervous system and immune system responsible for the development of Autism. Infection is highlighted yellow and is a central requirement to the development of most forms of Autism. It should be noted that Autism can be due solely to genetic (eg Rett syndrome, Fragile X) or other environmental factors (toxic exposure).

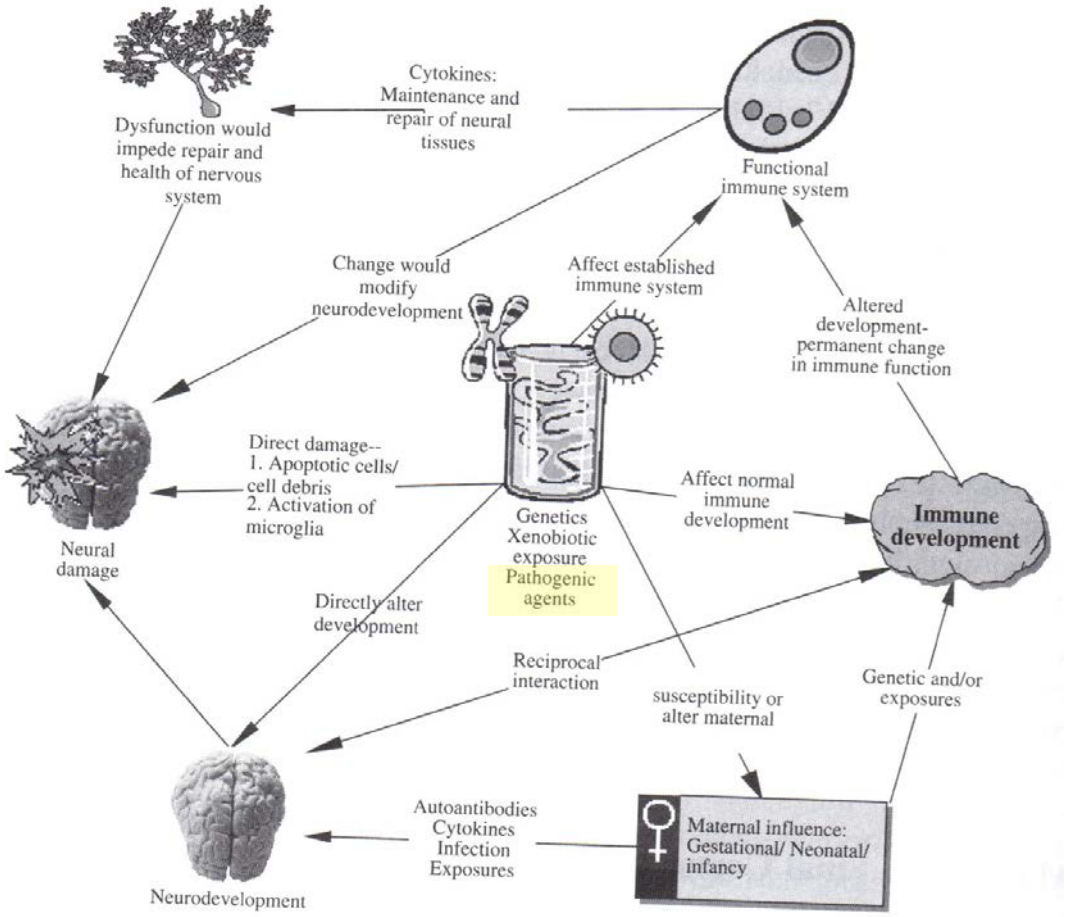

Figure 4 Schematic of Potential interactions between immune and nervous system.
Extract from Page 282 of the medical text "Autism Current Theories and Evidence, edited by A. Zimmerman", Part IV (Immunology, Maternal – Fetal Interaction, and Neuroinflammation), 2008, Humana Press

1. Lee BK, Magnusson C, Gardner RM, Blomström Å, Newschaffer CJ, Burstyn I, Karlsson H, Dalman C," Maternal hospitalization with infection during pregnancy and risk of autism spectrum disorders." Brain Behav Immun. 2015 Feb;44:100-5. doi: 10.1016/j.bbi.2014.09.001.

2.4 Maternal Autoimmunity

Maternal autoimmunity, particularly the production of anti-brain and anti-thyroid autoantibodies result in significant impact to the developing fetal brain and contribute substantially to the incidence of Autism.

In 2015, a study on virtually the entire pregnant population of Finland from 1987 to 2005, provided the world first biomarker based evidence that that a class of known maternal autoimmune disorders is related to autism in offspring[1]. The prevalence of maternal thyroid peroxidase antibody (TPO-Ab+) was significantly increased in pregnancies giving rise to autism cases (6.15%) compared to controls (3.54%). The odds of autism were increased by nearly 80% among offspring of mothers who were thyroid peroxidase antibody postive during pregnancy, compared to mothers negative for this autoantibody. Measures of maternal thyroid hormones did not differ between groups.

If this study finding were applied across to Australia, an 80% increase in the odds of autism equates to a 1 in 27 chance of having a child with Autism if the expectant mother is thyroid peroxidase antibody positive.

Thyroid peroxidase antibody (TPO-Ab+) is a Medicare covered test routinely administered in Australia which could be used to screen women prior to pregnancy. This is just one simple example of what Australia could be doing to kerb the increase in Autism and identify infection.

Attachment 2 provides a selection of 21 papers researching the relationships between Maternal Autoimmunity and the development of Autism.

Figure 5 presents a simplified summary of maternal infection during pregnancy.

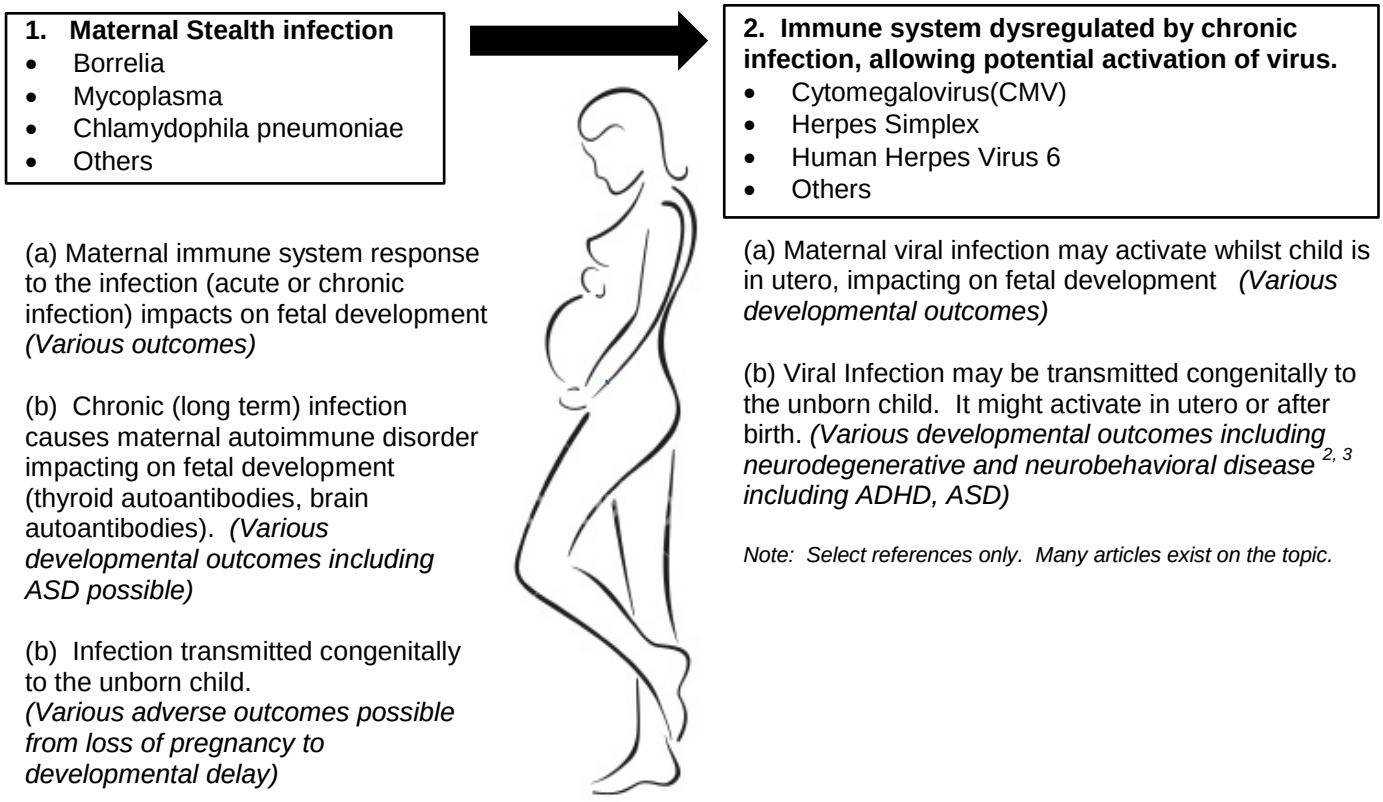

Figure 5 – Simplified Explanation of Maternal Infection influencing pregnancy outcome.

1. Brown AS, Surcel HM, Hinkka-Yli-Salomäki S, Cheslack-Postava K, Bao Y, Sourander A. "Maternal thyroid autoantibody and elevated risk of autism in a national birth cohort.", Prog Neuropsychopharmacol Biol Psychiatry. 2015 Mar 3;57:86-92.

2. Karim S, Mirza Z, Kamal MA, Abuzenadah AM, Azhar EI, Al-Qahtani MH, Damanhouri GA, Ahmad F, Gan SH, Sohrab SS1."The role of viruses in neurodegenerative and neurobehavioral diseases." CNS Neurol Disord Drug Targets. 2014;13(7):1213-23.

3. Garth L. Nicolson, Chronic Bacterial and Viral Infections in Neurodegenerative and Neurobehavioral Diseases, Lab Med. 2008;39(5):291-299

2.5 Autoimmunity due to Chronic Infection

As outlined in the previous sections, maternal infection and autoimmunity contribute significantly to the development of Autism. Heavily implicated in the development of Autism are maternal autoantibodies against the thyroid and against the brain. There is no controversy surrounding the autoimmune aspect of chronic borrelia infections, as such, a lengthy explanation will not be provided in this submission.

Infections such as chronic borreliosis have been established to cause autoimmunity or activate existing autoimmunity. There is a good deal of research regarding antibodies arising from borrelia infection. **Attachment 3** lists 10 papers describing Borrelia Infection causing Autoimmunity with some extracts included. There are several mechanisms by which infection can cause autoimmunity and these interactions are quite complex.

Example 3:

> Chronic borrelia infection has been proven to cause thyroid auto antibodies to be produced. It does this by molecular mimicry. (Molecular mimicry is bacterial cells or other microbial "triggers" with a similar appearance to the cells that make up parts of our physiology or "self" antigens.) In predisposed subjects, a microbial antigen could trigger autoimmunity because of its structural similarity to an autoantigen of the host.

Example 4:

> Borrelia burgdorferi, has been found to have 16 protein structures that can cross react with thyroid proteins (5 proteins that cross react with the thyroid-stimulating hormone (TSH) receptor antibodies, 2 that cross react with thyroglobulin antibodies, and 3 that cross react with thyroid peroxidase antibodies (TPO antibodies), and 6 with the sodium iodide symporter).

What is important to note, is that autoimmunity to both the thyroid and the brain which have been proven to contribute to the development of autism can be caused by borrelia infection.

2.6 Congenital Borrelia Infection

Borrelia is able to be congenitally transmitted (mother to baby in utero) and much research exists on the subject. **Attachment 4** provides a selection of 37 papers (some summarised) in relation to congenital borreliosis. These papers examine foetal outcomes, including outcomes on the basis of different treatments (antibiotics) administered during pregnancy.

It is necessary to point out, that due to early onset immune dysregulation associated with congenital borrelia infection, children may never present with antibodies against the borrelia infection they carry[1]. Detection of stealth infections is problematic in children presenting with congenitally acquired infection and many of the diagnostic tests rely on locating the organism by means of detecting the body's immune response (indirect testing), which may not be present. Furthermore, identification of stealth infection such as borrelia in even a developmentally normal person born with functional immune system is notoriously problematic. Technical studies may fail to consider this and the results of studies debunking Autism borrelia utilising indirect testing techniques should be objectively reviewed.

As an example of the unreliability of indirect testing, refer to Section 3.0 of this submission presenting results from the 28 pathology tests (both direct and indirect) used to identify the chronic borreliosis infection in a family of four where two children contracted the infection congenitally . Direct testing by DNA identification of borrelia would be preferred to indirect testing in diagnosing congenital infection. Those NATA/RCPA accredited Pathology laboratories employed to undertake the Medicare "Lyme" testing provide indirect testing.

2.7 Borrelia (And Other) Infections Contributing To Autism

The method by which late stage untreated maternal borrelia infections have been discussed in the previous sections. Particular attention is drawn to Example 1 in Section 2.2 Example 1 which establishes the mechanism by which maternal infection and subsequent production IL-17a results in the development of autistic like condition in a mouse model. IL-17a present in long term (late stage) borrelia infections.

The finding of increased IL-17a occurs in the Autistic population and correlates with the severity of autism. A study has found that children with autism have significantly higher serum IL-17A levels than children without autism and those children with severe autism had significantly higher serum IL-17A levels than those with mild to moderate autism[2].

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1. M Kuhn, R Bransfield, 2014, Divergent opinions of proper Lyme disease diagnosis and implications for children co-morbid with autism spectrum disorder, Medical hypotheses, September 2014, Vol. 83(3), pp. 321-5.

2. AL-Ayadhi and Mostafa,"Elevated serum levels of interleukin-17A in children with autism" Journal of Neuroinflammation 2012, 9:158

Attachment 5 provides a selection of 32 papers in relation to Borrelia infection and Autism

The issue of Autism borrelia was raised on the 18th of September 2015 at a round table meeting in the House of Representatives regarding Lyme (Borrelia) infection in Australia. During this meeting, Dr Richard Schloeffel, a prominent Australian doctor spoke, an extract from the Hansard follows:

> *"This is never about the money; it is about treating people who are chronically ill and, if you do not treat them adequately, some will be disabled for their whole life at enormous cost to the community and some will die. Some of these people are kids.*
>
> *We have 1,000 children in my practice with autism spectrum disorder. I am doing tests with my colleague who is a paediatrician and some of the research coming out of the states shows that 40 per cent of children with autism have borreliosis or co-infections. That may be the case if translated here.*
>
> *Autism used to be an uncommon disease; now it is one in 100 in Australia. In America it is one in 50; it used to be three in 1,000. So, what is happening to our children?*
>
> *I asked the paediatrician to do IGeneX tests and, low and behold, what did we find? These kids come up positive for Borrelia. We treat them, and I guide him in treating under five-year-olds and they get better and they stop being autistic."*

This issue is worthy of government attention, but cannot be addressed without accurate pathology and good policy surrounding borrelia. In 2013 the Australian Government invested $31 million over 8 years to establish the new 'Cooperative Research Centre (CRC) for Living with Autism Spectrum Disorders'. This funding focuses on the precision and reliability of diagnosis, early intervention and learning needs and integration into society. The Senate Inquiry may consider recommending additional funding, or redirecting funding, to develop and implement screening tools to stem the rapid increase in autism.

Professor Nicholson is President and Founder, Chief Scientific Officer and Emeritus Professor at the Institute for Molecular Medicine., Conjoint Professor in the Faculty of Science and Technology, University of Newcastle, Australia. Dr Nicholson studies chronic intracellular infections and has undertaken extensive research on Mycoplasma bacteria, in particular Mycoplasma fermentans which is the organism responsible for Gulf War Syndrome. 80% of the children born subsequently to the infected gulf war veterans are autistic. Professor Nicholson has identified a variety of infections present in common chronic conditions as listed in Table 2.

Table 2 – Condition and Infection Types commonly observed

Condition	Infections Commonly Observed, % of Cases
Autism Spectrum Disorders	Mycoplasma species (40-65%) Borrelia burgdorferi (10-30%) Chlamydia species (5-10%) Human Herpes Virus 6 (HHV-6) (15-30%) Cytomegalovirus (CMV) (5-10%) Fungal Infections (5-20%)
Amyotrophic Lateral Sclerosis	Borrelia, Mycoplasma species, Chlamydia pneumoniae, HHV6
Alzheimer's Disease	Borrelia, Chlamydia pneumoniae, HSV1 and other Herpes viruses
Multiple Sclerosis	Borrelia, Mycoplasma species, Chlamydia pneumoniae,HHV6 and other Herpes Virus
Parkinson's Disease	Helicobacer pylori, Coronavirus, Mycoplasma species
Chronic Fatigue Syndrome	Borrelia, Mycoplasma species, Chlamydia pneumoniae

Dr Robert Bransfield has also undertaken much research in relation to tick borne infections including borrelia and the associated development of Autism. He has identified those infections listed in table 2 above. A summary of his work as pertinent to Borrelia infection and Autism as found at the following webpage: http://beyondthebandaid.com.au/wp-content/uploads/2012/07/Lyme-Tick-Borne-Other-Chronic-Infections-Contributing-to-Autism-Spectrum-Disorders.pdf This work includes referenced technical explanation as to the Brain Imaging and Biochemical similarities between the presentations of Autism and ASD, Testing of Autism Spectrum Disorder Patients for Tick Borne Illness and similarities between tick borne illness / borrelia infection and autism including similarities between treatments.

2.7 Government Position Regarding Borrelia and Autism

Exerpt from letter received from the Offices of the Federal Minister for Health and the Federal Minister for Sport, the Hon. Sussan Ley MP, dated 29/09/2015 (Attachment 6)

For example, the children of the [redacted] family have been diagnosed with disorders on the autism spectrum. This is not caused by an infectious disease and specialist paediatric care is required. My Department is not aware of any verifiable evidence that classical Lyme disease is associated with autism in children. Additionally, there remains no verifiable repeatable evidence for Lyme disease transmission sexually or vertically from mother to child.

.

This Government position is incorrect and considerable evidence exists to the contrary:

In relation to Autism "not" being caused by an infectious disease:

Refer to Section 2.2. of this submission. Autism is caused by a number of environmental and genetic factors, one of which is infection. Infection is particularly important in the development of autism. The cytokine interlukin 17a in maternal infection results in autism like traits in offspring. Interlukin 17a is the immune system response to many viral and bacterial infections. Some of the infections known to produce elevated interlukin 17a are considered infectious diseases eg borrelia. Maternal infection has been reliably proven to result in higher incidence of autism (up to 30% increase) - Refer to Section 2.3 of this submission. A**ttachment 1** provides a selection of **26** papers providing research in relation to Maternal Infection and Autism.

Maternal autoimmunity (particularly thyroid and brain antibodies) has been extensively studied and there is no scientific debate that these antibodies contribute significantly to the development of autism. Refer to Section 2.4 of this submission. **Attachment 2** provides a selection of **21** papers providing research in relation to Maternal Autoimmunity and Autism. Autoimmune conditions are caused by infection and associated host immune response as driven in part by genetic factors. **Attachment 3** contains **10** papers discussing infection of Borrelia and the resultant autoimmunity, in particular thyroid and brain autoimmunity.

In relation to "no verifiable repeatable evidence" for Lyme disease transmission from mother to child

There is considerable evidence spanning 30 years demonstrating clear and inarguable congenital transmission of borrelia. Studies include foetal outcomes of maternal borrelia infection, including pregnancy outcomes associated with different maternal treatments for borrelia infection. **Attachment 4** contains **37** papers, some of which are summarised, discussing maternal transmission of borrelia to the developing child.

It is short sighted for the government policy to limit the position on borrelia autism to borrelia burgdorferi sensu stricto "classical Lyme". Classical Lyme Disease represents just one of the 39/40 species of borrelia known to infect humans, and other species of borrelia other than burgdorferi have been identified in maternal transmission and these studies are listed in Attachment 4.

In relation to "no verifiable evidence that classical Lyme disease is associated with Autism in Children"

As provided in this submission there is considerable evidence proving that lyme disease is associated with Autism and is causal in autism. **Attachment 5** contains **32** papers on this topic. Furthermore, borrelia causes elevated interlukin17a and this is observed in the autistic population (the level of interlukin 17a directly influencing the severity of the autism) Maternal interlukin17a has been shown to cause autism traits in offspring. A feature of chronic Lyme infection is elevated interlukin17a. Attachments 1 through to 4 provide a vast amount of information in support that Lyme disease does cause Autism.

