OUR MISSION: To advocate for the awareness of Trigeminal Neuralgia and related facial pain.
OURS GOAL: To have a unified understanding of Trigeminal Neuralgia and other related facial pain resulting in better pain management.
OUR VISION: An improved Quality Of Life.

AUGUST 2012

“Nothing great is ever achieved without much enduring.” ~ St. Catherine of Siena

“With ordinary talent and extraordinary perseverance, all things are attainable” ~ Thomas Fowell Buxton

Have you given it further thoughts?
A couple of support group leaders have expressed their wish to step down from their current duties come the end of the year. We are in the process of seeking suitable replacements. While we have a set of criteria for support group leaders, we also depend on individuals to express their willingness in taking on the onus. It need not be a sole custodian role; we can have co-host and hence share the responsibilities. However, if no one is willing to assist I am afraid we will just have to suspend the group. If you believe your support group is worth having, and that the support within the group is essential to TN sufferers, then please step forward or have a chat with your current SGL to see what this role entails; then contact me – Irene.

HELP WANTED: someone to produce our monthly newsletters. If you would like to give it a go please contact me.

HAVE YOU ANY FUNDRAISING IDEAS? We would love to hear from you. Or if you have a fund raising event that you wish to organise, please call me to discuss how we can get it off the ground.

I will be away August – September, grabbing a holiday before and after the IASP Congress in Milan. I will be checking my email periodically. Please use the tna_sydney@yahoo.com email to avoid jamming up the “tnaausstralia.org.au” account as it only has 10MB storage space.

MARK IN YOUR CALENDAR: our 5th National Conference will be held next year from 23rd – 25th August. Do start saving for it now if you haven’t already. The event would also mark our 10th Anniversary and it would be wonderful to have all our members there celebrating the occasion. Needless to say, we would also be bringing to you a fantastic meeting. You need to be there.

“A large oak tree is just a little nut that refused to give up.” ~ David McGee
~ Irene ~
Reduced Pain and Inflammation in Juvenile and Adult Rats Fed a Ketogenic Diet

David N. Ruskin1, Masahito Kawamura Jr2, Susan A. Masino3
1 Department of Psychology and Neuroscience Program, Trinity College, Hartford, Connecticut, United States of America, 2 Department of Pharmacology, Jikei University School of Medicine, Minato-ku, Tokyo, Japan

Abstract

The ketogenic diet is a high-fat, low-carbohydrate regimen that forces ketone-based rather than glucose-based cellular metabolism. Clinically, maintenance on a ketogenic diet has been proven effective in treating pediatric epilepsy and type II diabetes, and recent basic research provides evidence that ketogenic strategies offer promise in reducing brain injury. Cellular mechanisms hypothesized to be mobilized by ketone metabolism and underlying the success of ketogenic diet therapy, such as reduced reactive oxygen species and increased central adenosine, suggest that the ketolytic metabolism induced by the diet could reduce pain and inflammation.

To test the effects of a ketone-based metabolism on pain and inflammation directly, we fed juvenile and adult rats a control diet (standard rodent chow) or ketogenic diet (79% fat) ad libitum for 3–4 weeks. We then quantified hindpaw thermal nociception as a pain measure and complete Freund’s adjuvant-induced local hindpaw swelling and plasma extravasation (fluid movement from the vasculature) as inflammation measures. Independent of age, maintenance on a ketogenic diet reduced the peripheral inflammatory response significantly as measured by paw swelling and plasma extravasation. The ketogenic diet also induced significant thermal hypoalgesia independent of age, shown by increased hindpaw withdrawal latency in the hotplate nociception test. Anti-inflammatory and hypoalgesic diet effects were generally more robust in juveniles. The ketogenic diet elevated plasma ketones similarly in both age groups, but caused slowed body growth only in juveniles.

These data suggest that applying a ketogenic diet or exploiting cellular mechanisms associated with ketone-based metabolism offers new therapeutic opportunities for controlling pain and peripheral inflammation, and that such a metabolic strategy may offer significant benefits for children and adults.

Introduction

Pain and inflammation are hallmarks of diverse acute and chronic diseases. Chronic pain is one of the most commonly indicated health-related factors leading to poor quality of life and, across all cultures, patients with chronic pain have among the lowest reported quality-of-life scores of any medical condition. In parallel, accumulating evidence points to inflammation as not simply a consequence but an active contributor to pathologies such as atherosclerosis, stroke, metabolic syndrome and cancer. Without question, a great unmet public health need exists for safe, effective and non-addictive strategies to reduce pain and inflammation.