In the context of the large amount of information that exists in relation to maternal infection, autoimmunity, borrelia and autism, the Government position of requiring "verifiable repeatable evidence" in unclear, and this is an issue which the senate should seek clarification on. Likewise, clarification should be sought on the source of Government advice which is clearly not in line with medical science.

Maternal infection of borrelia can and does have devastating impact on the development of children and can and does contribute significantly to Autism. A national notification system for the infections contributing to Autism including Borrelia, and accurate record keeping on the number of persons with Autism in Australia are essential in addressing and managing the increase of Autism.

Attachment 1 **Research List**
Maternal Infection and Autism

Numerous papers exist in relation to this topic, a selection only is provided:

1. DeLong GR, Bean SC, Brown FR 3rd. Acquired reversible autistic syndrome in acute encephalopathic illness in children. Arch Neurol. 1981;38(3):191-4.

2. Markowitz PI. Autism in a Child with Congenital Cytomegalovirus Infection. J Autism Dev Disord. 1983;13(3)

3. Stubbs EG, Ash E, Williams CPS. Autism and Congenital Cytomegalovirus. J Autism Dev Disord. 1984;14(2).

4. Baron-Cohen S, Ring HA, Bullmore ET, Wheelwright S, Ashwin C, Williams SC. The amygdala theory of autism. Neurosci Biobehav Rev. 2000;24(3):355-64.

5. Singh VK and Jensen RL. Elevated levels of measles antibodies in children with autism. Pediatric Neurol. 2003;28:292-294.

6. Yamashita Y, Fujimoto C, Nakajima E, et al. Possible association between congenital cytomegalovirus infection and autistic disorder. *J Autism Dev Disord*. 2003;33:355-59.

7. De Tiege X, De Laet C, Mazoin N, et. al. Postinfectious immune-mediated encephalitis after pediatric herpes simplex encephalitis. Brain Dev. 2005;27(4):304-7

8. Libbey JE, Sweeten TL, McMahon WM, Fujinami RS. Autistic disorder and viral infections. J Neurovirol. 2005;11(1):1-10

9. Nicolson GL, Berns P, Gan R, et al. Chronic mycoplasmal infections in Gulf War veterans' children and autism patients. *Med Veritas*. 2005;2:383-87.

10. Ashwood P, Wills S, Van de water J. The immune response in autism: a new frontier for autism research. J Leukoc Biol. 2006;80(1):1-15.

11. Harry GJ, Cindy L, Brunssen SH. Maternal infection and white matter toxicity Neurotoxicology. 2006 Sep;27(5):658-70

12. Mankoski RE, Collins M, Ndosi NK, Mgalla EH, Sarwatt VV, Folstein SE. Etiologies of autism in a case-series from Tanzania. J Autism Dev Disord. 2006;36(8):1039-51

13. Nicolson GL, Gan R, Nicolson NL, et al. Evidence for Mycoplasma, Chlamydia pneunomiae and HHV-6 Co-infections in the blood of patients with Autism Spectrum Disorders. *J Neuroscience Res.* 2007;85:1143-48.

14. Bransfield RC, Wulfman JS, Harvey WT, Usman AI. The association between tick-borne infections, Lyme borreliosis and autism spectrum disorders Medical Hypotheses. 2007

15. Rosen NJ, Yoshida CK, Croen LA. Infection in the first 2 years of life and autism spectrum disorders. Pediatrics. 2007;119:61-9.

16. Lancaster K, Dietz DM, Moran TH, Pletnikov MV.Abnormal social behaviors in young and adult rats neonatally infected with Borna disease virus. Behav Brain Res. 2007;176(1):141-8.

17. Boorom KF. Is this recently characterized gastrointestinal pathogen responsible for rising rates of inflammatory bowel disease (IBD) and IBD associated autism in Europe and the United States in the 1990s? Med Hypotheses. 2007;69(3):652-9.

18. Nicolson GL. Chronic Bacterial and Viral Infections in Neurodegenerative and Neurobehavioral Diseases. Laboratory Medicine 2008. BJMP 2009:2(4) 20-28

19. Quesniaux V. Ryffel B., Padava,F. , 2009. "*Th 17 Cells: Role in Inflammation and Autoimmune Disease"* Berlin, Springer Science + Business Media, p 21
    "In mice, IL-17 is produced during certain infections (such as with Klebsiella pneumonia Bacteroides fragilis, Borrelia burgdorferi, Mycobacterium turberculosis, and fungal species) and during chronic tissue inflammation

(such as experimental autoimmune encephalomyelitis (EAE) and collagen-induced arthritis). In line with these observations in murine models, IL-17 expression has been detected in the target tissue during the progression of various human autoimmune diseases such as multiple sclerosis rheumatoid arthritis, and inflammatory bowel disease"

20. Oosting M, Van de veerdonk FL, Kanneganti TD, et al. Borrelia species induce inflammasome activation and IL-17 production through a caspase-1-dependent mechanism. Eur J Immunol. 2011;41(1):172-81.
Borrelia burgdorferi spirochetes cause Lyme disease, which can result in severe clinical symptoms such as multiple joint inflammation and neurological disorders. IFN-γ and IL-17 have been suggested to play an important role in the host defense against Borrelia, and in the immunopathology of Lyme disease. The caspase-1-dependent cytokine IL-1β has been linked to the generation of IL-17-producing T cells, whereas caspase-1-mediated IL-18 is crucial for IFN-γ production. In this study, we show by using knockout mice the role of inflammasome-activated caspase-1 in the regulation of cytokine responses by B. burgdorferi. Caspase-1-deficient cells showed significantly less IFN-γ and IL-17 production after Borrelia stimulation. A lack of IL-1β was responsible for the defective IL-17 production, whereas IL-18 was crucial for the IFN-γ production. Caspase-1-dependent IL-33 played no role in the Borrelia-induced production of IL-1β, IFN-γ or IL-17. In conclusion, we describe for the first time the role of the inflammasome-dependent caspase-1 activation of cytokines in the regulation of IL-17 production induced by Borrelia spp. As IL-17 has been implicated in the pathogenesis of chronic Lyme disease, these data suggest that caspase-1 targeting may represent a new immunomodulatory strategy for the treatment of complications of late stage Lyme disease.

21. Ousseny Zerbo, Yinge Qian, Cathleen Yoshida, ,Judith K. Grether, Judy Van de Water, Lisa A. Croen. "Maternal Infection During Pregnancy and Autism Spectrum Disorders" 2013, J Autism Dev Disord, DOI 10.1007/s10803-013-2016-3

22. Strickland AD, "Prevention of cerebral palsy, autism spectrum disorder, and attention deficit-hyperactivity disorder.", Med Hypotheses. 2014 May;82(5):522-8. doi: 10.1016/j.mehy.2014.02.003. Epub 2014 Feb 12.

23. Lee BK, Magnusson C, Gardner RM, Blomström Å, Newschaffer CJ, Burstyn I, Karlsson H, Dalman C," Maternal hospitalization with infection during pregnancy and risk of autism spectrum disorders." Brain Behav Immun. 2015 Feb;44:100-5. doi: 10.1016/j.bbi.2014.09.001.

24. Engman M., Sundin M., Miniscalco C., Westerlund J., Lewensohn-Fuchs I, Christopher Gillberg C.,, Fernell E.,Prenatal acquired cytomegalovirus infection should be considered in children with autism", Acta Paediatrica, Volume 104, Issue 8, pages 792–795, August 2015

25. Choi GB, Yim YS, Wong H, Kim S, Kim H, Kim SV, Hoeffer CA, Littman DR, Huh JR. Choi GB, Yim YS, Wong H, The maternal interleukin-17a pathway in mice promotes autism-like phenotypes in offspring. Science. 2016;351(6276):933-9.
Viral infection during pregnancy has been correlated with increased frequency of autism spectrum disorder (ASD) in offspring. This observation has been modeled in rodents subjected to maternal immune activation (MIA). The immune cell populations critical in the MIA model have not been identified. Using both genetic mutants and blocking antibodies in mice, we show that retinoic acid receptor-related orphan nuclear receptor gamma t (RORγt)-dependent effector T lymphocytes [for example, T helper 17 (TH17) cells] and the effector cytokine interleukin-17a (IL-17a) are required in mothers for MIA-induced behavioral abnormalities in offspring. We find that MIA induces an abnormal cortical phenotype, which is also dependent on maternal IL-17a, in the fetal brain. Our data suggest that therapeutic targeting of TH17 cells in susceptible pregnant mothers may reduce the likelihood of bearing children with inflammation-induced ASD-like phenotypes.

26. Sambor Grygorczuk, Joanna Osada, Renata Świerzbińska, Anna Moniuszko, Joanna Zajkowska, Maciej Kondrusik, Piotr Czupryna, Justyna Dunaj, Milena Dąbrowska, Sławomir Pancewicz, "Synthesis of Th17 cytokines by peripheral blood mononuclear cells stimulated with Borrelia burgdorferi sensu lato.", ESCMID Online Poster, contact Sambor Grygorczuk PhD Department of Infectious Diseases and Neuroinfections Medical University in Bialystock Poland . 2016. P0158.pdf
Lymphocytes of the Th17 subset and their characteristic cytokines, interleukin 17A (IL-17A), IL-17F and IL-22, play important role both in the response to extracellular bacteria and in the autoimmunity, including rheumatoid
arthritis and other immune-mediated forms of arthritis. IL-17A has been proven to be involved in the response against Borrelia burgdorferi sensu lato (B. burgdorferi sl), and, together with related cytokines, could contribute to the protracted and deleterious inflammation in the late stage of Lyme borreliosis, especially in neuroborreliosis and Lyme arthritis…….Median concentrations of IL-17A, IL-17F and IL-22 increased significantly in the presence of B. burgdorferi sl, with no difference between controls and Lyme borreliosis patients. The concentration of IL-17A was higher under stimulation with B. afzelii compared to B. burgdorferi ss andB. garinii. IL-17F and IL-22 concentrations did not depend on the B. burgdorferi sl species.

Attachment 2 **Research List**
Maternal Autoimmunity and Autism

A selection of papers on Maternal Autoimmunity and Autism. Numerous other papers on this topic exist.

1. Connolly AM, Chez MG, Pestronk A, Arnold ST, Mehta S, Deuel RK. Serum autoantibodies to brain in Landau-Kleffner variant, autism, and other neurologic disorders. J Pediatr. 1999;134(5):607-13.

2. Patterson PH. Maternal infection: window on neuroimmune interactions in fetal brain development and mental illness. Curr Opin Neurobiol. 2002;12(1):115-8.

3. Dalton P, Deacon R, Blamire A, et al. Maternal neuronal antibodies associated with autism and a language disorder. Ann Neurol. 2003;53(4):533-7.

4. Ashwood P, Van de water J. Is autism an autoimmune disease?. Autoimmun Rev. 2004;3(7-8):557-62.
5. Cohly HH, Panja A. Immunological findings in autism. Int Rev Neurobiol. 2005;71:317-41.

6. Vargas DL, Nascimbene C, Krishnan C, Zimmerman AW, Pardo CA. Neuroglial activation and neuroinflammation in the brain of patients with autism. Ann Neurol. 2005;57(1):67-81.[HTML] from nih.gov

7. Smith SE, Li J, Garbett K, Mirnics K, Patterson PH. Maternal immune activation alters fetal brain development through interleukin-6. J Neurosci. 2007;27(40):10695-702.

8. Zimmerman AW, Connors SL, Matteson KJ, et al. Maternal antibrain antibodies in autism. Brain Behav Immun. 2007;21(3):351-7

9. Braunschweig D, Ashwood P, Krakowiak P, et al. Autism: maternally derived antibodies specific for fetal brain proteins. Neurotoxicology. 2008;29(2):226-31.

10. Singer HS, Morris CM, Gause CD, Gillin PK, Crawford S, Zimmerman AW. Antibodies against fetal brain in sera of mothers with autistic children. J Neuroimmunol. 2008;194(1-2):165-72.

11. Croen LA, Braunschweig D, Haapanen L, et al. Maternal mid-pregnancy autoantibodies to fetal brain protein: the early markers for autism study. Biol Psychiatry. 2008;64(7):583-8.

12. Patterson PH. Immune involvement in schizophrenia and autism: etiology, pathology and animal models. Behav Brain Res. 2009;204(2):313-21.

13. Singer HS, Morris C, Gause C, Pollard M, Zimmerman AW, Pletnikov M. Prenatal exposure to antibodies from mothers of children with autism produces neurobehavioral alterations: A pregnant dam mouse model. J Neuroimmunol. 2009;211(1-2):39-48.

14. Morris CM, Zimmerman AW, Singer HS. Childhood serum anti-fetal brain antibodies do not predict autism. Pediatr Neurol. 2009;41(4):288-90.

15. Rout UK, Dhossche DM, “A pathogenetic model of autism involving Purkinje cell loss through anti-GAD antibodies”, Medical Hypotheses (Impact Factor: 1.07). 02/2008; 71(2):218-21

16. Currenti SA, “Understanding and determining the etiology of autism”, Cellular and molecular neurobiology, Cellular and Molecular Neurobiology, March 2010, Volume 30, Issue 2, pp 161-171

17. Goines P, Haapanen L, Boyce R, et al. Autoantibodies to cerebellum in children with autism associate with behavior. Brain Behav Immun. 2011;25(3):514-23.

18. Goines P, Van de water J. The immune system's role in the biology of autism. Curr Opin Neurol. 2010;23(2):111-7.

19. PH Patterson Maternal infection and immune involvement in autism.” Trends Mol Med. 2011 Jul; 17(7): 389–394.

20. Heuer L, Braunschweig D, Ashwood P, Van de Water J, Campbell DB., “Association of a MET genetic variant with autism-associated maternal autoantibodies to fetal brain proteins and cytokine expression.” Transl Psychiatry. 2011 Oct 18;1:e48. doi: 10.1038

21. Braunschweig D, Duncanson P, Boyce R, et al. Behavioral correlates of maternal antibody status among children with autism. J Autism Dev Disord. 2012;42(7):1435-45.

22. Fox E, Amaral D, Van de water J. Maternal and fetal antibrain antibodies in development and disease. Dev Neurobiol. 2012;72(10):1327-34.

23. Onore C, Careaga M, Ashwood P. The role of immune dysfunction in the pathophysiology of autism. Brain Behav Immun. 2012;26(3):383-92.

24. Braunschweig D, Van de water J. Maternal autoantibodies in autism. Arch Neurol. 2012;69(6):693-9.

25. M D Bauman, A-M Iosif, P Ashwood, D Braunschweig, A Lee, C M Schumann, J Van de Water6 and D G Amaral, "Maternal antibodies from mothers of children with autism alter brain growth and social behavior development in the rhesus monkey" Translational Psychiatry (2013) 3, e278; doi:10.1038/tp.2013.47

26. D Braunschweig, P Krakowiak, P Duncanson, R Boyce, R L Hansen, P Ashwood, I Hertz-Picciotto, I N Pessah and J Van de Water, "Autism-specific maternal autoantibodies recognize critical proteins in developing brain", Translational Psychiatry (2013) 3, e277; doi:10.1038/tp.2013.50

27. Brimberg L, Sadiq A, Gregersen PK, Diamond B. Brain-reactive IgG correlates with autoimmunity in mothers of a child with an autism spectrum disorder. Mol Psychiatry. 2013;18(11):1171-7.
A study of >2700 mothers of children with autism has found that the prevalence of anti-brain antibodies is about 4 times greater among mothers of children with autism (10.5%) than among the normal cohort (2.6%). 50% of autism mothers who carry anti-brain antibodies have Anti-nuclear antibodies

28. Piras IS, Haapanen L, Napolioni V, Sacco R, Van de water J, Persico AM.
Anti-brain antibodies are associated with more severe cognitive and behavioral profiles in Italian children with Autism Spectrum Disorder. Brain Behav Immun. 2014;38:91-9.

29. L Brimberg, A Sadiq, P K Gregersen, "B Diamond. Brain-reactive IgG correlates with autoimmunity in mothers of a child with an autism spectrum disorder**.** Molecular Psychiatry", 2013; DOI: 10.1038/mp.2013.101

30. Brown AS, Surcel HM, Hinkka-Yli-Salomäki S, Cheslack-Postava K, Bao Y, Sourander A. "Maternal thyroid autoantibody and elevated risk of autism in a national birth cohort.", Prog Neuropsychopharmacol Biol Psychiatry. 2015 Mar 3;57:86-92.

Attachment 3 **Research List**
Borrelia Infection causing Autoimmunity.

1. Suchanek G, Kristoferitsch W, Stanek G, Bernheimer H, “Anti-myelin antibodies in cerebrospinal fluid and serum of patients with meningopolyneuritis Garin-Bujadoux-Bannwarth and other neurological disease”, Medical Microbiology, Infectious Diseases, Virology, Parasitology,Volume 263 Issues 1-2, December 1986, Page 160-168
   Anti-myelin antibodies (AMA) of IgG, IgM, and IgA class were investigated by ELISA in CSF and serum from patients with meningopolyneuritis Garin-Bujadoux-Bannwarth (GBB), other inflammatory diseases of the nervous system (ID) comprising meningoencephalitis (ME), multiple sclerosis (MS), and Guillain-BarrÃ© syndrome (GBS), and various noninflammatory neurological diseases (NID). Anti-Borrelia antibodies (ABA) were determined by ELISA in GBB patients. In CSF, a high incidence of IgG-AMA, IgM-AMA, and IgA-AMA was found in GBB as compared with ID and NID. On average, positive AMA titers were higher in GBB than in ID and NID, IgM-AMA titers in GBB being most prominent. In serum, AMA were found in all but 2 patients investigated. On average, IgM-AMA titers were higher in GBB and MS than in other diseases; IgG-AMA titers in GBB and ME were relatively low. Antibody indices, calculated from titer values and Ig concentrations in CSF and serum, indicate intrathecal synthesis mainly of IgG- and IgA-AMA, and of IgG- and IgM-ABA. Participation of AMA in the pathogenesis of GBB may be envisaged, but needs further confirmation.

2. Leonard H. Sigal, Arthur H. Tatum, “Lyme disease patients' serum contains IgM antibodies to Borrelia burgdorferi that cross react with neuronal antigens.” Neurology September 1988 vol. 38 no. 9 1439
   Serum from patients with neurologic manifestations of Lyme disease had serum IgM antibodies that bound to normal human axons, whereas binding was absent or weak in patients without neurologic findings. Antiaxonal binding could be eliminated by absorption with Borrelia burgdorferi. A murine monoclonal antibody to the borrelial flagellin also bound to human axons

3. Baig S., Olsson T., Höjeberg B., Link H., “Cells secreting antibodies to myelin basic protein in cerebrospinal fluid of patients with Lyme neuroborreliosis Neurology April 1991 vol. 41 no. 4 581

   An autoimmune response to myelin basic protein (MBP) has been proposed to participate in the development of the chronic neurologic manifestations that may accompany Borrelia burgdorferi-induced Lyme disease. Using an immunospot assay, we counted cells secreting antibodies to MBP. Anti-MBP IgG antibody-secreting cells were detected in CSF from eight of 13 consecutive patients with Lyme neuroborreliosis irrespective of stage of disease..

4. Klempner M, Huber B “ Is it thee or me?—autoimmunity in Lyme disease” *Nature Medicine*, 5, 1346 - 1347 (1999)

5. Singh SK, Girschick HJ. 2004, Lyme borreliosis: from infection to autoimmunity, Clinical Microbiology and Infection, Vol.10 (7), pp. 598-614.
   Lyme borreliosis in humans is an inflammatory disease affecting multiple organ systems, including the nervous system, cardiovascular system, joints and muscles. The causative agent, the spirochaete Borrelia burgdorferi, is transmitted to the host by a tick bite. The pathogenesis of the disease in its early stages is associated largely with the presence of viable bacteria at the site of inflammation, whereas in the later stages of disease, autoimmune features seem to contribute significantly. In addition, it has been suggested that chronic persistence of B. burgdorferi in affected tissues is of pathogenic relevance. Long-term exposure of the host immune system to spirochaetes and/or borrelial compounds may induce chronic autoimmune disease. The study of bacterium-host interactions has revealed a variety of proinflammatory and also immunomodulatory-immunosuppressive features caused by the pathogen. Therapeutic strategies using antibiotics are generally successful, but chronic disease may require immunosuppressive treatment. Effective and safe vaccines using recombinant outer surface protein A have been developed, but have not been propagated because of fears that autoimmunity might be induced. Nevertheless, new insights into the modes of transmission of B. burgdorferi to the warm-blooded host have been generated by studying the action of these vaccines

6. Benvenga S, Guarneri F, Vaccaro M, Santarpia L, and Trimarchi F. “Homologies Between Proteins of *Borrelia burgdorferi* and Thyroid Autoantigens” Thyroid. November 2004, 14(11): 964-966.

7. Rysková O, Vyslouzil L, Honegr K, Lesná J, Horácek J, Skrabková Z. [Lyme borreliosis--incidence of serum anti-myelin antibodies]. Epidemiol Mikrobiol Imunol. 2002;51(2):60-5.
The method of enzyme immunoassay (ELISA) was used for detection of antibodies against the basic protein myelin (antimyelin antibodies) for a group of serum samples (n 36) with positive anti-borrelia immunoglobulins IgG and IgM (ELISA-Borrelia afzelii) and their immune complexes (ELISA-PEG). Antimyelin antibodies (ELISA-Doxa Kit-Myelin Basic Protein Antibodies) were assessed in 31% (n 11) of examined serum samples of patients with the working diagnosis of Lyme borreliosis. Statistical analysis (p 0.07) confirmed a more frequent incidence of antimyelin antibodies in younger female subjects (age 31 years) as compared with a group of sera (n 25) where the authors did not record the formation of immunoglobulins against the basic myelin protein (age 51 years). Neither the value of titres nor the frequency of detected anti-borrelia IgG and IgM and immune complexes did not differ significantly in the two groups. From the assembled results ensues that in the course of Lyme borreliosis, in chronic affection of organs an autoimmune reaction may develop where the basic myelin protein is damaged (demyelinizatio) and subsequently antimyelin antibodies are formed

8. Raveche E, Schutzer, S, Fernandes, H, Bateman, H, McCarthy, B, Nickell, S, Cunningham, MW, 2005, Evidence of Borrelia Autoimmunity-Induced Component of Lyme Carditis and Arthritis, J Clin Microbiol. 2005 Feb; 43(2): 850–856.
Abstract: We investigated the possibility that manifestations of Lyme disease in certain hosts, such as arthritis and carditis, may be autoimmunity mediated due to molecular mimicry between the bacterium Borrelia burgdorferi and self-components. We first compared amino acid sequences of Streptococcus pyogenes M protein, a known inducer of antibodies that are cross-reactive with myosin, and B. burgdorferi and found significant homologies with OspA protein. We found that S. pyogenes M5-specific antibodies and sera from B. burgdorferi-infected mice reacted with both myosin and B. burgdorferi proteins by Western blots and enzyme-linked immunosorbent assay. To investigate the relationship between self-reactivity and the response to B. burgdorferi, NZB mice, models of autoimmunity, were infected. NZB mice infected with B. burgdorferi developed higher degrees of joint swelling and higher anti-B. burgdorferi immunoglobulin M cross-reactive responses than other strains with identical major histocompatibility complex (DBA/2 and BALB/c). These studies reveal immunological cross-reactivity and suggest that B. burgdorferi may share common epitopes which mimic self-proteins. These implications could be important for certain autoimmunity-susceptible individuals or animals who become infected with B. burgdorferi.

9. Benvenga S, Santarpia L, Trimarchi F, Guarneri F.Human. “Thyroid autoantigens and proteins of Yersinia and Borrelia share amino acid sequence homology that includes binding motifs to HLA-DR molecules and T-cell receptor.” Thyroid. 2006 Mar;16(3):225-36.
We previously reported that the spirochete Borrelia burgdorferi could trigger autoimmune thyroid diseases (AITD). Subsequently, we showed local amino acid sequence homology between all human thyroid autoantigens (human thyrotropin receptor [hTSH-R], human thyroglobulin [hTg], human thyroperoxidase [hTPO], human sodium iodide symporter [hNIS]) and Borrelia proteins (n = 6,606), and between hTSH-R and Yersinia enterocolitica (n = 1,153). We have now updated our search of homology with Borrelia (n = 11,198 proteins) and extended our search on Yersinia to the entire species (n = 40,964 proteins)……

10. Lünemann JD, Gelderblom H, Sospedra M, et al. Cerebrospinal fluid-infiltrating CD4+ T cells recognize Borrelia burgdorferi lysine-enriched protein domains and central nervous system autoantigens in early Lyme encephalitis. Infect Immun. 2007;75(1):243-51.

Attachment 4 **Research List**
Maternal Transmission of Borrelia

1980 - 1989

1. Schlesinger PA, Duray PH, Burke BA, Steere AC, Stillman MT. Maternal-fetal transmission of the Lyme Disease spirochete, B. burgdorferi. Ann Intern Med. 1985;**103**(1):67-8.

   Mothers with active Lyme Disease,Treated: 14.6% of the pregnancies with sequelae, Untreated: 66.7% of thepregnancies with sequelae, Unknown as to treatment: 30.3% with sequelae. Specific adverse outcomes included:cardiac 22.7%, neurologic 15.2%, orthopedic 12.1%, opthalmic 4.5%,genitourinary 10.6%, miscellaneous anomalies 12.1%, 2nd trimester demise 12.1%. Highest rate of adverse outcome (72.7%)in women with infection acquired prior to or during first trimester. Pregnant woman developed Lyme disease during her first trimester, but did not receive antibiotic therapy. Her infant, born at 35 weeks gestational age, died of congenital heart disease during the first week of life. Histologic examination of autopsy material showed the Lyme disease spirochete in the spleen, kidneys and bone marrow of the infant.

2. Lampert F. Infantile multisystem inflammatory disease: another case of a new syndrome. Eur J Pediatr. 1986;144(6):593-6.
3. Markowitz LE, Steere AC, Benach JL, Slade JD, Broome CV. Lyme disease during pregnancy. JAMA. 1986;255(24):3394-6.
4. Macdonald, Alan B. "Human fetal borreliosis, toxemia of pregnancy, and fetal death." *Zentralblatt für Bakteriologie, Mikrobiologie und Hygiene. Series A: Medical Microbiology, Infectious Diseases, Virology, Parasitology* 263.1-2 (1986): 189-200.
5. Macdonald AB, Enach JL, Burgdorfer W. Stillbirth following maternal Lyme disease. NY state Med J. 1987;87:615-16

   Woman with untreated Lyme disease in first trimester of pregnancy gave birth at term to a stillborn. Borrelia burgdorferi cultured from the liver and spirochetes seen in heart, adrenal glands, liver, brain and placenta of baby.

6. Lavoie PE, Lattner BP, Duray PH, et al. Culture positive, seronegative, transplacental Lyme borreliosis infant mortality. Arthritis Rheum.1987; **3**:S50.
7. Carlomagno G, Luksa V, Candussi G, et al.(1988) Lyme Borrelia positive serology associated with spontaneous abortion in an endemic Italian area. Acta Eur Fertil 19(5), 279-81
8. Weber K, Bratze H, Neubert U, et al. Borrelia burgdorferi in a newborn despite oral penicillin for Lyme boreliiosis during pregnancy. Paediatric Infect Dis J. 1988 Apr;7(4):286-9.

   Woman received penicillin orally for one week for an erythema migrans rash during the first trimester of pregnancy and delivered infant at term who died 23 hours later of what was believed to be "perinatal brain damage". Borrelia burgdorferi was identified in the newborn's brain.

9. MacDonald AB. Gestational Lyme borreliosis. Implications for the fetus. Rheum Dis Clin North Am. 1989;**15**(4):657-77.

   Demonstrating transplacental transmission of the spirochete from mother to foetus is possible. Reports that varieity of manifestions of gestational Lyme Borreliosis mimic the diversity of prenatal syphilis. Foetal death, hydrocephalus, cardiovascular anomalities, neonatal repiratory distress, hyperbilirubinemia, intrauterine growth retardation, cortical blindness, sudden infant death syndrome and maternal toxaemia of pregnancy.

10. Nadal D, Hunziker UA, Bucher HU, Hitzig WH, Duc G. Infants born to mothers with antibodies against Borrelia burgdorferi at delivery. Eur J Pediatr. 1989;148(5):426-7.

1990 - 1999

11. Rahn, Daniel W., and Stephen E. Malawista. "Lyme disease: recommendations for diagnosis and treatment." *Annals of internal medicine* 114.6 (1991): 472-481.
12. Schutzer SE, Janniger CK, Schwartz RA (1991) Lyme disease during pregnancy. Cutis 47(4), 267-8.
13. Strobino BA, Williams CL, Abid S, et al. (1993) Lyme disease and pregnancy outcome: a prospective study of two thousand prenatal patients. Am J Obstet Gynecol 169(2 Pt 1), 367-74
14. Jovanovi R, Hajri A, Cirkovi A, et al.(1993) [Lyme disease and pregnancy]. Glas Srp Akad Nauka Med (43), 169-72
15. Hercogova et al. Could borrelia found in the placenta influence the fetus? Study of 19 women with EM during pregnancy. 6th Int Conf Lyme Borreliosis.1994
16. Kumi-Diaka J, Harris O. Br Vet J. Viability of Borrelia burgdorferi in stored semen. 1995 Mar-Apr;151(2):221-4
17. Gardner T. Infectious Diseases of the Fetus and Newborn, 5th edition, (1995) Chapter 11, page 447 – 528Schmidt BL, Aberer E, Stockenhuber C, Klade H, Breier F, Luger A. Detection of Borrelia burgdorferi DNA by polymerase chain reaction in the urine and breast milk of patients with Lyme borreliosis. Diagn Microbiol Infect Dis. 1995;21(3):121-8.
18. Williams, C. L., et al. "Maternal Lyme disease and congenital malformations: a cord blood serosurvey in endemic and control areas." *Paediatric and perinatal epidemiology* 9.3 (1995): 320-330.
19. Silver, Robert M., et al. "Fetal outcome in murine Lyme disease." *Infection and immunity* 63.1 (1995): 66-72.
20. Silver H. (1997) Lyme Disease During Pregnancy. Inf Dis Clinics of N. Amer. Vol 11, No 1, van Holten J, Tiems J, Jongen VH (1997)

21. Van Holten J, Tiens J, Jongen VH. “Neonatal Borrelia duttoni infection: a report of three cases. “ Trop Doct 1997 April;27(2),115-6
22. Strobino, Barbara, Syed Abid, and Michael Gewitz. "Maternal Lyme disease and congenital heart disease: a case-control study in an endemic area." *American journal of obstetrics and gynecology* 180.3 (1999): 711-716.

2000 - 2009

23. Bach G. Sexual Transmission of Lyme Disease. Microbes and Mental Illness Symp; Am Psych Assn Inst Psych Services. 2000
24. Gardner T. Lyme disease. 66 Pregnancies complicates by Lyme Borreliosis. Infec Dis Fetus and Newborn Infant. Saunders, 2000.
25. Elliott, Daniel J., Stephen C. Eppes, and Joel D. Klein. "Teratogen update: Lyme disease." *Teratology* 64.5 (2001): 276-281.
26. Harvey, WT; Salvato, P: ‘Lyme Disease’: Ancient Engine of an Unrecognized Borreliosis Pandemic? Medical Hypotheses (2003) **60**(5), 742-59.
27. Goldenberg, Robert L., and Cortney Thompson. "The infectious origins of stillbirth." *American journal of obstetrics and gynecology* 189.3 (2003): 861-873.
28. Stricker, R.B., D.H. Moore, and E.E. Winger.(2004). Clinical and immunologic evidence of transmission of Lyme disease through intimate human contact. J. Invest. Med. 52, S15
29. Jones CR, Smith H, Gibb E, Johnson L. Gestational Lyme Disease Case studies of 102 Live Births. Lyme Times. 2005;Summer:34-6.
30. Onk G, Acun C, Kalayci M, Cagavi F, et al.(2005) Gestational Lyme disease as a rare cause of congenital hydrocephalus. JTurkish German Gynecology Association Artemis, 6(2), 156-157
31. Goldenberg, Robert L., Jennifer F. Culhane, and Derek C. Johnson. "Maternal infection and adverse fetal and neonatal outcomes." *Clinics in perinatology* 32.3 (2005): 523-559.
32. Jones, CR. Lyme Disease and Autism. LIA Conf;2007
33. Hercogova J, Vanousova D (2008) Syphilis and borreliosis during pregnancy. Dermatol Ther 21(3), 205-9

2010 to present

34. Lakos, András, and Norbert Solymosi. "Maternal Lyme borreliosis and pregnancy outcome." *International Journal of Infectious Diseases* 14.6 (2010): e494-e498.

    This is a retrospective evaluation going back over 22 years being a review of 95 cases of pregnant women with Lyme disease. Antibiotic treatment administered intravenously to 66 women, orally to 19 and infection remained untreated in 10 pregnancies. Adverse outcomes in 8 of 66 (12%) treated women, 6 of 19 (31%) of orally treated women and 6 of 10 (60%) untreated women. Loss of pregnancy and cavernous hemangioma (grossly dilated blood vessels and vascular channels) was the most common adverse outcome. Of the three pregnancies where the mothers had late Lyme borreliosis and in which the infection was acquired long before conception and remained untreated before and during the whole period of pregnancy, none resulted in any fetal or newborn harm within those limits examined by the study. Study did not examine the placenta or fetus for direct Borrelia invasion in the cases of pregnancy loss.

35. Mylonas I (2011) Borreliosis During Pregnancy: A Risk for the Unborn Child? Vector Borne Zoonotic Dis. 11:891-8
36. Jones, CR. ILADS Conf; 2011

    Mothers with Lyme disease who were not treated with antibiotic during pregnancy had a 50% chance of passing Lyme on to their baby. In mothers treated with one antibiotic during pregnancy, that likelihood went down to 25%. In mothers treated with two antibiotics during pregnancy, rate dropped to below 5%.

37. Haseena Hussein, Adrienne Showler, Darrell H.S. Tan. “Tick -borne relapsing fever in pregnancy.” CMAJ. 2014 February 4; 186(2): 131–134.

Attachment 5 **Research List**
Borrelia and Autism

1. Eloi Akintunde, M. Rose, M. Krakowiak, P., Heur, L., Ashwood, P., Hanse,n R., Hertz-Picciotto, I., Van de Water, J., "Increased production of IL-17 in children with autism spectrum disorders and co-morbid asthma" Journal of Neuroimmunology, Sept 2015 Vol 286 pp33-41

2. AL-Ayadhi and Mostafa,"Elevated serum levels of interleukin-17A in children with autism" Journal of Neuroinflammation 2012, 9:158
   Background: The T-helper (Th)1/Th2 dichotomy dominated the field of immune regulation until interleukin (IL)-17-expressing T cells (Th17) were proposed to be a third lineage of helper T cells, the key players in the pathogenesis of autoimmune disorders. Autoimmunity to brain tissue may play a pathogenic role in autism. IL-17A is a pro-inflammatory cytokine that has been shown to play an important role in various autoimmune neuroinflammatory diseases. The aim of this study was to measure serum levels of IL-17A in relation to the degree of the severity of autism. Results: Children with autism had significantly higher serum IL-17A levels than healthy controls (P <0.001), with increased serum levels of IL-17A found in 48.9% of the autism group. Patients with severe autism had significantly higher serum IL-17A levels than those with mild to moderate autism (P = 0.01), and raised serum IL-17A levels were significantly more common in children with severe autism (67.9%) than in those with mild to moderate autism (17.6%), P = 0.001. Conclusions: Serum IL-17A levels were raised in the group with autism, and the levels correlated significantly with the severity of autism. This is the first study to measure levels of IL-17A in relation to the severity of autism, to our knowledge. Further research, with a larger subject population, is warranted to determine whether the increase of serum IL-17A levels plasma has a pathogenic role in autism, and whether anti- IL-17A therapy could be useful.

3. Krupp LB, Hyman LG, Grimson R, Coyle, PK, Melville, P, Ahnn, S, Dattwyler, R, Chandler, B, 2003, Study and treatment of post Lyme disease (STOP-LD): A randomized double masked clinical trial, Neurology, 24 June 2003, Vol. 60(12), pp. 1923-30.

4. Jones CR, Smith H, Gibb E, Johnson L., 2005, Gestational Lyme Disease Case studies of 102 Live Births. Lyme Times 2005:34–6. Summer.
   Jones et al. performed a comprehensive case history review on the charts of 102 gestational borrelia infection / Tick Borne Illness cases. 9% had been diagnosed with autism and 56% with attention deficit disorder. Psychiatric symptoms included irritability or mood swings (54%), anger or rage (23%), anxiety (21%), depression (13%), emotional (13%), OCD (11%) and suicidal thoughts (7%).
   Neurological symptoms included headache (50%), vertigo (30%), developmental delays (18%), tic disorders (14%), seizure disorders (11%), involuntary athetoid movements (9%) and hypotonia (7%).
   Sensory sensitivity symptoms included photophobia (43%), hyperacuity (36%), motion sickness (9%) and other (tactile, taste or smell) (23%).
   Cognitive symptoms included poor memory (39%), cognitive impairments (27%), speech delays (21%), reading/writing (19%), articulation (17%), auditory/visual processing (13%), word selectivity (12%), and dyslexia (18%).
   GI symptoms were common and included GERD (27%), abdominal pain (29%), diarrhea or constipation (32%), and nausea (23%).
   As a control, 66 mothers with Lyme disease who were treated with antibiotics prior to conception and during the entire pregnancy; all gave birth to normal healthy infants.

5. Blanco, K, and Geoffrey P. R, 2007, Lyme Disease and Autism a New Paradigm." *Townsend Letter: The Examiner of Alternative Medicine* Apr. 2007

6. RC Bransfield 2007, Lyme disease, comorbid tick-borne diseases, and neuropsychiatric disorders, Psychiatric Times, Dec 2007, Vol. 24 (14), p. 59.
   Many recall the phrase" To know syphilis is to know medicine." Now Lyme disease (Lyme borreliosis), the new" great imitator," 1 is the ultimate challenge to the breadth and depth of our knowledge.

7. Bransfield RC, Wulfman JS, Harvey WT, Usman A,. 2008, The association between tick-borne infections, Lyme borreliosis and autism spectrum disorders, Medical Hypotheses. Vol 70(5), pp. 967-74.
   Tick-borne infections, including Lyme disease contribute to developing autism spectrum disorders (ASD) by direct effects, promoting other infections and immune effects during fetal development and infancy. Combined with other predisposing and contributory factors these infections may provoke immune reactions in susceptible individuals that result in inflammation, molecular mimicry, kynurenine pathway changes, increased quinolinic acid and

decreased serotonin, oxidative stress, mitochondrial dysfunction and excitotoxicity that impair the development of the amygdala and other neural structures and neural networks resulting in a partial Klüver–Bucy Syndrome and other deficits resulting in autism spectrum disorders and/or exacerbating ASD from other causes.
Supporting data includes multiple cases of mothers with Lyme disease and children with ASD; fetal neurological abnormalities associated with tick-borne diseases; similarities between tick-borne diseases and ASD regarding symptoms, pathophysiology, immune reactivity, temporal lobe pathology, and brain imaging data; positive reactivity in several studies with ASD patients for Lyme disease (22%, 26% and 20–30%) and 58% for mycoplasma; similar geographic distribution and improvement in autistic symptoms from antibiotic treatment.

8. Nicholson G., "Chronic Bacterial and Viral Infections in Neurodegenerative and Neurobehavioral Diseases", Lab Med. 2008;39(5):291-299.
Often patients with neurodegenerative or neurobehavioral diseases have chronic, neuropathic infections that could be important in disease inception, progression or increasing the types/severities of signs/symptoms. Although controversial, the majority of patients with various neurodegenerative or neurobehavioral conditions, such as amyotrophic lateral sclerosis, multiple sclerosis, Alzheimer's disease, Parkinson's disease and autistic spectrum disorders, show evidence of central nervous system and/or systemic bacterial and viral infections. For example, using serology or polymerase chain reaction evidence of Chlamydia pneumoniae, Borrelia burgdorferi, Mycoplasma species, human herpesvirus-1 and -6 and other bacterial and viral infections revealed high infection rates that were not found in control subjects.
Although chronic infections were not found in some studies and the specific role of chronic infections in neurological disease pathogenesis has not been determined or is inconclusive, the data suggest that chronic bacterial and/or viral infections could be common features of progressive neurodegenerative and neurobehavioral diseases.