Dietary therapy has long been coveted as a strategy to treat a variety of clinical conditions, including pain and inflammation. For example, polyunsaturated fatty acids reduce nociception by activating peroxisome proliferator-activated receptors (PPARs), and olive oil polyphenolic compounds reduce experimental inflammation. In addition to specialized dietary approaches, chronic caloric restriction reduces inflammation in several models. Benefits of metabolic therapy are demonstrated unequivocally in disorders of amino acid metabolism (such as phenylketonuria), familial hypercholesterolemia, and disorders of fatty acid transport and oxidation. Overall, metabolism has clear effects on the central nervous system and a host of peripheral tissues, and strategies that exploit broadly the therapeutic benefits of metabolism are becoming more compelling in translational and clinical research.

Evidence is building steadily on the effectiveness of a ketogenic diet – a high-fat, low-carbohydrate regimen – in treating epilepsy, brain cancer, type II diabetes and neurodegeneration. For decades the ketogenic diet has been used successfully to treat epilepsy, particularly pediatric and medically refractory epilepsy, and its efficacy has been validated by a host of multi-center, retrospective and randomized, prospective clinical studies. The restricted carbohydrate content of a ketogenic diet minimizes glucose metabolism and increases ketolysis, i.e., the use of ketone bodies (acetone, acetoacetate, β-hydroxybutyrate) as alternate energy sources. Established cellular consequences and recently hypothesized mechanisms of ketogenic diet therapy coalesce to suggest that a predominantly ketone-based metabolism may reduce inflammation and nociception as compared to glucose-based metabolism.

To date, published data characterizing the relationship among ketogenic diets, pain and inflammation are limited. A pilot clinical study showed that a ketogenic diet reduced inflammation in non-alcoholic fatty liver disease, and a need for more research on this topic has been noted recently. Data characterizing ketogenic diets and pain are also limited, although the use of anticonvulsant drugs as antihyperalgesic/antiallodynic agents for neuropathic pain suggests that an anticonvulsant ketogenic diet might be effective in reducing pain. In the present study we evaluated the therapeutic
potential of a ketogenic diet directly by quantifying standard measures of pain and inflammation in juvenile and adult rats. We found that maintenance on an ad libitum ketogenic diet for three weeks attenuates thermal nociception and decreases a peripheral inflammatory response significantly in both age groups. These results indicate that metabolism-based strategies may offer new therapeutic opportunities with broad clinical implications.

Discussion

Here we demonstrate hypoalgesic and anti-inflammatory effects of a ketogenic diet. In juvenile and adult rats we show that ad libitum feeding of a ketogenic diet reduces nociception, as assessed by hindpaw withdrawal latency, and peripheral inflammation, as assessed by CFA-induced hindpaw swelling and plasma extravasation. To date the clinical applications of ketogenic strategies have focused primarily on its established success with pediatric epilepsy and emerging success with diabetes; recent translational research is expanding clinical implications to include brain cancer, brain injury, and Rett syndrome. New therapies are particularly urgent for pain, inflammation and inflammatory pain, and the present data suggest more translational research is needed for ketogenic diet therapy and analogous metabolic treatments.

There are a number of mechanisms thought to underlie the efficacy of ketogenic diet therapy, but an incomplete understanding of critical cellular mechanisms has hampered efforts to develop alternate pharmacological strategies and, in parallel, limited clinical predictions and applications of this type of metabolic therapy. However, published experimental research and hypotheses regarding the success of ketogenic diet therapy point to its clinical potential for pain and inflammation. With respect to central pain mechanisms and neuronal activity, ketogenic metabolism is thought to increase levels of adenosine and/or GABA, two powerful inhibitory substances in the nervous system, through augmented oxidative phosphorylation and shifted glutamate: aspartate aminotransferase equilibrium, respectively. There is abundant evidence that increasing central inhibition by activating adenosine A₁, GABA A or GABA B receptors produces hypoalgesia in acute pain tests. In addition to central mechanisms, a high polyunsaturated fatty acid content in ketogenic diets should enhance potassium conductances in peripheral neurons through PPAR activation. Therefore, we speculate that mechanistically-separate inhibitory processes in the central and peripheral nervous system could combine to mediate ketogenic diet-induced thermal hypoalgesia. Given the positive effects of adenosine and GABA agonists in treating chronic inflammatory and neuropathic pain and the central hyperexcitability in chronic pain, ketogenic diets might be especially effective analgesics/hypoalgesics for diverse types of chronic pain. The success of dietary therapy even in pharmacoresistant epilepsy suggests that it may also be effective for intractable pain.

In addition to decreased nociception, we show that pretreatment with a ketogenic diet reduces subcutaneous inflammation significantly in juvenile and adult animals. There are multiple possible mechanisms. Ketone body metabolism results in a decreased production of reactive oxygen species, known to contribute to inflammation. Adenosine acting through A₁ and A₂ receptor subtypes limits inflammation in a wide variety of peripheral and central tissues, including inflammation due to subcutaneous inflammogens. Polyunsaturated fatty acid-induced PPAR activation inhibits NFκB and AP-1, both pro-inflammatory transcription factors. It is possible that each is involved, and more research is needed to elucidate the primary mechanism underlying this peripheral effect. In addition to specific cellular mechanisms, overall protein restriction reduces inflammation in some situations, and caloric restriction is anti-inflammatory in general. All animals in this study were fed ad libitum, thus it is unlikely that caloric restriction is occurring in the present study in the adult animals, a group that showed no difference in weight but did exhibit significantly reduced inflammation (and nociception) on the ketogenic diet. Nevertheless, a combined calorically-restrictive and ketogenic diet may be even more effective against inflammation (and potentially nociception) than either dietary component alone; similar conclusions have been made concerning the anticonvulsive and anticancer effects of dietary treatments. Furthermore, although we used the ketogenic diet as a pretreatment, clinical evidence suggests that it can reduce pre-existing liver inflammation.

In the one published study of nociception and the ketogenic diet, Ziegler et al. described decreased (rather than increased) tail-flick latency in rats fed a ketogenic diet. Though both the hotplate and tail-flick involve thermal nociception, this difference may be related to methodological differences including rat strain, body part tested, diet composition, stimulus strength (latencies are generally longer in our study), and length of dietary treatment (12 wk in Ziegler et al.). The length of treatment might be particularly important, as a number of studies using several different measures have demonstrated non-monotonic effects of ketogenic diets at time scales of days to weeks. These variables should be examined in future studies, along with other pain modalities. Complementing our direct experimental evidence, multiple hypotheses regarding the mechanisms underlying the success of ketogenic diet therapy coalesce to suggest that this metabolic treatment will reduce pain and inflammation. Yet despite widespread interest in dietary therapies for pain and inflammation (and myriad diseases that implicate inflammation as either a cause or a consequence of the pathology) systematic study of ketogenic strategies as anti-inflammatory or hypoalgesic strategies is just beginning. Unlike myriad dietary regimens with limited or inconsistent

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Trigeminal Neuralgia Association Australia

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proof-of-efficacy, a ketogenic diet offers recognized and established clinical benefits. Accordingly, there is a focus on elucidating critical mechanisms underlying the success of ketogenic diet therapy in treating epilepsy as well as mechanisms underlying emerging benefits in clinical conditions such as diabetes, brain injury, brain cancer and Rett syndrome. The data presented herein suggest that ketogenic diets offer promising therapeutic potential for diverse inflammatory or painful conditions, across age groups, without the added difficulty of maintaining caloric restriction. Based on these results and many decades of clinical experience with diet-based therapies for pediatric epilepsy, a novel anti-inflammatory and hypoalgesic application of ketogenic diet therapy (or an analogous future pharmacological strategy) would be effective, non-addictive and relatively free of major side effects.

### Materials and Methods

Male Sprague-Dawley rats were bred in the Trinity College vivarium with animals originally purchased from Charles River (Storrs Mansfield, Connecticut, USA). All experiments were carried out in accordance with the NIH Guide for the Care and Use of Laboratory Animals and with approval of the Trinity College animal care and use committee. Either shortly after weaning at 21 d or as adults (85–110 d), matched groups of male Sprague-Dawley rats were switched to a ketogenic diet (AIN-76 Modified, High fat, #3666; Bio-Serv, Frenchtown, New Jersey, USA) or maintained on their standard diet (Purina 5001; PharmaServ, Framingham, Massachusetts, USA). Sprague-Dawley rats become ketotic within 5 d of ad libitum feeding of this particular ketogenic diet. All animals were weighed twice weekly until the start of testing. All animals appeared healthy and normally active during dietary treatment.