9. Bransfield RC, 2009, Preventable cases of autism: the relationship between chronic infectious diseases and neurological outcome. Pediatric Health, Vol. 3(2), pp. 125-40.
There is evidence that chronic infections and immune reactions associated with them may contribute to causing autism spectrum disorders. These infections include Babesia, Bartonella, Borrelia burgdorferi, Ehrlichia, Human heprevirus-6, Chlamydia pneumoniae and Mycoplasma (in particular Mycoplasma fermentans). Maternal immune reactions to infections appear to adversely affect fetal brain development and possible pathophysiological mechanisms include both inflammatory cytokines such as Interluken-6 and maternal autoantibodies to fetal neural tissue of the same kilodalton mass as those seen with Borrelia burgdorferi and some other chronic infections. The timing of the infection and immune response is critical in determines the pathophysiology. It is advisable to evaluate women who are pregnant or planning on becoming pregnant for chronic infections, especially if they demonstrate symptoms of an infection or a systemic illness with persistent inflammatory symptoms. They and the newborn should be treated when indicated.

10. M Kuhn, S Grave, R Bransfield, S Harris 2012, Long term antibiotic therapy may be an effective treatment for children co-morbid with Lyme disease and autism spectrum disorder, Medical hypotheses, May 2012, pp.606-15.
Patients diagnosed with Lyme disease share many of the same physical manifestations as those diagnosed with an Autism Spectrum Disorder (ASD). In this study four male children (ages 26–55months) who have an ASD diagnosis and one male child (age 18months) ...

11. RC Bransfield 2012, The psychoimmunology of Lyme/tick-borne diseases and its association with neuropsychiatric symptoms, The open neurology journal, 2012, Vol. 6, pp. 88-93.
Abstract: Disease progression of neuropsychiatric symptoms in Lyme/tick-borne diseases can be better understood by greater attention to psychoimmunology. Although there are multiple contributors that provoke and weaken the immune system, infections and ...

12. RC Bransfield, 2012, Relationship of Inflammation and Autoimmunity to Psychiatric Sequelae in Lyme Disease, Psychiatric Annals, September 2012, Vol. 42(9), pp. 337-341.
The term "Lyme disease" only refers to an infection by B. burgdorferi (which has four different species that are pathogenic in humans and 300 different strains), or to a tick-borne infection that may include B. burgdorferi and/or other tick-borne pathogens and opportunistic infections.

13. Autism and lyme Disease, RC Bransfield, M Kuhn - JAMA, 2013, 28 August 2013, Vol. 310(8), pp.856-7.

14. M Kuhn, R Bransfield, 2014, Divergent opinions of proper Lyme disease diagnosis and implications for children co-morbid with autism spectrum disorder, Medical hypotheses, September 2014, Vol. 83(3), pp. 321-5.
Abstract: This paper proposes that some children with an autism spectrum disorder (ASD) in the United States have undiagnosed Lyme disease and different testing criteria used by commercial laboratories may be producing false negative results.

Biochemical Similarities between Autism Spectrum Disorder and Borrelia Infection / Tick Borne Illness

- Testing patients with autism and BI/TBI also reveals biochemical similarities. Disorders of an oxidoreductive system in CSF and serum, increases of superoxide dismutase, increased glutathione peroxidase activity, increased concentration of serum malondialdehyde and decreased glutathione have been detected in neuroborreliosis and BI.
- In autism, several studies have suggested alterations in the activities of antioxidant enzymes such as superoxide dismutase and glutathione peroxidase, altered glutathione levels and homocysteine/methionine metabolism, increased malondialdehyde levels and reduced glutathione.
- Impaired methylation & sulfation in both ASD & BI/TBI

Biochemical Similarities: References

15. Pancewicz SA, Skrzydlewska E, Hermanowska-Szpakowicz T, Stankiewicz A, Kondrusik M. Evaluation of oxidoreductive potential of patients with neuroborreliosis. Przegl Epidemiol. 2002;**56**(3):425-33.
16. Pancewicz SA, Skrzydlewska E, Hermanowska-Szpakowicz T, Zajkowska JM, Kondrusik M. Role of reactive oxygen species (ROS) in patients with erythema migrans, an early manifestation of Lyme borreliosis. Med Sci Monit. 2001;**7**(6):1230-5.
17. Chauhan A, Chauhan V. Oxidative stress in autism. Pathophysiol. 2006;**13**(3):171-81.
18. Chauhan A, Chauhan V, Brown WT, Cohen I. Oxidative stress in autism: increased lipid peroxidation and reduced serum levels of ceruloplasmin and transferrin--the antioxidant proteins. Life Sci. 2004;**75**(21):2539-49
19. James SJ, Cutler P, Melnyk S, Jernigan S, Janak L, Gaylor DW, Neubrander JA. Metabolic biomarkers of increased oxidative stress and impaired methylation capacity in children with autism. Am J Clin Nutr. 2004;**80**(6):1611-7.

Brain Imaging Similarities

- Both BI/TBI and ASD patients demonstrate significant temporal lobe dysfunction. In autism the cerebral cortex, hippocampus, and amygdala showed trends toward being disproportionately smaller in the developing autistic brain. In addition smaller amygdala volume correlates with impairments in nonverbal social impairment in autistic patients. Infectious encephalopathies associated with autistic symptoms have demonstrated lesions of the temporal lobes. PET scanning of neuroborreliosis patients demonstrates the most striking finding was hypometabolism, which correlates with decreased activity, in the temporal lobes in 74% patients.
- Both BI/TBI and ASD demonstrate predominately white matter encephalopathy. Regional cerebral blood flow suggests that Lyme encephalopathy may primarily affect cerebral white matter.
- Disruption of white matter tracts between regions implicated in social functioning may contribute to impaired social cognition in autism.
- Both ASD and BI/TBI patients demonstrate sensory hyperacusis and this clinical observation is supported by brain imaging of patients with BI that demonstrates increased thalamus activity and increased activity in auditory and visual areas of cortex.

Brain Imaging Similarities References

20. Herbert MR, Ziegler DA, Deutsch CK, et al. Dissociations of cerebral cortex, subcort and cerebral white matter volumes in autistic boys. Brain. 2003;**126**(5):1182-92.
21. Nacewicz BM, Dalton KM, Johnstone T, et al. Amygdala volume and nonverbal social impairment in adolescent and adult males with autism. Arch Gen Psych. 2006;**63**(12):1417-28
22. DeLong GR, Bean SC, Brown FR 3rd. Acquired reversible autistic syndrome in acute encephalopathic illness in children. Arch Neurol. 1981;**38**(3):191-4.
23. Newberg A, Hassan A, Alavi A. Cerebral metabolic changes associated with Lyme disease. Nucl Med Commun. 2002;**23**(8):773-7.
24. Fallon BA, Keilp J, Prohovnik I, Heertum RV, Mann JJ. Regional cerebral blood flow and cognitive deficits in chronic Lyme disease. J Neuropsychiatry Clin Neurosci. 2003;**15**(3):326-32.
25. Morgen K, Martin R, Stone RD, et al. A. FLAIR and magnetization transfer imaging of patients with post-treatment Lyme disease syndrome. Neurol. 2001;**57**(11):1980-5.
26. Steinbach JP, Melms A, Skalej M, Dichgans J. Delayed resolution of white matter changes following therapy of B burgdorferi encephalitis. Neurol. 2005;64(4):758-9.
27. Belman AL, Coyle PK, Roque C, Cantos E. MRI findings in children infected by Borrelia burgdorferi. Pediatr Neurol. 1992;8(6):428-31.
28. Fernandez RE, Rothberg M, Ferencz G, Wujack D. Lyme disease of the CNS: MR imaging findings in 14 cases. AJNR Am J Neuroradiol. 1990;11(3):479-81.
29. Logigian EL, Johnson KA, Kijewski MF, et al. Reversible cerebral hypoperfusion in Lyme encephalopathy. Neurol. 1997;49(6):1661-70.
30. Barnea-Goraly N, Kwon H, Menon V, Eliez S, Lotspeich L, Reiss AL. White matter structure in autism: preliminary evidence from diffusion tensor imaging. Biol Psych. 2004;55(3):323-6.
31. Chung MK, Dalton KM, Alexander AL, Davidson RJ. Less white matter concentration in autism: 2D voxel-based morphometry. Neuroimage. 2004;23(1):242-51.
32. Moeller, JR. Functional Neuroimaging findings in Chronic LYD; Research as a Tool to Solve Medical Controversies. Lyme and TBD Research Cen Columbia Univ. 2007 .

Attachment 6 Government Correspondence

Letter dated 29/09/2015 from the offices of the Federal Minister for Health and the Federal Minister for Sport, the Hon. Sussan Ley MP, as sent to our local Federal Member for Herbert, Mr Ewen Jones MP.

THE HON SUSSAN LEY MP
MINISTER FOR HEALTH
MINISTER FOR SPORT

Ref No: MC15-015312

Mr Ewen Jones MP
Member for Herbert
PO Box 226
AITKENVALE QLD 4814

Dear Mr Jones Ewen

Thank you for your representations of 28 August 2015 on behalf of [redacted] and [redacted], regarding Lyme disease.

I am sorry to learn that the [redacted] family are suffering from a number of medical conditions.

My Department, through the Chief Medical Officer (Professor Chris Baggoley) has taken an interest in Australian patients who are experiencing and sharing their stories of a chronic debilitating illness which some medical and health practitioners have ascribed to Lyme disease or a Lyme disease like syndrome.

Lyme disease is a common tick-borne illness in the United States of America, Europe and parts of Asia which is caused by an infection with a bacterium carried by ticks. I am advised that, as yet, neither a causative agent nor a vector for Lyme disease has been identified conclusively in Australia, despite research on this topic.

Patients presenting with a chronic debilitating illness for which no cause can be identified, is a particularly difficult situation. I am advised by my Department's medical practitioners that patients should receive treatment based on an accurate diagnosis. When an accurate diagnosis is not available, symptoms should be ameliorated where possible. When a clinician has doubt about the accuracy and quality of diagnostic tests, it is important to first do no harm and where possible relieve pain and suffering. In the situation of patients presenting with a chronic debilitating illness this does not automatically mean long term, multiple antimicrobial therapy. Each patient should be investigated for the cause of their symptoms. For example, the children of the [redacted] family have been diagnosed with disorders on the autism spectrum. This is not caused by an infectious disease and specialist paediatric care is required. My Department is not aware of any verifiable evidence that classical Lyme disease is associated with autism in children. Additionally, there remains no verifiable repeatable evidence for Lyme disease transmission sexually or vertically from mother to child.

The Department of Health welcomes the exciting and ground breaking research publication from Professor Peter Irwin and his team from Murdoch University. This Australian Research Council funded work is published in: www.parasitesandvectors.com/content/8/1/345/abstract, and has revealed amongst the large number of bacteria in ticks at least one new species for further investigation. While no Lyme disease *Borrelia* bacteria were found in Australian ticks, the Murdoch team was able to readily detect Lyme disease bacterial DNA in the ticks from Germany. In one Australian tick collected from a wild echidna, out of a sample of 196 ticks, the DNA from a *Borrelia* species associated with relapsing fever was detected. The clinical significance of this finding is still to be determined and should not be overstated. My Department will remain engaged with Professor Irwin to consider the implications of this research for human health in Australia. It is anticipated that research on ticks taken from humans will be published later in 2015.

The Chief Medical Officer has had the opportunity to speak with Professor Irwin, who emphasised that it is not yet appropriate to link the bacteria that he found in the ticks with them causing disease in humans. Nothing can be assumed without further research.

In a country where the presence of Lyme disease has not been confirmed, such as Australia, it is not possible to reliably diagnose Lyme disease on clinical signs and symptoms alone as there are many other diseases (infectious and non-infectious) that can have similar clinical features.

Tests to diagnose Lyme disease are technically complex and require specialist expertise. They should only be conducted in laboratories that are accredited in Australia and compliant with AS ISO 15189 (accredited laboratories are listed at www.nata.asn.au). Such accredited Australian diagnostic laboratories are able to diagnose Lyme disease by serology in patients who have returned from overseas areas where Lyme disease is endemic.

The medical testing accreditation scheme is run jointly by the National Association of Testing Authorities (NATA), Australia with the Royal College of Pathologists of Australasia based on policy guidance provided by the National Pathology Accreditation Advisory Council. The policy guidance takes the form of standards and guidelines which pathology laboratories must adopt in order to be accredited.

Individual laboratories apply to NATA for accreditation in medical testing and then undergo an assessment by NATA selected assessors. Once accredited, laboratories become members of NATA.

The Institute of Clinical Pathology and Medical Research's microbiology laboratory has medical testing accreditation and is generally regarded as the reference laboratory in Australia for Lyme disease serology.

My Department has recently contracted with the National Serology Reference Laboratory to undertake an evaluation of the serological assays currently used for the diagnosis of Lyme disease in some specialist Lyme disease laboratories in Australia and overseas as well as accredited pathology laboratories in Australia. The specimens for this evaluation have been collected from individuals in Australia and overseas both with and without symptoms of Lyme disease. The results will be used to examine the performance characteristics of these laboratory tests and hopefully resolve the conundrum of discordant results in laboratories in Australia and overseas.

In 2013, a working group of the Communicable Diseases Network Australia (CDNA) assessed the need for the national notification of Lyme disease in Australia. The assessment was made against CDNA and the Public Health Laboratory Network endorsed criteria. These include the outbreak potential of the disease, whether national notification is required to facilitate public health follow-up and the feasibility of data collection.

CDNA considered that that national notification of Lyme disease is not currently warranted given there is currently no definitive evidence of Lyme disease being acquired in Australia and also the difficulties associated with developing an acceptable case definition. Rather they concluded that other methods for monitoring the disease would be more appropriate. One possible way would be for states and territories to work with public health laboratories to improve data on the number of cases and to ensure the appropriateness of laboratory protocols and practices.

CDNA did note that the need for national notification would be reassessed if any new evidence of locally acquired disease and the presence of a competent vector became available.

In terms of access to treatment, GPs in Australia are able to refer their patients to specialist clinics at public hospitals. For example, in a case of suspected Lyme disease, an appropriate referral could be to the infectious diseases clinic at the local hospital. Outpatient appointments at public hospitals are free-of-charge to the individual. In Australia, infectious diseases physicians are the appropriate specialists to support patients with questions and concerns about Lyme disease. If patients are not satisfied with the advice that they are being given they should seek a second opinion from another infectious diseases physician. If Lyme disease is diagnosed, the antibiotics for treating the disease are readily available. Apart from Lyme disease and possible Lyme disease like illnesses, infectious diseases physicians in Australia are knowledgeable about tick-bite associated infections, especially in patients presenting with a rash and fever.

The Chief Medical Officer convened the Clinical Advisory Committee on Lyme Disease (the Committee) to provide advice on the evidence for Lyme disease in Australia, diagnostic testing, treatment and research requirements. The Committee met five times and the outcomes of those meetings are available on my Department's website at www.health.gov.au/lyme-disease. The Committee has now ceased, with the last meeting being held on 15 July 2014. A report that details the progress achieved against each of the Committee's terms of reference is also available on the website.

Even though the Committee has now ceased, my Department's interest is being maintained. My Department continues to:

- consult with members of the Committee;
- monitor progress made in research;
- act as a point of contact within the Australian Government for the Lyme disease community, including medical practitioners and state and territory health authorities;
- seek advice from international partners; and
- write to patient groups and medical practitioners to update them of any progress.

Over the time that my Department has been engaged in this work it has become apparent that many members of the medical profession remain unaware that Lyme disease infection should be considered in Australians returning from travel in endemic areas and tourists from those locations. My Department has developed an *Australian guideline on the diagnosis of overseas acquired Lyme disease/borreliosis* which is now available on my Department's website. The document is focused on infection acquired in endemic areas overseas. It is hoped as we learn more about the chronic debilitating illness affecting Australians we will be able to modify the document to accommodate their situation.

I remain concerned about Australians suffering a chronic debilitating illness with unexplained symptoms that some healthcare practitioners have ascribed to Lyme disease and will continue to monitor the situation and encourage efforts made by Australian medical practitioners and medical scientists to undertake research to define the cause of this chronic debilitating illness.

Thank you for raising your constituents' concerns.

Yours sincerely

[redacted]

The Hon Sussan Ley MP
29 SEP 2015

SECTION B

PERSONAL ACCOUNTS - BORRELIA INFECTION

SUBMISSION CONTENTS:

Terms of Reference c - the process for diagnosis of patients with a Lyme-like illness, with a specific focus on the laboratory testing procedures and associated quality assurance processes, including recognition of accredited international laboratory testing.

Terms of Reference f - the signs and symptoms Australians with Lyme-like illness are enduring, and the treatment they receive from medical professionals; and

Terms of Reference g - any other related matters.

3.0 FAMILY INFECTION – SUPPORTING PATHOLOGY

My family has undertaken extensive pathology to identify borrelia, all of which is presented in Table 1. Positive test results are shaded green, equivocal are orange with negative results not shaded.

Table 3 – Pathology Tests Borrelia

Person	Date	Laboratory	Test	Result	Test Type (Indirect or Direct)	Comment Refer to Attachment D for test explanation
Adult 1	Jun-15	InfectoLAB	Borrelia Immunoblot	IgM Borderline IgG Positive	Indirect	Diagnosis Borrelia Infection.
Adult 1	Jun-15	InfectoLAB	Borrelia Elispot-Lymphocyte-Transformation Test	Positive	Direct	Diagnosis Borrelia. Cellular activity against borrelia.
Adult 1	May-15	Australian Biologics	Borrelia Mikrogen recomLine Immunoblot	IgM Positive IgG Borderline	Indirect	Diagnosis Borrelia. Values exactly on lower limit of the detection range for IgG.
Adult 1	Aug-15	Australian Biologics	Borrelia Serum Culture PCR	Equivocal	Direct	DNA of bacteria present in 2 samples, not present in 2 samples. Retest recommended.
Adult 1	Sep-15	ICPMR Westmead	Borrelia ELISA	Negative	Indirect	No antibodies detected. Negative result is not proof of no infection, rather than of no detectable immune response to those markers the test can detect.
Adult 1	Jun-15	InfectoLAB	Immune System CD-57 Flow Cytometry	Positive	Indirect	The CD57-cell-count indicate a chronic immune-suppressive situation which may be caused by Borrelia burgdorferi.
Adult 2	May-15	Australian Biologics	Borrelia Blood Culture PCR	Positive	Direct	Borrelia bacteria in the blood. Active Infection.
Adult 2	Jul-15	Australian Biologics	Borrelia Serum Culture PCR	Positive	Direct	Borrelia bacteria in the serum. Active Infection.
Adult 2	Jun-15	InfectoLAB	Borrelia Elispot-Lymphocyte-Transformation Test	Positive	Direct	Diagnosis Borrelia. Cellular activity against borrelia.
Adult 2	Jun-15	InfectoLAB	Borrelia Immunoblot	IgM Negative IgG Negative	Indirect	Borrelia IgG and IgM not detected. Negative result is not proof of no infection, rather than of no detectable immune response.
Adult 2	Apr-15	Australian Biologics	Borrelia Mikrogen recomLine Immunoblot	IgM Negative IgG Positive	Indirect	Diagnosis Borrelia.
Adult 2	Sep-15	ICPMR Westmead	Borrelia ELISA	Negative	Indirect	Antibodies Detected. Not proof of active infection, just of immune response to those markers test can detect.
Adult 2	Jun-15	InfectoLAB	Immune System CD-57 Flow Cytometry	Positive	Indirect	The CD57-cell-count indicate a chronic immune-suppressive situation which may be caused by Borrelia.
Child 1	Aug-15	Australian Biologics	Borrelia Serum Culture PCR	Not Detected	Direct	DNA of bacteria not detected. Note this is not proof of absence of infection, just proof that the sample did not contain borrelia.
Child1	May-15	Australian Biologics	Borrelia Mikrogen recomLine Immunoblot	IgM Borderline IgG Positive	Indirect	Diagnosis Borrelia. Values well within detection range for IgG and at the lower limit of the detection range for IgM.
Child1	Jul-15	InfectoLAB	Borrelia Elispot-Lymphocyte-Transformation Test	Positive	Direct	Diagnosis Borrelia. The Elispot indicates a cellular activity against Borrelia.
Child 1	Jul-15	InfectoLAB	Borrelia Immunoblot	IgM Negative IgG Negative	Indirect	Borrelia IgG and IgM not detected. Negative result is not proof of no infection, rather than of no detectable immune response.
Child1	Sep-15	ICPMR Westmead	Borrelia ELISA	Positive	Indirect	Antibodies Detected. Not proof of active infection, just of immune response to those markers test can detect.
Child 1	Sep-15	ICPMR Westmead	Borrelia Western Blot	Negative	Indirect	No antibodies detected. Negative result is not proof of no infection, rather than of no detectable immune response with testing assay used (only sensitive to B. burgdoferi and B. afzelii)
Child 1	Jun-15	InfectoLAB	Immune System CD-57 Flow Cytometry	Negative	Indirect	Immune system on lowest end of normal range. The CD57-cell-count indicate the presence of a chronic immune-suppressive situation
Child 2	May-15	Australian Biologics	Borrelia Mikrogen recomLine Immunoblot	IgM Negative IgG Borderline	Indirect	Diagnosis Borrelia. Values were exactly on the lower limit of the detection range for IgG. Negative IgM result is not proof of no infection, rather than of no detectable IgM.
Child 2	Jul-15	Australian Biologics	Borrelia Serum Culture PCR	Equivocal	Direct	DNA of bacteria present in 2 samples, not present in 2 samples. Retest recommended.
Child 2	Jul-15	InfectoLAB	Borrelia Immunoblot	IgM Negative IgG Negative	Indirect	Borrelia IgG and IgM not detected. Negative result is not proof of no infection, rather than of no detectable immune response.
Child 2	Jul-15	InfectoLAB	Borrelia Elispot-Lymphocyte-Transformation Test	Borderline	Direct	Diagnosis Borrelia. Borderline means that values were at the lower limit of the detection range.
Child 2	Sep-15	ICPMR Westmead	Borrelia ELISA	Negative	Indirect	No antibodies detected. Negative result is not proof of no infection, rather than of no detectable immune response to those markers the test can detect.
Child 2	Oct - 15	IGeneX	Borrelia IgG and IgM Western Blot	Negative	Indirect	No antibodies detected. Negative result is not proof of no infection, rather than of no detectable immune response with testing assay used
Child 2	Oct - 15	IGeneX	Multiplex Whole Blood & Serum	Negative	Direct	Detects specific DNA sequences for B. burgdorferi, B. afzelii, B andersonii and B garinii.
Child 2	Jun-15	InfectoLAB	Immune System CD-57 Flow Cytometry	Positive	Indirect	The CD57-cell-count indicate a chronic immune-suppressive situation which may be caused by Borrelia.