After 3 weeks, and during continuing dietary treatment, rats were tested on a hotplate (Columbus Instruments, Columbus, Ohio, USA) at each integer temperature between and including 46° and 51°C; one temperature was tested per day, in ascending order. Based on preliminary testing, this temperature range started at a temperature that rarely produced a nocifensive response within 60 s (46°C) and went up in integers to the temperature that produced a response by approximately 10 s in control diet animals (51°C). To quantify thermal nociception at each temperature, rats were placed on the hot plate and the latency recorded to hindpaw-associated nocifensive behavior, typically suspension of the hindpaw or hindpaw-directed licking. Once such signs were observed, animals were removed immediately. To prevent any tissue damage, rats that reached 60 s without a response were removed and scored as 60 s.

After 4 weeks of dietary treatment, rats received an intraplantar injection of CFA (a suspension of heat-killed Mycobacterium tuberculosis, undiluted) in the right hindpaw to induce a consistent and sustained local inflammation. Hindpaw sizes were measured by volume displacement and paralleled total body size the amount of injected CFA was adjusted accordingly to give an equivalent dose per paw size. CFA injection volume ranged from 100 µl (juveniles on ketogenic diet) to 190 µl (adults). Hindpaw size was measured by volume displacement just before and at 48 h after injection, a time-point selected to approximate the maximal inflammatory response; diet treatments were continued during this 48 h interval.

After final volume measurements, the dye Evans Blue was injected intravenously (60 mg/kg in a tail vein) to assess plasma extravasation (fluid movement from the intra- to the extravascular space), a major component of the inflammatory response. Tail vein injections were unsuccessful in four rats, and these were excluded from analysis. Two h after injection, rats were sacrificed by anesthesia overdose. After allowing intravascular dye to drain, hindpaw tissue was soaked in formamide at room temperature for several days to leach out extravascular dye. Duplicate aliquots of fluid from hindpaw, hindpaw or hindpaw-associated tissue were measured for optical density at 630 nm to quantify the level of Evans Blue. After anesthetic induction, rats were removed immediately. To prevent any tissue damage, rats that reached 60 s without a response were removed and scored as 60 s.

In a separate cohort of animals, trunk blood was collected after 3.5 wk of dietary treatment to assess levels of circulating ketones. These animals were not injected with CFA or Evans blue. Plasma β-hydroxybutyrate was measured with a Precision Xtra monitor and ketone test strips (Abbott Laboratories; Abbott Park, Illinois, USA). Chemicals and CFA were purchased from Sigma-Aldrich (St. Louis, Missouri, USA). Data were analyzed with unpaired t-test or two-way repeated-measures analysis of variance as appropriate. Data are presented as mean±standard error.

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Imagine treating childhood epilepsy with bacon, heavy cream and hot dogs. This may sound like an unlikely approach, but the extremely high-fat and low-carb ketogenic diet has been shockingly effective in treating kids with drug-resistant epilepsy. ABC News' senior health and medical editor Dr. Richard Besser sat down with the director of pediatric epilepsy at Massachusetts General Hospital, Dr. Elizabeth Thiele, to discuss this unusual approach to fighting epilepsy.

Dr Richard Besser: So what is the ketogenic diet?

Dr Elizabeth Thiele: The ketogenic diet is a high-fat, low carbohydrate diet, and it was developed in the 1920s after people noticed that when epileptics fasted, for various reasons, seizures would be markedly reduced.

Besser: So the ketogenic diet mimics what you’d see in someone who’s fasting?
Thiele: Right. When this was noticed, this observation was made in the 1920s, people started thinking, "Gee, what happens when someone fasts?" And when a person fasts, your body starts breaking down your fat stores. Obviously, fasting is not great for a treatment for epilepsy or other conditions because it doesn't provide adequate nutrition, so the thought was, "Gee, how could we mimic starvation and trick our bodies into thinking we're starving by using fats as the main energy source?"

Besser: So this treatment is solely based on diet?
Thiele: This treatment is solely based on diet.

Besser: No medicines, nothing else?
Thiele: We do supplement vitamins, because with the high-fat, kids can become deficient in some vitamins -- so while on the diet, all children are supplemented with vitamins and also calcium.