In Australia, my family is not considered to have borrelia. This is concerning considering of the 28 tests results my family have, 15 are positive, 2 are equivocal and 11 are negative. Indirect tests represent the majority of all negative tests (9 negative indirect tests). Indirect tests measure immune response which is problematic in borrelia because borrelia dysregulates the immune system, so this outcome could be anticipated in a late stage borrelia infection. Direct testing shows an active infection. Only 2 of the 10 direct tests ordered were negative. Family test results indicate immune dysregulation and active infection. As ICPMR Westmead only undertakes indirect testing, 4 of the 11 negative indirect test results were from ICPMR Westmead.

Statistics presented by Test Type:

Direct Tests

Number of positive Direct Tests:	6
Number of equivocal Direct tests:	2
Number of negative Direct Tests:	2
TOTAL	**10**

Indirect Tests

Number of positive Indirect Tests:	9
Number of negative Indirect Tests:	9
TOTAL	**18**

Statistics presented by Test Result Type:

Positive Results

Direct tests	6	
Indirect tests demonstrating immune system exposure to borrelia	6	
Indirect tests demonstrating chronic immune system suppression	3	
TOTAL	**15**	

Equivocal Results

Direct tests	**TOTAL**	**2**

Negative Tests

Direct tests	2	
Indirect tests demonstrating immune system exposure to borrelia	8	(4 from ICPMR)
Indirect tests demonstrating chronic immune system suppression	1	
TOTAL	**11**	

4.0 PERSONAL ACCOUNT [redacted]

4.1 Diagnosis

I have four positive test results demonstrating infection with Borrelia. Testing confirms immune response to borrelia as well as evidence of active infection with Borrelia. A further test demonstrates the typical immune system dysregulation observed in late stage chronic borrelia (chronic Lyme) infection.

Laboratory	Test Type	Result	Comment on Result
InfectoLAB	Immune System CD-57 Flow Cytometry	Positive	The CD57-cell-count indicate a chronic immune-suppressive situation which may be caused by Borrelia burgdorferi.
Australian Biologics	Borrelia Blood Culture PCR	Positive	Borrelia bacteria in the blood. Active Infection. Direct testing.
Australian Biologics	Borrelia Serum Culture PCR	Positive	Borrelia bacteria in the serum. Active Infection. Direct testing
InfectoLAB	Borrelia Elispot-Lymphocyte-Transformation Test	Positive	Diagnosis Borrelia. Cellular activity against borrelia. Direct testing
Australian Biologics	Borrelia Mikrogen recomLine Immunoblot	IgM Negative IgG Positive	Diagnosis Borrelia. Evidence of immune response to borrelia. Indirect testing.

I have additional diagnosis as follows:
1. Epstein Barr Virus
2. Arboviral infection unidentified
3. Babesia - I am symptomatic of babesia. No testing that could have identified babesia duncani was completed on me due to cost restrictions and unavailability of this test in Australia. My youngest son does have a positive diagnosis for active babesia duncani infection and examining his medical history reveals this infection likely present from birth. Likewide, my oldest son has been symptomatic from birth but did not have pathology testing done that could identify this infection.
4. Bartonella - I am symptomatic of bartonella. Testing for bartonella henselae was negative, but relied on an immune response which I know to be unreliable. Both my children have positive immune response to bartonella henselae and I believe that I was the source of this infection.

4.2 Where I Contracted Borrelia / Tick Borne Illness

I have had multiple insect bites over the years, both prior to and during illness, some with accompanying inflammation and rash. I grew up playing in the bushland of Queensland. I have travelled to several countries with endemic borrelia including Argentina, Peru, Vietnam, Indonesia, Papua New Guinea. I have worked in close proximity to, and within the colonies of large migratory birds. I have worked in the most remote parts of the Australian coastline. Personally, I suspect that I have contracted infections progressively, from more than one bite.

4.3 Summary of Symptoms, Specialists and Procedures

All the following conditions have been diagnosed since I started becoming symptomatic of tick borne illness: I have been unwell for more than 13 years.

1. Muscle weakness and atrophy resembling limb girdle muscular dystrophy. Associated mobility issues. Now largely resolved by intervention with diet and supplementation.
2. Fasciculation of most muscles in my body.
3. Nerve damage and associated symptoms (shooting pain, loss of feeling, pins and needles)
4. Seizures lasting up to 2.5 hours in duration and with paralysis following. Periods of absence.
5. Raynaud's phenomena
6. Sjogren's syndrome
7. Several elevated autoantibodies
8. Breast cysts
9. Cognitive including episodic headaches
10. Intermittent problems with speech production (loss of speech coherency/slurring)
11. Intermittent and highly variable problems with vision relating the brain, not the eyes.
12. Food allergies and intolerance.
13. Multiple Chemical Sensitivities and allergies.
14. Sensory Processing issues related to Auditory and Visual fields.
15. Inability to detect temperature with any accuracy (hot water a problem).
16. Muscles perform large involuntary movements.

17. Intermittent fatigue not related to activity or sleep.
18. Issues with regulation of mood.
19. Cramping
20. Poor short and long term memory
21. Recurring infection in urinary tract. Urine specific gravity below lowest range despite hydration level
22. Intermittent symptoms of inappropriate antidiuretic Hormone secretion.
23. Intermittent symptoms of diabetes insipidus.
24. Constant moderate headache through top of neck/back of skull not related to skeletal issues.
25. Exocrine pancreatic insufficieny
26. Eczema
27. Heart irregularities
28. Significantly impaired detoxification systems

I consulted the following specialists, many prior to my diagnosis:
1. Symptoms of neurological disorder (neurologist)
2. Symptoms of endocrine disorder (endocrinologist)
3. Symptoms of metabolic disorder (metabolic specialist)
4. Symptoms of heart disorder (cardiologist)

I have had surgery for muscle biopsy.

I have had numerous scans (Xray, CT, MRI, Spect, Ultrasound) and pathology tests completed. This was at enormous expense to myself and the taxpayer. The vast majority of this expense incurred prior to diagnosis. If I had been diagnosed earlier, it is likely much of this testing would not have been required.

I presented to emergency at hospital on four occasions, twice with seizure, twice with pregnancy complications now understood to be related to tick borne disease.

4.4 Financial Impact

Treatment of borrelia infection is far less expensive than treatment of autism, particularly considering the lifetime cost of disability to both the government and the individual. It makes no financial sense not to treat borrelia.

If we had known to treat the borrelia instead of the autism, on the basis of the available research, I believe that would have ameliorated a large number of developmental problems in our children.

The family's savings were exhausted prior to diagnosis of borrelia in addressing the complex health and intervention requirements of the children and myself. Had the diagnosis of borrelia been evident earlier, finances would not have been so depleted and treatment would have been possible.

I do not have any diagnosis that qualifies me for home care or support. Support available for my children is limited to that under Medicare and there is no in home assistance.

4.5 Employment Impact

My health at the moment is not stable enough to permit a return to employment, and nor is the health of my children.

4.6 My Story

I grew up in a small rural town and left home at age 17 to study to pursue a tertiary education. I had extremely limited financial support and I self-funded throughout my degree, working in restaurants and engineering firms, to graduate with a bachelor degree five years later. I did not receive government support. I was extremely fit.

Upon graduation I had considerable work experience and pursued construction engineering. Projects I worked on were varied and included the upgrade of the Queen street mall in Brisbane, in particular I coordinated the construction of the central structure. My work took me to Papua New Guinea and to the very remote jungles of Indonesia in Borneo. I backpacked solo through Vietnam. I project managed the design and construction of new maritime channels in far north Queensland. I travelled more often than not, and nearly always to remote parts of the Australian coastline. Beyond that of a spider or mosquito bite, I was naïve to the fact that I could become ill from tick or bird lice. I never used insect repellent. Whilst working in the remote Torres Strait I collected spider specimens for a prominent Australian doctor and leading spider expert. I would regularly have to shovel a foot of guano from the top of platforms

anywhere up to 20m above the ocean in order to access the control cabinet. I climbed large towers on land and around large bird nests. I was bitten by bird lice from large migratory birds too many times to count.

It was during my time as an engineer I became obviously sick. When in the office I would frequently go to the sick room during breaks to rest. I remember being terribly unwell but I was managing.

My oldest son was born extremely sick after a complicated and life threatening labour (medical history Section 6.5). My health also deteriorated over this time and that was in part due to the extreme stress of having such an unwell child and considerable sleep deprivation. This was the year that he had three operations and several unknown viral illnesses as well as giardia and norwalk virus

I returned to part time work and hired an aupair to help with my son who was not eating, sleeping or developing normally and was sick. My whole salary went to payment of the aupair, but this break from the home provided opportunity to manage my situation more efficiently. My son was nearly continuously ill that year and had three operations and many bouts of unknown 'viral' illness. I remember taking my son to the doctors not less than six times over the space of a few days knowing how ill he was, to be dismissed each time. The last dismissal being on the morning of the same day that I rushed him to hospital, his blood oxygen was 80, and he was stabilised for emergency surgery that night and received surgery the following morning. Review of video footage taken between my son's first and second birthdays revealed the extent of the regression that occurred during this time.

Life was very busy. I did not prioritise my health over that of my family and it deteriorated but I dismissed this as I was still managing and looking well. I could still ride a mountain bike every morning to work, but could not lift my legs in many directions and frequently lost coherency, had intermittent vision and other neuromuscular problems. It did not affect my work performance. I sought medical assistance on numerous occasions.

With my son accompanying, I presented myself to another new doctor with the list of neurological symptoms I was suffering in the hope I would be diagnosed. Due to my son's behaviour/presentation, the doctor stopped the consultation and exclaimed that my child had autism and the remainder of the appointment was organising his assessment. Prior to that my concerns in relation to my child's development had been largely dismissed by doctors on the basis of maternal incompetence and over reaction. On one occasion when I received a copy of a medical report regarding my son, I was incensed and appalled to read that his health issues were suggested as 'Munchausen by proxy'. Another more observant specialist did alert the GP in his letter to an underlying medical problem.

We had to go through two diagnosis processes in NSW. The first expensive private diagnosis was undertaken, with criteria met for ASD & ADHD, however this was not accepted by the government as my son was not age 3. The second diagnosis was completed only a few months later by government services and by then my son was the correct age.

Medical advice was that the cause of Autism was not known and I could do nothing but accept that. I knew this to be incorrect and purchased a small library of medical text books and started to uncover the truth about autism and identify those few doctors able and willing to help. It was expensive but massive gains were made in my son's health and presentation. Some witnesses to this transformation have described it as a 'miracle'.

At 14 weeks gestation with my second child, I had what was termed a large retro placental haemorrhage diagnosed during emergency hospital admission. I was advised I would miscarry, as this did not occur and there were great concerns in the following months regarding the child's development due to the significant blood loss which continued throughout the pregnancy.

Like his brother, my youngest son was born ill from the onset by emergency cesarian (medical history Section 5.5). Given that we had by this time, considerable experience in dealing with complicated medical issues, he was medically managed more competently than his brother. He stopped breathing numerous times in his sleep every single day/night for the first few years. It was traumatic. As well as other medical conditions and just like his brother, he presented with intermittent spiking fevers of unknown origin and was admitted to the emergency room and to hospital for short times on numerous occasions. Not at any stage was he investigated for any tick borne or congenitally acquired conditions. He was simply sent home when the fever resolved itself without any cause being found or follow up investigation.

My younger son regressed noticeably into Autism following surgery to insert grommets at 7 months age. A month prior to this I had presented my children and myself as a 'case review' at a conference attended by roughly a dozen doctors being trained by one of the best in the world, a doctor that was expert in Autism from the USA and impossible to get a consult with even in that country. Results of biochemical indicators in myself and my children were reviewed and it was concluded that [redacted] had all associated parameters required to regress (autism) and there was nothing I could do to prevent this. This was not upsetting, it was expected news. What is upsetting is that medicine even 5 years ago was well able to measure those chemicals and processes in the human body and sufficient information existed in order to identify the risk group of women and children in relation to autism, and yet five years later still nothing is being done by mainstream medicine. There is no official 'autism checklist', although I have seen such a

book in my local library. After my children's diagnosis, many of the issues they presented with were dismissed as "just autism" by medical professionals.

Autistic regression involved an obvious loss of motor function, regression to primitive reflexes and [redacted] ability to verbalise completely disappeared. He was rendered mute. This entire regression was witnessed to by the therapists who at that time were working with his older brother. He developed a condition the paediatric gastroenterologist had never seen before involving projectile vomiting. We were told that he would never speak, the damage was simply too great, so I removed him from speech therapy and taught him and myself sign language. He acquired 200 signs with great rapidity and his speech returned as a result of this brain rewiring. He now speaks on the 98% for his age.

The following years involved intensive speech therapy / occupational therapy and several tutoring sessions with university student every week for both my children. I coordinated the therapy programs and everything done at home for my children was therapy orientated. I was overwhelmed with the care requirements of my children which included dealing with the systemic nightmare associated with 'education' of children with disability in Australia. There was not much time for me to look after my health.

I had rapid health deterioration following exposure to di-isobutyl phthalate spill inside a new Australian manufactured mattress I purchased (as confirmed by allergy specialist and chemical testing of spill inside mattress). I was prescribed steroids to counteract the extensive burning, peeling and swelling on my face and body that I presented with for the eight months it took me to figure out the source of the allergy. During this time I started to rapidly lose my ability to move many of the large muscle groups in my hip and shoulder girdle. I intermittently lost the ability to see, and could not cross the road without locating a sloped gutter/path and shuffling up it backwards as a baby would do when learning to walk. This had a profound impact on my ability to care for my family. I now understand that steroids should never be given to borreliosis patients.

I was referred to a neurologist who did testing and advised my doctor in writing that I had limb girdle muscular dystrophy. This diagnosis was later revoked following a negative muscle biopsy and no explanation for my condition provided. After seeing many doctors in a short time, including a metabolic specialist who wrote back to my doctor a list of possible medical conditions (none of which I had) including 'conversion disorder', I gave up on the medical profession and did not consult them for a further two years. It confirmed for me that the medical profession was unable to assist with any complex health presentation and in preference to treatment, would instead place blame.

I undertook my own research, and implemented supplementation and dietary protocols that resulted in me regaining the ability to walk and resume use of many of the limb girdle muscles.

In 2015 I located very recent research into maternal autoimmunity and autism. From there I identified the three stealth infections likely to cause my children and my own health conditions and found a private lab to test for them. It took great courage to present the request for private pathology to the doctor, as I had shunned the medical profession for two years. When the results came back, I had my answer. It was a moment of immense relief and excitement, as to me it represented the opportunity for great healing in my family.

The realisation that two of the illness the family had contracted (borrelia and babesia) was not recognised in Australia and that a political medical controversy existed, was to me astounding.

Since the diagnosis of tick borne disease I have been, yet again, faced with enormous pressure to research to understand this illness and identify treatment providers. The children present a limited window of brain plasticity, managing the causative infection is a very high priority in healing them. As the infection in me is very long term and of autoimmune presentation, typical treatment strategies failed. I now have life threatening seizures. There is no accessible, affordable and suitable medical treatment for me and my children in Australia and our finances are exhausted and do not permit us to seek overseas treatment.

4.7 Social Impact

I have found it very difficult to explain my children's Autism treatments and my family's illness to many of my friends and family due to the reaction they give which is disbelief and suspicion based on an inability to understand and accept why the government and our medical services are not providing recognition/treatment. If the medical profession doesn't recognise the illness or doesn't acknowledge or accept the treatment, then very little sympathy or constructive assistance is provided. The disability and illness is confronting, the way my family manages it seems to be moreso.

I do not have the same opportunity to socialise. My life is one of prioritizing my health needs against the health needs, research needs and therapy needs of the other family members. I have to advocate for my children's education, formulate treatment plans and intervention strategies and implement this in a system where there is extremely limited assistance. Priortising therapy targets, specialist medical assistance and medication/supplementation against what

little money there is. My life is consumed with managing the inevitable disaster zone that accompanies disability. The paperwork alone is unbelievable.

The family is subject to the inevitable discrimination that accompanies the incorrect perception that Autism ADHD is a parenting / discipline issue. Too many times to count I have been the subject of ridicule and hostility and witnessed my children subject to the same.

As a parent I have been faced with the requirement to undertake years of medical research and identify not only the likely infections but also the diagnostic testing and treatment strategies for my family's health issues. This requirement is still ongoing and additionally I am frequently faced with the burden of advocacy and education, all the while we are sick. We are required to fund the entire research and treatment exercise despite paying an insane amount of tax for our 'healthcare' and our private healthcare which has been useless in this instance.

I carry guilt that I infected my children, but I hold the medical system and the government wholly accountable. I am proud that my intervention and research into Autism and related conditions has vastly improved my children's outcome and resulted in me identifying the tick borne infections underlying my family's ill health. I only wish that the infections had been identified earlier and that I could return to the life that I worked so hard for.

4.8 My Experience with the Medical Profession

The most difficult aspect of this disease not been the loss of health, family, friends, career and associated stress, it has been in dealing with the medical professionals in Australia in relation to the ignorance that exists underlying etiology of autism and borrelia infection.

It is the responsibility of the medical profession to keep up to date with current research, to convey accurate information to the general public and provide accurate advice to government bodies to prevent epidemic of preventable health conditions such as those related to improper diet, lifestyle, infection and disability. I have not seen this occur.

It is the responsibility of the medical profession to diagnose and treat children that present with illness, and they did not do this. I lost count of the number of times I heard phrases such as "There is nothing you can do to treat Autism" or "We don't know what causes it" or "it is just Autism" to dismiss very real medical issues. I am astounded that with 1 in 49 Australian school children holding an ASD diagnosis, not one doctor or pediatrician I have met to date can clearly explain autism with a sufficient degree of understanding. We do know enough to screen, prevent and reverse much of the Autistic disorder. If it were 1 in 49 children born without teeth, there would be public outcry, money would be offered for research, a specialist field created. The silence on the issue of autism is deafening as address of the issue requires address of the political system that enables the causative environmental and infectious factors to exist.

Many borrelia patients experience the ignorant doctor, the fearful doctor, the dismissive doctor and the doctor that ridicules for the first time as a result of their tick borne infection. My children and I have faced this from the onset in dealings with the medical profession in relation to autism. I have faced redress for giving my children a special diet (now this is accepted mainstream), nutritional therapy (again, now accepted mainstream). By the time the medical profession accepts and understands the pivotal role stealth infections play in the development of psychiatric diagnosis, neurodevelopmental disorders and neurodegenerative diseases, it will be far too late, as it already is.

My family has attended hundreds of medical appointments with upwards of 50 doctors and specialists. Not one of these medical professionals considered tick borne infections of borrelia, bartonella, babesia, rickettsia or mycoplasma or for that matter, any vector borne disease with the exception of Dengue, Barmah Forest and Ross River (mosquito borne). Identification of my family's illnesses was a product of personal research.

4.9 Refusal to Recognise and Treat Borrelia

My family have faced outright denial of the presence of borrelia (overseas acquired, locally acquired, congenitally acquired) in Australia and a refusal of treatment.

I had experience with a doctor whom I had consulted with extensively during my period of greatest illness, after agreeing that I did not have MS and following presentation of my positive borrelia result, he continued to encourage me to obtain an MS diagnosis. The doctor went on to discredit the testing laboratory Australian Biologics by saying that "The industry is full of charlatans", but would not provide a reason as to why he wouldn't consider this diagnosis. He was firm in that it was his position as a GP to send me to specialists and they would diagnose me, however I was directed to go and see another GP if I wanted a referral to the infectious disease specialist. He was extremely agitated and directed me to the door. I was advised the service was free and not to come back.

Another doctor to whom I presented my diagnosis was good enough to write me a referral but was quite firm in letting me know that I could not have this disease because I lived in Australia.

I presented to the infectious disease specialist with two positive pathology results, medical history and travel history in hand. Pathology at that stage comprised of one indirect test showing immune system response to borrelia and one direct test showing borrelia DNA in my bloodwork. I was dismissed without even being offered testing for any other tick borne illness – including the IPCMR Westmead Medicare testing. The reason the specialist (who identified as an American) gave for dismissal was soley related to 'belief' that I did not have "Lyme". I never thought I had Lyme (borrelia burgdorferi sensu stricto), I was quite sure I had a borrelia infection. A full record of this dismissal is contained in Attachment 7 as taken by a witness.

It begs the question as to why are we importing the fallout from a broken American system into Australia? The controversy surrounding testing, vested research/patent interests and issues with the insurance industry have been extensively documented. The response to my family complaint as issued to both State and Federal Ministers provided no support or recognition, but revealed the extent of the issues in Australia. This has been further examined in my husband's technical submission (refer to submission by [redacted]).

In total, after presentation of positive borrelia pathology, of the doctors / specialists my family have consulted:

- **3 have expressed fear to treat borrelia** (note some compassionate doctors will provide partial treatment despite this fear);
- **4 have denied the presence of the disease** even when presented with multiple positive pathology results, travel history and medical history.
- **3 have advised that they could not/would not assist** given the disease complexity and their current knowledge and;
- **4 have advised it was not their position description** (job) to treat the infection.

Note some doctors have used multiple excuses.

4.10 Medical and Travel History

This history has been provided for the benefit of whose people reading this submission, in the hope that the information will assist others presenting with similar symptoms.

1991 Removed tick (Mackay, QLD), presented with rash. Significant fatigue followed, to doctor on two occasions as I was sleeping more than I was awake. No tests of any type were taken, I recovered over a period of months.

1992 to 1996 University, Bachelor Civil Engineering. Numerous antibiotics for recurrent tonsillitis, sinus and urinary tract infections, eczema. Very fit, ran long distance.

1997 to 2008 Recurrent urinary and kidney infections, eczema. Diagnosed with Raynaulds Phenomena. Very fit, Bushwalking, Rockclimbing, Running.

1996 to 1997 Worked in the remote jungles of Borneo, travelled to Singapore, Vietnam and worked for a short time in very remote region of Papua New Guinea. While in PNG I contracted skin rash, dermatologist advised folliculitis he told me he had never seen a case so severe. Problems with spots appearing upon my vision field and was frequently unable to stand from sitting without blacking out. MRI done, issue not found. Gym work and Running.

1997 to 2000 Worked across Queensland on numerous construction sites. Gym work and running.

2000 to 2004 Worked in many remote Australian coastal locations as a civil engineer / maritime engineer. This included a considerable amount of time in the Torres Straight and far north of the country, but extended to far southern regions of Australian waters. I worked typically on remote islands and also in locations of large migratory bird population. Went to New Zealand to deliver technical paper to conference.

2001 Multiple infections in sinus and tonsils, numerous antibiodics. ENT Surgery cauterise nose remove polyps, septoplasty, reduction of inferior turnibates, tonsillectomy, bony tissue removed from frontal sinus and osteomeatal complex functional nasal endoscopic surgery. Ongoing urinarty tract infections, one lasting for six months, low blood pressure problems, ongoing for months with medical monitoring, three unknown but significant viral infections.

2002 Bronchitis, Lower Back Pain, Multiple Urinary Tract Infections, Endometriosis.

2003 – Transferred interstate. Work mostly in remote areas of Australian waters and coastline. Injured L4/L5 disc, doctor noted impaired leg movement, ordered MRI "Paraesthesia left lower leg". L4/5 midlineannulus tear

and moderate broadbased posterior disc bulge contacting both L5 roots found, but not explaining paraesthesia. Neurologist noted numerous nerve issues in arms and legs and ordered MRI spine and completed a nerve conduction test. Requested muscle biopsy but I did not continue at this stage as he had commented that all nasty diseases were ruled out.

2004 - UTI persisting for four months despite at least 8 courses antibiotics. Generally very fit and compensated for movement issues. Continued deterioration of ability to move, evident problems climbing ladders.

2005 – Travelled to South America, Argentina and Rural Peru whilst pregnant. Upon return to Australia was very sick for approx. 2 months with illness unidentified despite investigations. Pubic symphysis disorder during pregnancy.

2006 – Birth of first child. 10 day overdue, non-progressive labour, uterine hyper stimulation to oxytocin administered giving contractions of 40min life threatening.

2006 to 2008
Worked in Queanbeyan (NSW) employing au pair to assist with very sick child. Health deteriorated:

- Heart – Developed heart murmur, echocardiogram.
- Hands - Difficulty opening jars, applying pressure (squeezing), dropping things (judging pressure). Marked weakness in right hand and then both hands, Marked degeneration in ability to sense temperature. Hand shakes.
- Legs – Difficulty ascending stairs, lifting legs, tripping a lot more than usual, bumping into things
- Cognitive – Episodic headaches.
- Feeling 'dumbed down', great effort to speak coherently; eg: it was extremely difficult for me to read out loud from a book. I was in work meetings and would go in fine, and by end of meeting be slurring my speech and sounding drunk by the end of the meeting. Difficulty articulating speech at end of day. Words being spoken in the wrong order or inappropriate use of words. Feeling tired and having frequent low dull headaches, dribbling.
- Eyes – went to optometrist for check as was getting eyes blacked out and fuzzy. No problem found.
- Legs erupting into large nodule type white bumps all over and then bumps disappearing 12 hours later. Occurred several times per week for several months.
- Significant unidentified illness (assumed viral at the time) resulting in secondary infection requiring 5 courses of antibiotics.
- Metallic taste in mouth.
- Testing revealed considerable deficiencies in fat soluble vitamins (Vitamin D was 0).
- Still continuing with exercise, riding my bike across a mountain to work and back.

2009

- First son diagnosed with Autism and ADHD. My heart murmur resolved itself. Insomnia. Pregnant second child. Large retroplacental haemorrhage 14wks gestation, 10 x 15 x 2.5cm told I would miscarry; did not, bed rest; told baby would not develop normally and had several scans showing baby developing normally despite blood loss throughout entire pregnancy.
- Interstate move and resignation from work.
- Birth of second child by emergency caesarean, subsequent infection post surgery. Second child unwell.

2010 - 2011

- No longer able to work as I was unwell with two sick children, one requiring intensive early intervention. Sleep deprivation (unwell children and insomnia).
- Daily headaches. Occasional vision loss, saw optometrist, no cause found.
- Regular loss of speech coherency (like drunk). Daily cramping of many muscles.
- Poor short and long term memory.
- Very Low blood cholesterol despite high cholesterol diet.
- Frequent disorientation in familiar environment.

2012 – 2013
Prolonged 8 month long allergic reaction to di-isobutyl phthalate spill inside mattress (as confirmed by allergy specialist and testing of mattress). Skin peeling, mucosal tissue inflammation. Steroids prescribed.

- Significant loss of muscle function following muscle fasciculation, mobility highly impaired. Iliopsoas, Quadriceps, Gluteus Medias and Maximus, Hamstrings, Hip adductors, Rhomboids, Inferior Trapezius all 2/5 (cannot move against gravity). Many other muscle groups 3/5 (move against gravity but not against force), Diagnosed with Limb Girdle Muscular dystrophy by Neurologist on basis of nerve conduction testing and other testing and presentation, to have diagnosis removed following a negative muscle biopsy result.
- Cardiac problems, isolated unifocal VPC occasionally in bigeminy, normal clinical ECG and echo VPC on Holter Monitor, MRI gave further findings, low to normal blood pressure, some very low blood pressure.
- Low cholesterol
- Metallic taste in mouth
- Cramping

- Multiple painful lumps in breast that came and went. Mammogram and ultrasound done. No cancer, no treatment.
- Repeat episodes of severe pelvic pain and urinary symptoms. Repeated urine samples demonstrate increased white and red cell counts but no bacteria and do not respond to antibiotics. Urine specific gravity reliably below lower limit for seven concurrent tests regardless of hydration level. Ultrasound of pelvis clear.
- Doctor unable to determine problem sends me to Metabolic Specialist who lists possible other tests to do and also lists ''conversion disorder'' in a letter, but did not say it to my face.
- Gave up on the medical specialists at this stage. Did not have funding to continue given large financial burden of two disabled children on one income.

2014

- Self-supplementing, identified intolerance to sugar and starch using blood glucose monitor. Removed starch and sugars from diet with remarkable improvement.
- Gradually improved on muscle function and regained use of many muscles.
- Other symptoms still present but reduced in severity.
- Muscle fasciculation and intermittent function loss continue.
- Dentist and Optometrist note Sjogren's Syndrome.
- Intermittent and regular shooting pains.

2015 to Present.

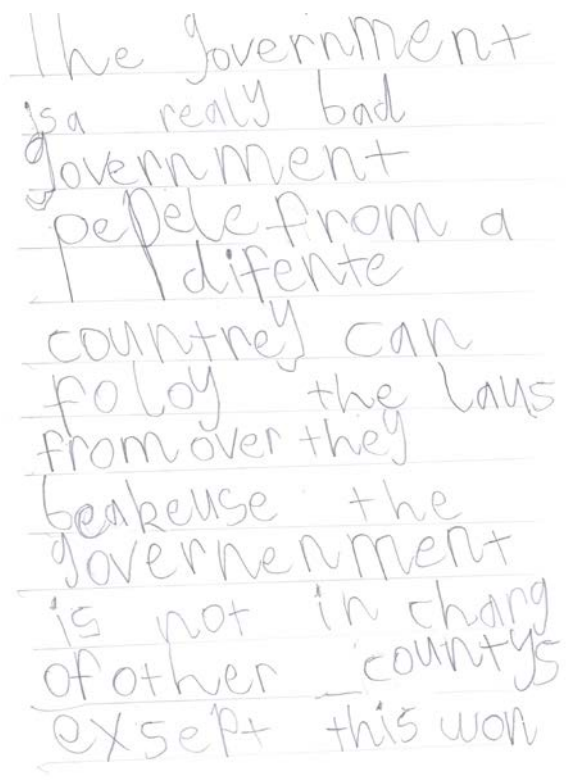
- Crawling sensations under skin. Fasciculation's now deeper and longer (some last up to three days). Unable to stand still with eyes shut. Balance poor.
- Eyesight glasses prescribed for convergence insufficiency and poor accommodation with very divergent eyes suggesting difficulties with oculomotor muscle control. Opthamologist confirmed issue was related to brain not eye function.
- Swallowing difficulty.
- Extreme intermittent pain through side of face originating above ear.
- Difficulty moving tongue and tongue tremor noted by Physiotherapist.
- Four positive tests for borrelia infection.
- Immune system dysregulation.
- Episodic Fatigue
- Epstein Barr virus reactivates at least twice.
- Blastocyctis infection identified and treated.
- Significant pancreatic insufficiency necessitating digestive enzyme replacement.
- Postural Orthostatic Tachycardia Syndrome
- Autonomic nervous system dysfunction
- SPECT scan of the brain, MRI of the brain
- Referred to Infectious Disease Specialsit and denied treatment on basis of lack of belief that I had borrelia (2 positive diagnosis including positive blood culture).
- Pathology - syndrome of inappropriate antidiuretic hormone (ADH) secretion and diabeties insipidus suspected but not sufficient symptoms to make diagnosis. Endocrinologist recommended I see an IDS.
- Short course of antibiotics unsuccessful due to inability of body to detoxify.
- Seizures commenced Nov 2015, life threatening lasting up to 2.5hours. Up to three a day.
- Further two positive diagnoses for Borrelia. (now had four positive diagnosis)
- Saw endocrinologist, recommended I see an IDS.

5.0 PERSONAL ACCOUNT [redacted]

5.1 Letter

The following letter was written by [redacted], age [redacted] yrs.

Page 1 of 3



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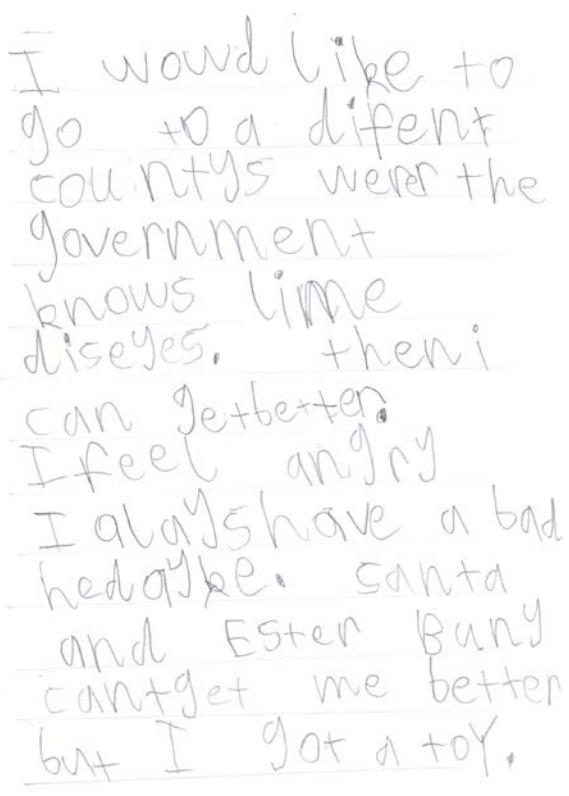

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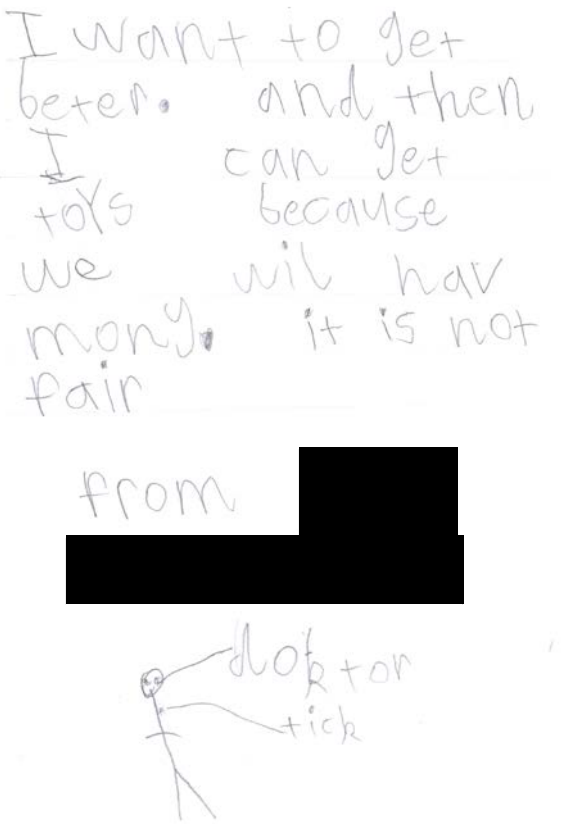


Translation of letter

"The government is a really bad government. People from a different country can follow the laws from over there because the government is not in charge of other countries except this one.

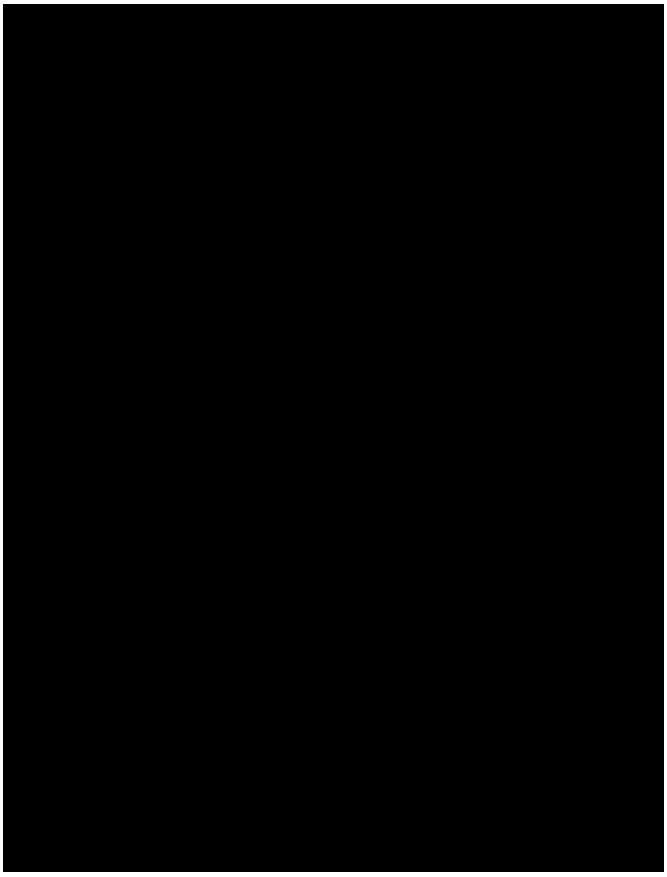
I would like to go to a different country where the government knows Lyme disease. Then I can get better.

I feel angry, I always have a bad headache.

Santa and Easter bunny can't get me better but I got a toy. I want to get better. And then I can get toys because we will have money. It is not fair.

From [redacted]"

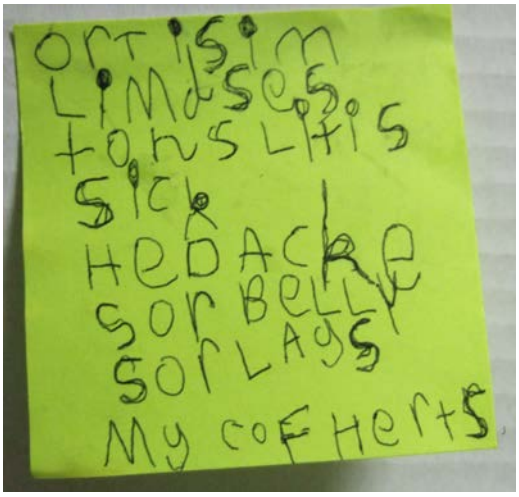
Illustration of a doctor being bitten by a tick.



Rash on face every 4 to 6 weeks lasting 2 weeks.
Over a year of these rashes. Now resolved (babesia?).

Post it Note List

List found 1st June 2015. Age 5 yrs.



"Autism, Lyme Disease, Tonsillitis, Sick, Headache, Sore Belly, Sore Legs, My Cough Hurts"

5.2 Diagnosis

Medical Diagnosis:

Laboratory	Test Type	Result	Comment on Result
Australian Biologics	Borrelia Mikrogen recomLine Immunoblot	IgM Negative IgG Borderline	Results indicate an immune response to borrelia falling exactly on the lower limit of the detection range for IgG (late stage of infection)
Australian Biologics	Borrelia Serum Culture PCR	Equivocal	DNA of bacteria present in 2 samples, not present in 2 samples. Retest recommended.
InfectoLAB	Borrelia Elispot-Lymphocyte-Transformation Test	Borderline	Diagnosis Borrelia. Borderline means that values were at the lower limit of the detection range.
InfectoLAB	Immune System CD-57 Flow Cytometry	Positive	The CD57-cell-count indicate a chronic immune-suppressive situation which may be caused by Borrelia.

[redacted] has two positive and one equivocal test result for borrelia.