Besser: So on this diet, some children, who are having dozens of seizures a day, will become seizure-free?
Thiele: We've had several children having hundreds of seizures per day become completely seizure-free, oftentimes within a few weeks.

Besser: So on this diet based solely on diet and vitamins?
Thiele: Absolutely. While on this treatment, hopefully, the children are taken off all of their medications if the diet successfully controls their seizures, and then they just continue on the diet for a period of time and supplement it with vitamins and calcium.

Besser: So on this you've seen children who've been having dozens of seizures a day become seizure-free?
Thiele: We've seen many children who are having hundreds of seizures a day become seizure-free oftentimes within just a few weeks of being on the diet.

Besser: That's miraculous.
Thiele: It's miraculous. No, it's a miracle. And many children who go on this diet have already been on six or eight or 10 anti-convulsion medications without effective seizure control or with side effects that can't be tolerated. And they go on this diet and become seizure-free. About a third of children who go on this diet become completely seizure-free.

Besser: I would imagine for some of these families, they've completely given up hope that their child will be seizure-free.
Thiele: That's true. By the time a lot families learn about the diet or come to talk to us about the diet, their children have been on numerous medications, the children are continuing to have seizures, they're having side effects from the medication and many people view this diet as their last chance at controlling their child's seizures. Now with increasing awareness of the diet and the fact that more and more people are learning about it and hearing about it, many people come to consider the diet earlier in the treatment of their epilepsy, oftentimes after only one or two medications instead of after eight to 10 medications.

Besser: How effective is this treatment?
Thiele: It's very effective. And this has been looked at. The ketogenic diet has been used for more than 80 years, and every time anyone has looked at the efficacy of the diet in study form, about a third of children who go on the diet
become seizure-free, about a third of children have a great than 50 percent reduction in seizures. and for the other third, the diet doesn't work and that's often because the children have trouble tolerating the restrictions.

**Besser:** When you think about seizures and the complexity of seizures, and then you think about treating this with diet, you have to wonder how does this work?

**Thiele:** How the diet works is a very big question, because when children are on this diet, they're restricted from having birthday cake, from having French fries, from having potato chips, from having Halloween candy. So you're taking a child who's already living with epilepsy, and you're further restricting their world. So it would be much easier if we could figure out how this diet works and put it in a pill form so people could take it that way. Because it's so extremely effective, and it continues to be more effective than any medicine we have, there's an increasing amount of interest in the basic science community about what the mechanism of the diet is. It would help us understand epilepsy. And the diet is also expanding in other areas, especially neurologic disease, and there's increasing interest of using a similar diet in cancer. So obviously, understanding the mechanisms of the diet would help further help its utility in these other areas.

**Besser:** What kinds of things are being looked at for treatment with a ketogenic diet?

**Thiele:** Right now there's some preliminary evidence in Parkinson's disease and Alzheimer's disease that a diet similar to the ketogenic diet may be very effective. And there's actually a critical trial, ongoing, in ALS, or Lou Gehrig's, disease of using a similar diet and there's a lot of evidence, mainly animal model evidence, that similar diets may be very effective in helping to treat cancer.

**Besser:** Treating cancer?

**Thiele:** Treating cancer. There's evidence in prostate cancer and there's a lot of evidence in some brain cancers, like neuroblastoma. And I think that's because cancer cells are rapidly dividing so they have a very high metabolic rate and they use a lot of energy. And so the ketogenic diet basically shuts down the cell's energy production and makes the cells rely more heavily on fat metabolism, and cancer cells, I think, are not thought to do that as effectively.

**Besser:** Is this diet used to treat adults with seizures as well?

**Thiele:** There is less experience using the ketogenic diet with adults. When it was first described in the 1920s, it was described both in children and adults, and it is effective in adults, but it's honestly harder for adults to maintain and tolerate the restrictiveness. Since I've been at the Mass General we have started several adults on it, and it can be extremely effective, but again, the restrictiveness. The diet is kind of like the Atkins diet, so it really has a limit to 10 grams of carbohydrates a day--which isn't a lot.

That's why there's medications on the classic ketogenic diet now, a modified Atkins diet that's being used at Hopkins, and the modified diet we developed here, called the low glycemic index treatment, and both of those diets are less restrictive, and both of those diets appear to be probably almost as effective as the classic ketogenic diet. So there is increasing interest in using those diets in the adult population because it's thought that adults can tolerate those better because they are so much less restrictive.