Additonally, he had positive test results for the following:

- Babesia Duncani (IGeneX)
- Bartonella Henselae (Medicare Test)
- Yersinitis

Disability Diagnosis:

Autism Spectrum Disorder
Attention Deficit Hyperactivity Disorder
Sensory Processing Disorder
Visual Processing Disorder

Medical Symptoms:

- Intermittent fevers
- Bone and nerve pain
- Frequent headaches
- Allergies (numerous to food and medicine)
- Night Sweats (now resolved)
- Facial Rash (now resolved)
- Gastroesophageal reflux disease (now resolved)

5.3 Travel History

[redacted] has never travelled outside of Australia. Either the borrelia, babesia duncani and bartonella henselae is locally acquired or it is congenitally acquired. Mother's medical record and [redacted] medical record indicate congenital transmission.

5.4 Mother's Statement

Please read [redacted] extensive medical history. [redacted] has never known good health or a carefree childhood.

[redacted] brother has autism and all his family members are sick with borrelia and assorted tick borne infection. In the early years following his autistic regression, most of [redacted] time was spent playing therapy games, learning sign language, doing occupational therapy exercises, literacy and numeracy tasks and going from therapy appointment to medical appointment. It was, and is exhausting for him and the whole family, particularly in the context that his brother was also attending many medical appointments and therapy sessions.

[redacted] was taken to see over 20 doctors and specialists including Peadiatrician, Gastroenteroligist, Metabolic Specialist, Allergy Specialist and a Neurologist. Not one of these medical professionals considered borrelia, babesia or

bartonella in their differential diagnosis. Many times when presenting repeatedly to hospital with spiking fevers he was dismissed without cause found, and with no follow up. He stopped breathing multiple times in his sleep every night for the first two years of his life, often after being notified by the baby breathing monitor and we had moved him so that he would breathe, he would visibly stop breathing again before we had left the room. This was traumatising. [redacted] would projectile vomit typically in excess of 5 times per day upon expectation of food. He would get excited about his meal and then projectile vomit before eating. The eating disorder clinic and paediatric gastroenterologist had never seen anything like it before.

If [redacted] had been observed in hospital for just a few days perhaps one of his infections may have been identified. All disability diagnosis were organised privately, the public health system did not have professionals employed that were able to accurately assess [redacted] or provide appropriate intervention. He was not considered to be entitled to government therapy as he did not present with two problems (speech plus occupational therapy). It was the opinion of the government occupational therapist that his development was satisfactory. An occupational therapy assessment undertaken the month prior by a private occupation therapist proved significant developmental delays. There was not a good chance that the public system would have resulted in a timely autism diagnosis to warrant appropriate intervention.

[redacted] has been seen by numerous allied health professionals (eating disorder clinic, psychology, occupational therapy, speech therapy, audiologists, optometrists).

[redacted] tick borne infections were identified by his parents, not by his doctors, and testing was requested by his parents. Likewise, [redacted] autism and related developmental diagnosis were identified by his parents, not by his doctors and his parents organised diagnosis and intervention. [redacted] has significant developmental and social issues resulting in an ASD ADHD diagnosis and has associated behavioural presentation. The Government State School System has provided him with exactly ZERO teacher aide hours per week. The family pays for tuition at home when able.

The healthcare, education and disability support systems have been of very limited benefit to [redacted]

5.5 Medical History

0m to 3m – Allergies, Allergic colitus, Stops Breathing in Sleep, GORD, Infections

- 03/12/2009 - Very unsettled baby. Born full term caesarean section due to dubious placenta position and retro placental haemorrhage that occurred when baby gestational age 14weeks and continued to bleed thought pregnancy.
- 14/12/2009 – Breast Fed. Diagnosed allergic colitis by paediatric gastroenteroligist (musus and blood in stools). Pathology shows numerous leucocytes despite being breastfed without mother consuming gluten, dairy, fish, egg, limited soy. Stopped breathing in sleep numerous times (up to 10 times per night). Had to be shaken away to restart breathing. Gradually worsening over a 2 week period.
- 31/12/2009 – Hospital for two nights. During this time allergic colitis confirmed stool samples leukocytes. Intolerance to chick pea..
- 13/01/2010 – Allergy specialist [redacted]. Prick testing did not show allergy, refer report. Changed from breast milk to amino acid based Elecare formula. Given Gaviscon infant to help with reflux.
- 9/01/2010 – Peadiatric review. GORD (Gastro oesophageal Reflux Disease) by peadiatic gastroenterologist.
- Problems with stopping breathing in sleep ongoing. Baby breathing monitor used. Often Coughs/Struggles for breath in sleep.

3m to 7m - Numerous Infections

- 07/03/2010 – Dr [redacted]. Bilateral Middle Ear Infection. Amoxicillin 100mg/ml
- 24/06/2010 – Vomiting, decreased fluid (60ml in 24hrs). [redacted]. Infection not identified, viral infection assumed.
- 25/06/2010 – Dr [redacted]. Viral Illness assumed.
- 27/06/2010 – [redacted]. Breathing problems. Diagnosed ear infection left middle. Antibiotics prescribed.
- 28/06/2010 – Dr [redacted]. Ongoing left middle ear infection and tonsillitis. Amoxicillin w Clauvlanic acid 400mg-57ml.
- 29/06/2010 – Dr [redacted] Still very ill, some improvement.
- 2/7/2010 – After hours GP Dr [redacted]. Ears and throat red, no sign of active infection, Pustules over chest, back and arms (Macropapular rash). Antibiotics ceased as doctor thinks it is reaction to penicillin. Improvement over the weekend.

7 months to 1y – Unknown Illness, Grommet Surgery, Autistic Regression

- 08/07/2010 - Dr [redacted]. Temp>39C. Ongoing fevers 2 weeks. No obvious infection. Urine test non-specific. Referred to paediatrician for urgent assessment. Paediatrician not available at hospital. Prescribed EMycin 200.
- 08/07/2010 - Admission to [redacted]. On and off fevers 2wks commencing with what was thought to be viral flu. Otitis medial and tonsillitus. Rash and diarrhoea, high temperatures, leukocytes, thought to be urinary tract infection. No bacterial infection cultured. No real further action, continuation of antibiotics he was already taking.
- 08/07/2010 – [redacted]. Urine and stool tests. Not able to ID source of infection. Ears and throat a little red, tonsils enlarged. Antibiotics started to work? – fever gradually reduced. Refer record. Viral illness?
- 19/07/2010 – Dr [redacted]. Complete Digestive Stool Analysis, Bloodwork, Intestinal Permeability, IgG, Urine comprehensive profile done. Subsequent Hair analysis shows very high levels of antimony, arsenic bismuth, cadmium, gadolinium, lead, tin. Nutrient elements are below reference range for calcium, magnesium, sodium, zinc. IgG 93 Food sensitivity panel tests Rice 4+ (very strong positive), Cashew nut 1+ (mild reaction). Mother questioned as to what she was

exposing him to in relation to the high heavy metals in [redacted] and his brother. Greatly upset, mother organised expensive testing of house dust and soil outside house to identify a heavy metal source. None was found.

- 21/07/2010 – Grommet insertion left and right ears. Refer record. Following surgery, [redacted] regresses very obviously, losing motor function and the ability to do things he could previously. All babbling stopped and he became mute. [redacted] given: Sepaflurane (gas) fentanyl (narcotic) clonidine, dexamethasone, paracetamol.
- 24/07/2010 - Admission to [redacted]. Lethargic, feverish, decreased fluid, vomiting. Brother had URTI. No action taken.
- 28/07/2010 – negative celiac disease serology, low serum bicarblne, elevated aST, high calcium, low globulins, low cholesterol. Iron studies and serum Vit B12 unusual, Free T3 high, igA high, , low normal vit D.
- 24/09/2010 – Dr [redacted]. Blood and urine and biochemical parameter. Pyrrole 7.5, Histamine 0.7. No paediatric parameters for comparison. Zinc Ok, Vitamin D low normal range. Refer records.
- 26/10/2010 – [redacted], developmental physiotherapist assessment. [redacted] not crossing midline or self righting (head tilt). Therapy program put into place.
- 08/11/2010 – Speech Pathology Assessment [redacted]. Delay of expressive communication noted, normal social and comprehension.
- 01/11/2010 – Australian hearing audiology assessment. Sufficient hearing for normal speech and language development.

1y - Anaphylaxis, cause unknown

- 4/12/2010 - Anaphylaxis. Treated by ambulance adrenalin and taken to hospital. Blistering top and bottom lip, swollen lips and tongue and cheeks, rash over chest later developing. Refer hospital records. Thought to be due to peanut shell found holding. Epipen prescribed. Later appointment with Allergy Specialist ruled out peanut as a cause. Cause still unknown.
- 8/12/2010 and 9/12/2010 – Subsequent visits to Dr [redacted] as [redacted] continued to puff up in the face for no apparent reason.

1y to 1y2m - Yesinia

- 06/01/2011 – Low grade fever (below 38.5C) and cough. “Bronchial” cough still persisting, no discharged mucus from nose or throat evident.
- 17/01/2011 – Watery mucousy frequent bowel motions with fevers above 38.5C not responding to panadol.
- 20/01/2011 – Assessment by [redacted] District Health Services Children’s Community Therapy Service. Speech, Occupational Therapy and Physiotherapy. Refer to report. Expressive language and speech development delayed.
- 21/01/2011 – Chest Xray to rule out Pneumonia as fever source. Chest clear. Stool sample and urine pathology. Test results back on 24/01 show infection of Yesinia.
- 22/01/2011 – Vomiting diarrhoea.
- 24/01/2011 – Antibiotics commenced. Bactrim Sulfamethozazole and Trimethoprim 200/40mg in 5ml.
- 25/01/2011 – Fevers in excess of 38.5C ongoing with diarrhoea up to 15 motions per day. [redacted] advises that [redacted] be admitted to hospital on 27/01 if fever does not resolve.
- 26/01/2011 – [redacted] to hospital dehydrated and vomiting up antibiotics, Jaundiced in appearance. Ondansetron administered and fluids given.
- 27/01/2011 – stool sample shows infection Yersinia. High Fever 39C. Antibiotics Bactrum prescribed.

1y2m to 1y 6m - Recurrent Infections, Developmental Delays ongoing

- 07/02/2011 – Low grade fever. Sores on knees which lasted over a month. Culture negative.
- 20/02/2011 – Profuse discharge right ear. Ear infection. Left grommet in, right unable to be seen due to discharge. Bactrum antibiotics prescribed.
- 24/02/2011 – Ear infection follow up. Scar evident on right ear. No grommets obvious in either ear.
- 2/3/2011 ongoing infections in ear and TM perforations prompts doctor to refer to hospital for admission for possible IV antibiotics, paediatric and ENT review.
- 16/03/2011 –Bilateral middle ear infection, right ear drum on verge of rupture, rupturing later that day profuse discharge. Keflor 125mg/5ml.
- 13/04/2011 – Chest infection. Keflor 125mg/5ml. Ceased 17/4/2011 vomiting frequently – query reaction to colourE127 in medication.
- 19/04/2011 – Profuse discharge from Left ear. Both grommets sitting down base of ear canal, grommet possibly blocked with wax plug and infection forced out, causing profuse discharge – not consistent with observations on 24/02. Antibiotics Keflor 125mg/5ml.
- 21/04/2011 – Fever. ‘Bacterial’ infection of the back of the throat developed since 19/04 despite Keflor.
- 23/04/2011 – Speech therapy review [redacted]. [redacted] restricted to making five sounds (a, e, da, ma, na) which do not require lateral tongue movement and appears unable to move tongue laterally. Consistent with vomiting and restricted food textures. Receptive language further developed than expressive language. Delays evident in expressive language. Sign language (makaton) to be learnt.
- 27/04/2011 – Assoc Prof [redacted] allergy specialist. Prick test revealed no peanut anaphylaxis. Unknown anaphylaxis. Metabolic testing ordered. Refer record.
- 30/04/2011 – Brisbane doctor – fever, food refusal, pulling at ear. Right ear infection and throat infection diagnosed. Antibiotics Keflor 125mg/5ml.
- 02/05/2011 – “The doctors at [redacted] Clinic” – Keflor ineffective. Condition worsening. Right ear infection and tonsillitis. Doctor reluctant to administer more oral antibiotics and referred onto hospital for review. Refer record.
- 03/05/2011 – Hospital reviewed case, administered septrin Trimethoprim Sulphamethaxazole 40mg – 200mg/5ml.
- 05/05/2011 – Dr [redacted]. Improvement in tonsils and ears noted. Antibiotics continued, run out on 08/05.
- 09/05/2011 – Paediatric Neurologist and also a Metabolic Specialist (RCH Brisbane) Dr [redacted] seen to determine underlying reasons for regression following anaesthetic, unusual development and health concerns. Ruled out urea acid cycle dysfunction.
- 10/05/2011 – Dr [redacted] Medical centre. Tonsillitis with sheet of pus over both sides, swab taken. Rulide (Roxithromycin 50mg) antibiotics.

- 12/05/2011 – Dr [redacted] ENT. Tonsillitis. **Recommends tonsillectomy, adenoids out and** bilateral grommet insertion. Pathology ordered.
- 12/05/2011 - Telephone consult Dr [redacted]. Testing MTHFR (not detected), high lactate, other blood work done.
- 13/05/2011 – Dr [redacted] Medical Centre. Tonsillitis indiscreet pustules
- 16/05/2011 – Dr [redacted] Medical Centre. Tonsillitis is clear. Antibiotics continued for two more days.
- 20/05/2011 – [redacted] Medical Centre. Tonsillitis. Rulide 50mg 1 course followed by 0.5 tablet per day up until time of surgery.
- **Provision of further ENT surgery denied despite infections listed below. Local anaesthetists decline to provide anaesthetic services due to prior regression following anaesthetic. Instead GP continued to dose with long term low dose antibiotics (erythoromycin rulide) for approx. 18months. This fixed the situation.**

SUMMARY OF INFECTIONS 1y2m to 1y6m
5 Ear infections – 20/02, 16/03, 19/04, 30/04, 02/05
3 Eardrum perforations – 20/02 right, 16/03 right, 19/04 Left
3 Tonsillitis – 02/05, 10/05, 20/05
2 Throat infection – 21/04, 30/04(query tonsillitis)
1 Chest Infection -13/04
Total antibiotics – 8 total courses in 13 weeks. Thought allergic to penicillin and resistant to Keflor.

1y 6m to 2y - Developmental Delay, Eating Disorder

- 12/09/2011 – [redacted] District Health Services Children's Community Therapy Service review.
- Coughs and vomits prior to eating or when thinking of food (eg after you say lolly, he runs to the kitchen and vomits waiting expectantly). Does not handle textures, does not seem to swallow normally. Problems observed in hospital by feeding clinic and by Paediatric gastroenterologist who has not seen anything like it before and would have not believed it if it had not been for the extensive video footage
- 28/09/2011 – Letter to GP from Gastroenterologist and feeding clinic in relation to vomiting in expectation of food. No follow up, thought to be behaviour in response to food expectation.
- 23/11/2011 - [redacted] District Health Services Children's Community Therapy Service review Speech assessment. Receptive language high average but "severely delayed speech sound development suggesting significant motor speech disorder". This is despite normal hearing.

2y to 3y – Delayed and uneven development, Therapy, Illness

- Numerous and various infections over the year, many unidentified despite doctors consultations.
- Speech Therapy 18 speech therapy appointments over 7.5months largely with [redacted] District Health Services Children's Community Therapy Service yielding no improvement in severely delayed speech. Told by therapist it was unlikely he would ever talk. Took him out of this speech therapy and taught him sign language.
- 01/02/2012 –Occupational Therapy Assessment finding issues with motor proficiency gross and find, motor planning, body awareness, sensory processing, activity endurance sensory modulation ant gravitational positioning and postures and development grasps. Report recommending diagnosis of Pervasive Developmental Disorder Not Otherwise Specified under DSM IV.
- Nov 2012 – Gastro symptoms persist for many months following what was thought to be a viral gastro infection. Referral to gastroenterologist.

3yrs to 4yrs - Therapy, Infections, Diagnosis PDD-NOS

- 02/2013 - Dr [redacted]. Bloods taken. [redacted]. Specialist supplementation program.
- 14/03/ 2013 - Report by private occupational therapist to paediatrician confirming ASD symptoms in accordance with DSM V criteria.
- 15/03/ 2013 - [redacted] Diagnosed Pervasive Development Disorder, - Not Otherwise Specified by Paediatrician Dr [redacted]
- 29/03/2013 - [redacted] has acquired 83 signs and is using his mouth to make noise. [redacted] District Health Services Children's Community Therapy Service report speech sound development is severely delayed for age. Refused to provide further speech services as he needed two areas of difficulty in order to quality for assistance under their service and they considered his OT assessment they did to be satisfactory. It was obvious he had serious OT issues and I had a private OT report stating so. They were not provided with private OT report as there was little point continuing therapy with a therapist unable able to identify rather evident delays.
- 30/04/2013 [redacted] Speech Pathology, Speech therapy assessment. Expressive language not able to be assessed due to production difficulties. Age appropriate receptive language. At home program with tutoring by speech therapy students 4 times per week for next two years.
- Tutoring at home 4 times per week (OT, Speech). Speech therapy fortnightly and OT weekly for a year and a half.
- 27/05/2013 – Metabolic Specialist Review
- Coxsackie Virus infection
- Whooping Cough
- Influenza Type A and several other infections (do not have a copy, but all but are on GP files and can be obtained)
- 09/2013 – [redacted] Speech Pathology, Speech therapy review.

4 to 5yrs – Infections, Therapy, Developmental Assessment ASD

- Molluscum contagiosum and several other infections this year. On GP File.
- Continuation of intensive therapy at home and with speech and occupational therapists.
- 13/12/2013 - Chest Xray.
- 16/12/2013 - Occupational therapy report listing areas of developmental concern with recommendation to continue therapy.

- 07/01/2014 – Speech Pathology Assessment. 96% for receptive language, 95% for expressive language. After years of intense therapy. Speech therapy reduced to follow ups as required to ensure comprehension on track.
- 16/07/2014 – Occupational therapy report showing very uneven profile of development and sensory processing anomalies. Visual sequential memory <1 percentile. Other aspects range from 31st to 84th percentile.
- 20/11/ 2014 - Full clinical psychological assessment. Recommended diagnosis under DSM V of Autism Spectrum Disorder without intellectual impairment severity level 2 "Requiring Substantial Support" for categories of social communication and restricted, repetitive behaviours. Other clinical concerns include generalised anxiety (meltdowns, strong presence of both motor and vocal tics) and very underdeveloped executive function (early indicators present for ADHD, difficulties transferring intellectual potential into academic achievement (learning difficulties). Statistically significant gap between IQ and EF.

5y to present - Diagnosis ASD, Therapy, Borrelia, Bartonella, Babesia infections.

- 11/12/2014 – Occupational Therapy letter to school regarding support requirements.
- 10th Feb 2015 – Paediatrician removed PDD-NOS diagnosis and replaced it with a diagnosis of Autism Spectrum Disorder under DSM-V.
- 2014 to 2015 – Implementation of a lower starch diet (in addition to no additive, no gluten, soy, dairy, low salicylate and other allergens excluded). Molluscum contagiosum disappears. Sleeps much better though night. Generally health improved however extremely volatile with periods of rage (violent and angry behaviour) several times daily. Periods of extreme rage lasting for up to two hours. Every night heavily sweats to soak the bed. Regularly has fevers up to 38C without any cause, spontaneously resolving. Every 4 to 6 weeks base of face swells and burns and peels. No allergy identified.
- 2015 – Commences prep year at school. Occupational therapy still fortnightly.
- Diagnosed with visual processing disorder (eyes work, brain does not work so well). Trifocal glasses prescribed to assist with insufficiency.
- Diagnosed with ADHD.
- March 2015 – Speech Therapy to correct comprehension of direction issues.
- April 2015 – Diarrhoea. Spiking fevers up to 39C intermittent. Sore red throat. Headache, Sore legs and arms. Dr [redacted] prescribes Erythromycin antibiotics for suspected bacterial infection. Antibiotics subsequently ceased due to infection not responding (Dr [redacted]). Suspect viral infection.
- 12/05/2015 – Immunoblot IgG Test for Borrelia burgdorferi show positive test result Australian Biologics
- April to June 2015 - Bacterial gut infection Dientamoeba identified
- June 2015 to August 2015
    - Positive pathology for bartonella henselae infection
    - Positive pathology for active babasia duncani infection (IGeneX)
    - Additional positive pathology for borrelia infection (active infection – direct testing positive) from InfectoLAB Germany

END OF MEDICAL RECORD

6.0 PERSONAL ACCOUNT [redacted]

6.1 Letter

Letter from [redacted], Age [redacted] years.

Dictated letter from [redacted], dysgraphia prevents him from writing. Verbal skills previously assessed as savant. Dictated as spoken on 07/04/2016 at 3:12pm. Prompting statements : Tell the government who you are, why you are writing and what it is like to have borrelia.

Dear Government,

I am an Australian boy and I have borrelia lyme disease and it is the worst thing you can imagine. Imagine if you were me getting no medical help. Doctors being defiant and saying "no it doesn't exist" when you have got so much evidence. Why aren't you recognising it? You are being controlled by evil people. Why are you wasting Australia's money on cancer research when lots more kids like me have autism because they are sick? I like being autistic but I don't like being sick. All the kids with autism I know are sick and the doctors don't try to make them better or find out why they are sick they put them on drugs that make them extremely strange and don't make them better. When I go to the disabled children's Christmas party most of the kids are zoned out and can't play.

I was born feeling sick, I have always been feeling sick and I have never felt what it is like to be well and I think that feeling horrible is feeling normal and it is your fault that I don't get better. And you took away my teacher aide hours. I am barely coping and I am failing at almost everything. Last year I had help every day and now I have one aide hour every week. I can infect lots of people at my school just by one mosquito biting me or some head lice biting me. At my school there are lots of mosquitos and head lice.

I should not have been disabled, I should have been a normal kid. It is the doctors and your fault that I am disabled and sick. My arm fasciculates and my bones hurt and it feels like it did when I broke my arm. I have lots of trouble sleeping. I have ADHD and I have terrible trouble concentrating. Imagine having microscopic spirochetes drilling through you killing you.

It is horrible watching my Mum have seizures almost every morning. I thought the neighbour was getting taken to hospital but when the medic came in the hallway I looked into my Mum's bedroom and she was having a seizure, a big one. I got scared to the bone. I do not want Mum to die. Once I could not get to school because my Mum was having a seizure and I was the only one to help.

I don't have a play station or any gaming console and there is just concrete on the lounge room floor and I never get to go to Dreamworld or anywhere exciting, when I go on a plane it is to see doctors far away. I have had lots of needles and they are scary.

When I grow up I am going to write a lot more senate submissions that are a lot stronger than this one. You should not be treating people like lab hamsters.

Yours truly,

[redacted]

[redacted]

6.2 Diagnosis

Medical Diagnosis:

[redacted] has 3 positive test results for borrelia:

Laboratory	Test Type	Result	Comment on Result
Australian Biologics	Borrelia Mikrogen recomLine Immunoblot	IgM Borderline IgG Positive	Diagnosis Borrelia. Values well within detection range for IgG and at the lower limit of the detection range for IgM.
InfectoLAB	Borrelia Elispot-Lymphocyte-Transformation Test	Positive	Diagnosis Borrelia. The Elispot indicates a cellular activity against Borrelia.
ICPMR Westmead	Borrelia ELISA	Positive	Antibodies Detected. Not proof of active infection, just of immune response to those markers test can detect.

Additionally, he had positive test results for the following:

- Mycoplasma pneumoniae (Medicare Test)
- Bartonella Henselae (Medicare Test)
- Yersinitis
- It is suspected that he has babesia duncani - no pathology testing in Australia, and IGenex Tests not ordered.

Disability Diagnosis:

1. Autism Spectrum Disorder (DSM V)
2. Attention Deficit Hyperactivity Disorder (DSM V)
3. Disorder of Mathematics (Dyscalculia) (DSM V)
4. Anxiety Disorder (DSM V)
5. Sensory Processing Disorder
6. Visual Processing Disorder (encompassing Dyslexia)
7. Central Auditory Processing Disorder
8. Dysgraphia

Medical Symptoms:

- Muscle Fasciculation
- Intermittent fevers
- Bone and nerve pain
- Frequent headaches
- Fatigue
- Reactive Arthritis in hips and knees
- Allergies (numerous to food and medicine)
- Partial Bowel Obstructions (now resolved)
- Gastroesophageal reflux disease (now resolved)

6.3 Travel History

[redacted] has never travelled outside of Australia. Either the borrelia, mycoplasma pneumonaie, bartonella henselae and babesia duncani (likely) are locally acquired or it is congenitally acquired. Mother's medical record and [redacted] medical record indicate congenital transmission.

6.4 Mother's Statement

In his [redacted] years of life, [redacted] has had more pain and sickness than most. He has never known good health. Refer to medical summary in Section 6.5.

To aid in interpreting the letter he wrote (Section 6.1). [redacted] regularly faces discrimination, for his disability, for his medical condition and for the financial situation the inequity in education, disability and medicine has forced on our family.

In the early years of school, he was too unwell for over a term every year to attend. He required full time teacher aide to attend school such was his condition. The family unwisely paid for this teacher aide, which cost approximately $15,000 every year for 3 years. Now [redacted] with his significant disability is in the state school system where he receives just 1 teacher aide hour every week. This assistance is not sufficient to enable education, or even attendance without serious disadvantage including to the rest of the class.

When 7, [redacted] was pinned to the wall and threatened by a male parent at school in front of a dozen other parents who did nothing to stop the abuse. When I intervened (this was the year I was struggling to walk) I was ridiculed and verbally abused, to the approval of the onlookers, but the parent was allowed to continue attending school. This incident encouraged others, and for the last four weeks of [redacted] time at that school, every week he received physical injury from bullying.

We moved [redacted] to a state school where [redacted] suffered such severe abuse in the four weeks he attended that it was been reported to the Child Protection Unit by his psychologist, but again, nothing was done to the perpetrator. [redacted] was so emotionally damaged he could not attend school for a following term.

[redacted] has had several operations for grommet insertion, tonsillectomy and gastroscopies. This has included emergency surgery. There was an autistic regression between the age of 1 and 2 years as evidenced in video, and this regression was a gradual loss of function.

From the time he was born, [redacted] did not sleep more than 6 hours in a 24 hour period, and at 20 minute increments only. He would sleep only if held upright. This was exhausting and continued up until age 3.5yrs . He cried nearly continuously and after gastroscopy was diagnosed with Gastroesophageal Reflux disease. He would reliably vomit several times per day.

[redacted] was breast fed for 16 months and he refused food and fluid past this point. He was presenting with failure to thrive when he was prescribed amino acid based formula, which he promptly rejected. There was a period of two years when the only fluid he had would take had to be administered by syringe barrel into his mouth, 10ml at a time. [redacted] would regularly refuse food for periods of 1 week (not even a teaspoon) and one two occasions up to 3 weeks (not a single teaspoon of food).

[redacted] would not / could not drink or eat due to sensory issues. If he did manage to eat, food was restricted to the one texture or the one type only. He did not eat at meals and would take food only if he was distracted (sensory issue) in small quantities. Management involved following him for hours every day placing minute parcels of food into his mouth and hoping that he would be able to tolerate / not vomit.

Up until he was age 4.5 he could not detect hunger or thirst and up until he was 7 he could not detect other bodily functions and he did not sleep through the night until he was 9 years of age.

[redacted] has suffered numerous stomach ulcers and partial bowel obstructions (now resolved). He had considerable gastrointestinal tract problems including infection and bacterial imbalances (now resolved). Since birth he has had numerous (too many to count) infections of unknown origin causing rash and spiking fevers that spontaneously resolve.

[redacted] regularly has fluid pooling in his hip and knee areas (reactive arthritis). On one occasion he was unable to walk for a period of days.

[redacted] is an extremely intelligent child and is quite aware of the hopeless nature of the situation when he attempts to describe the symptoms of sensory disorder and nervous system dysfunction to adults charged with, and responsible for care and appropriate treatment (teachers, medical professionals). When he does manage to communicate the medial issues/conditions, they are frequently met with a lack of experience or lack of belief or lack of willingness or all three. Historically these medical issues / conditions were dismissed on the basis of autism or more frequently, dismissed on the basis of bad behaviour and discipline deemed appropriate. Since borrelia, mycoplasma and bartonella diagnosis, he can provide an explanation most adults grasp.

The forced awareness of the world and his position in it, his high intelligence and his great disability have resulted in anxiety and a different interpretation of the world than most children of his age. He has risen above the humiliation and is exceedingly brave and is frequently the first child to help another. This is not the first letter [redacted] has written to the Government regarding disability. He hopes that one day he will have the same opportunities to medical care, education and inclusion in society as others do.

6.5 Medical History

This is a condensed version of a lengthy medical history

Prebirth –10 days overdue, non progressive labour with hyper-stimulation reaction to oxytocin 40min long contractions

0 to 6m –Very unsettled -less total of 6hrs sleep in 24 hour period, near constant crying when awake, allergic colitis, GORD, allergy to pepti junior (partialy hydrolysed formula). Breastfed on exclusion diet, gaviscon for reflux.

6m to 1y – Sleep issues ongoing, Allergy specialist, Nutritionist, refusal to eat, reaction to Zantac (petit mal seizure), unidentified 'viral' infections, neutropenia, reflux managed on Losec. Chiropractic and osteopath large adjustment with very marked improvement in gross motor. Frequent extended complete food refusal (up to 3wks duration) – food clinic, mineral deficiencies, Grand mal seizure reaction to Ibuprofin, Iron deficiency.

1y to 1.5y – Sick more often than well (4 x ear infections, 2 x tonsillitis, 3 x 'viral' illness). Feeding clinic, complete food refusal, gastroscopy, breast milk dried up, allergy to cefaclor monohydrate.

1.5 to 2y - grommets inserted bilaterally, subsequent infection and rapid decline in health resulting in emergency surgery to removed infected grommet and reinsert. Conjunctivitis, chest and sinus infection showing Moraxella catarrhatis. Extended periods of food refusal waking in excess of 10 times a night, multiple respiratory infections requiring antibiotics. Developmental delays.

2y to 2.5y – Norovirus and giardia simultaneously for 6 wks, sleeping and eating issues continue, several episodes of unknown 'viral' illness and tonsillitis.

2.5y to 3y – Developmental assessments results ADHD, ODD, ASD with IQ 125. Diagnosed with sensory processing disorder. Behaviour improved with diet changes. Grommets grow out, 2 x partial bowel obstructions, several infections of tonsillitis and middle ear. Numerous antibiotics.

3y to 4y – Diagnosed ASD & ADHD. Elevated copper, very low zinc and poor methylation processes. Partial bowel obstruction, gastroscopy, ongoing reflux without structural cause. Brother [redacted] born, 2 x helpers hired to supervise life endangering behaviour. Medication attempted - Ritalin causing seizures and weight loss and Strattera increasing psychotic symptoms. Diagnosed with Visual Processing and Auditory processing problems and receives targeted intervention for eating disorder. Tonsillectomy and grommets inserted bilaterally, stress ulcer following surgery. Serum Cu and Zn normalized following supplementation. Oxytocin for sleep issues. Started AEIOU.
Applied Behavioral Analysis implemented to assist with eating disorder.

4y to 5y – Under methylation and pyrroluria treated accompanying remarkable improvement. Self harm decreases, concentration, fine motor, sleep and appetite improve. Grommets out, several ear infections. Constant low grade fevers of unknown origin sometimes with accompanying rash regularly spiking to 39C unknown cause. Reactive arthritis in left hip from Yersinia infection. Mitochondrial testing yields no answers.

5y to 9 yr – Diagnosed Central Auditory Processing Disorder, Sensory Processing Disorder, Visual Processing Disorder (incl. dyslexia), Dyscalculia, Dysgraphia, Generalised Anxiety Disorder, GERD. Significant heavy metals. Extended periods of illness (unknown origin), Targeted speech therapy, psychology, occupational therapy, and tuition. Multiple health issues too many to list. Partial bowel obstructions continue. Psychological damage from inappropriate schooling.

9yr to present: Dientamoeba infection. Positive pathology for Borellia, Bartonella and Mycoplasma p. Diet changes (possible only due to sensory issues improving) greatly improve general health and GI symptoms. Starts to sleep through the night. Problems with temperature regulation, muscle fasciculation, cramps, complains of heart problems intermittently. Intermittent fluid on hips causing mobility problems. Daily joint pains.

Attachment 7 Record of Meeting

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WITNESS: I am [redacted] I am a senior registered surveyor in the state of Queensland.

WHAT: I was present at a medical interview between Infectious disease specialist, [redacted]

WHERE: [redacted]

WHEN: about 3:30pm, Tuesday 26 May 2015

Description of Events:

This description is as I remember the events on the evening of the same day the interview happened.

Before the doctor's appointment, [redacted] prepared a summary of her medical history on three A4 sheets which I check read for her. She made a copy for the hospital. She also compiled a large binder of documentary evidence of her chronic illness and the many inaccurate diagnoses and unsuccessful treatments she had experienced over more than a decade. [redacted] took all these documents with her to the interview.

We entered the [redacted] at about 2:30 pm; registered, and then waited for about an hour and a half. [redacted] was called into the interview room by an intern doctor who reviewed [redacted] medical conditions summary and 2 lab test results from 'Australian Biologic Testing Services' that showed positive for Borrelia Spirochete bacteria.

The intern doctor performed an examination of [redacted] and arm strength by opposing her action. She also checked her reflex response with a round rubber hammer. The Infectious Disease doctor, [redacted] entered the room during this examination and read [redacted] summary for the remainder of the examination. The inter doctor reported verbally to KM that she found nothing unusual except for weak flexor muscles in [redacted] left side.

The doctor, KM, introduced herself as American with experience in Papua NG and discussed [redacted] summary with her clarifying the places [redacted] had lived in and had travelled to. She initially commented after reading the file that [redacted] had an interesting career. She took notes on the back side of the summary as [redacted] described episodes of her illness and drew a sketch to illustrate [redacted] description of a saw tooth descent into chronic sickness since about 2001. She also made a note of [redacted] comment that her career as a civil engineer 'died' with the birth of her extremely sick first son and annotated the saw tooth sketch and wrote 'career died' on the top of the page. The doctor asked about the various types of sickness. The following things were mentioned - skin, neuro-muscular, cardiac, kidneys, vision, cognitive, mouth and speech. [redacted] described what had happened and how variable these conditions were. [redacted] showed the doctor the test results for some of the diagnoses she had with her as documentary evidence and also explained her stance that further testing along these same lines would duplicate what was already done and documented; and was an unnecessary expense. The doctor was told that [redacted] had 2 sons with severe ASD and the all-out concerted attempt being made to reverse this condition by [redacted] and her husband, [redacted] was discussed. The doctor also understood that [redacted] has a low level of trust regarding the ability of doctors to successfully treat her based on a long experience of failures. [redacted] also informed the doctor that her neuro muscular condition greatly improved when she removed sugar and starch from her diet after discovering her sensitivity to them by self-performed blood sugar testing over several weeks.

The doctor asked about the origins of the lab results showing positive results to the presence of Borrelia bacteria in [redacted] blood. [redacted] told the doctor of her study into the research linking Autism

in children to mothers with auto-immunity disorders caused by bacterial stealth infections and how the lab doctor she had spoken with by phone at the 'Australian Biologic Testing Services' laboratory who was analyzing her blood for stealth infections had suggested she be tested for Borrelia bacteria. It was made clear that this testing was done at [redacted] own expense and with the consent of her GP. It was also made very clear to the doctor that [redacted] had travelled extensively overseas into remote SE Asian and Papuan jungles as well as the remote Torres Straight Islands where she potentially got infected by the Borrelia bacteria and coinfections that accompany it.

The positive results of the 2 tests for Borrelia from 'Australian Biologic Testing Services' were rejected outright by the doctor on the sole basis that she did not know the laboratory and so did not credit it's testing. [redacted] then showed the doctor the recently published 'Australian Chronic Infectious Disease Society Guidelines' for the treatment of Borreliosis, or Lyme Disease; and explained how well the descriptions matched every one of her symptoms. Astoundingly, the doctor rejected this authoritive document from the 'Australian Chronic Infectious Disease Society' outright, saying that she had no prior knowledge of ACIDS or the guideline document. The doctor made a note of the guideline document name and the version number. She said that 'these are not our people' and that she disagreed with their treatment methods. I was very surprised by that remark. [redacted] suggested that the doctor should do some professional development. The doctor pointedly asked [redacted] why she thought she had Lyme disease although to that point in the conversation the word 'Lyme' had not been uttered. [redacted] was both flabbergasted and angered by this question and answered that she had two positive lab results for Borrelia infection and a history of chronic illness that was described by the ACIDS guideline with a high degree of collation.

The doctor then told [redacted] that she did not believe she had Lyme disease, and because [redacted] had a belief that she most likely did have Lyme, then she could not help her. She said something to the effect that she believed Lyme to be different to what [redacted] described. The issue as I understood it was that the trust she considered necessary to exist between patient and doctor could not be established. The doctor repeatedly expressed her 'belief' stance but never gave a single reason for it. [redacted] asked her what that belief was and the doctor replied that it was in line with the USA's Infectious disease's guidelines. Note, the doctor was an American. She mentioned there was controversy regarding Lyme disease. [redacted] stressed that this was Australia not America and that it was unlikely that Australia had an imaginary dotted line surrounding it that Borrelia bacterial could not cross over. The doctor did not noticeably react to [redacted] remarks or respond to a logical example she gave along the lines that disbelieving that an obviously corroded structure in a desert could possibly be corroded on the basis that deserts have low rainfall. Neither did she respond to my comment that belief was subjective and did not necessarily reflect reality; nor that were the lab tests empirical measurement to be considered.

I asked the doctor what testing would be acceptable to her. Her response was 'North Ryde Hospital'. [redacted] said that the ACIDS guideline listed the laboratories with the capability to perform accurate testing for Borrelia and its coinfections and found this listing and showed it to her. North Ryde was not included but other major hospitals were. I pointed out to her that these were reputable mainstream institutions and explained the testing performed by 'Australian Biologic Testing Services' that analyses [redacted] blood was reputedly to be best practice; but there was no comment from her. She offered no reason for her belief that [redacted] did not have Lyme disease, nor made suggestion why she was sick, or offer to treat [redacted]. She did ask what [redacted] wanted her to do. [redacted] responded by requesting to be treated in accordance with the ACIDS guideline.

Soon after this point the interview finished when [redacted] terminated the conversation and left the room angry and disappointed, saying something along the lines that as she did not have Borreliosis then she could go and donate blood and be an organ donor. I remained in the room and spoke with the doctor for another 10 minutes to ensure the doctor understood what [redacted] has suffered and the reason for her bitter disappointment and anger with the medical profession generally. An experience obviously expanded by this interview.

I explained the heroic all-out effort that [redacted] were making for their sons to the point of exhausting their financial resources. I explained that their eldest son [redacted] was manifesting some similar symptoms to [redacted] condition about 3 years ago. I told her about the medical study both have undertaken in their quest to help their autistic sons and discover why [redacted] is so sick in so many ways.

I also wanted to understand what the doctor might have done for [redacted] and did not. I asked her what the harm was in an initial treatment of Antibiotics to address the likely coinfections that were discernable from [redacted] symptoms that must be addressed first before tackling the Borrelia according to the ACIDS guidelines. The doctor mentioned her non-acceptance of going on long term antibiotics and I agreed with the danger and ineffectiveness of that (note that this is not recommended by the ACIDS guidelines). I made her aware that I was conversant with the American Lyme treatment debate. She neither agreed to short term ABs nor suggested any other treatment. I was given to understand that testing from North Rhyde hospital was the only acceptable option but that was not directly offered.

I believe the doctor is cognisant of [redacted] plight and she did expressed sympathy for [redacted] numerous times. However, the outcome for [redacted] is a denial of treatment by the Public Hospital system. The doctor demonstrated a closed mind and fixed attitude regarding her 'belief' and rejected everything [redacted] provided as evidence of her condition. My efforts did not change her stance in the slightest. The doctor provided nothing to [redacted] in today's interview except more despair about ever being healed. The only reason for not providing aid was a difference of belief in what Lyme disease was. [redacted] provided extensive documentary evidence of her chronic illness; she provided empirical test results from a best practice laboratory; and she provided the doctor with an authoritive guideline that might well be a key for treating her. All these were disregarded on the basis of 'belief' that seem to me more political than scientific. As a witness to this interview, I think [redacted] was abandoned.

I finished my conversation with the doctor on a cordially note hoping not to jeapodise the possibility of a future more scientific assessment of [redacted] and her children.

The intern doctor was also witness to this interview.

I solemnly and sincerely declare that this interview took place and that I witnessed it. This description is as I remember the events on the evening of the same day the interview happened. I make this declaration conscientiously believing all I have recorded to be true.

[redacted] DATE