**Besser:** What are some of the downsides of being on this diet?

**Thiele:** For a kid, a big downside is not being able to eat your own birthday cake, going to other kid's birthdays and not being able to eat the cake or other things the child's having. For the family, a big downside is not being able to go to restaurants. The family can go to a restaurant, but if their child is on the classic ketogenic diet, they have to take that child's meal because on the classic ketogenic diet, the meals are composed by grams of each food substance and the family weighs each component out on a gram scale, and that's not possible to do in a restaurant setting. Another advantage of the modified diets, our low glycemic and the modified Atkins, are, because they are less restrictive, the families can go to restaurants and children can order off menus. Children can eat school lunches that their school provides, just have an understanding that what's on the menu would be compatible with their diet and what would not be. But I think for most kids it's really not being able to eat candy, not being able to eat ice cream, not being able to eat French fries and that's a lot of other things that children enjoy doing.

**Besser:** Being different from other children.

**Thiele:** Being different from other children, and these are children who already feel different from other children because they have epilepsy. And it's a group of kids who are, really to me, superheroes, because they try to be normal kids even though some of them are dealing with 50,100 seizures a day.
**Besser:** Describe a typical meal for someone on a ketogenic diet.

**Thiele:** The diet is composed of what is called the ketogenic diet ratio. And that ratio means the grams of fat in a meal to the grams of carbohydrates plus protein.

And many children get started on a 4:1 ratio, meaning they get 4 grams of fat to every gram of carbohydrate plus protein. So a typical meal would be bacon, and often lots of it, with sometimes a small amount of carbohydrate in the form of vegetable or fruit. What many children on the diet, really children on the classic ketogenic diet, don’t get, is breads, they don’t get grains, they don’t get rice because all of those foods are high carbohydrate foods.

**Besser:** So it really reverses totally the food pyramid for these kids?

**Thiele:** Pretty much. We have a graph that we show that shows the typical American diet, and I think the typical American diet is close to 60 percent carbohydrates, which I’m not sure is great, and then on the ketogenic diet about 90 percent of the calories come from fat. And so many people are concerned. "Gee, high fat. What does that do for risk of heart disease in later years?" And that has never been looked at, unfortunately, but the diet’s been used for over 80 years in this country, and at least as far as I’m aware, there isn’t really an increased cardiovascular risk. We do follow children extremely carefully when they’re on this diet, since they’re growing children and we’re changing their nutrition and what we do see is that the lipids, the cholesterol will increase slightly when children start the diet but that tends to normalize over time.

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**DISCLAIMER**

The information provided in this Newsletter is of a general nature only and is not intended to replace medical advice. Any views of a medical or therapeutic nature expressed are the views and opinions of the author and are not necessarily the views of Trigeminal Neuralgia Association Australia.

Before considering or undertaking any medical or therapeutic treatment described please seek advice from a Qualified Medical Professional.

Trigeminal Neuralgia Association Australia does not accept liability for any adverse consequences that may arise from following any treatment or advice described in this Newsletter.

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60 percent carbohydrates?? 😊

😊 Take off the bread roll = Ketogenic Diet.
At my first TNA (USA) national conference, (some 10 years ago) I learned that Lyme disease could produce symptoms similar to that of trigeminal neuralgia. Since then I have searched for Lyme disease in Australia. At that time, I read the work of some local scientist/s that claimed Lyme disease does not exist in Australia simply because Australia doesn't have the “Deer ticks” which carry the Lyme disease bacteria or any trace of the Lyme disease bacteria here. But I have met local “TN” folks who have been bitten by the cattle or bush ticks... and so I have always wondered...

Lyme disease
Last updated: 26 March 2012

Lyme disease is caused by the bacterium Borrelia burgdorferi. Typical symptoms include fever, headache, fatigue, sore muscles and joints, and a characteristic skin rash called erythema migrans.

Although locally-acquired Lyme borreliosis cannot be ruled out, there is little evidence that it occurs in Australia. There is a continuing risk of overseas-acquired Lyme disease being imported into NSW.

What is the disease?
- Lyme disease is caused by the bacterium called Borrelia.
- The first symptom is usually a characteristic pink or red rash that starts as a small red spot that gradually spreads in a much larger circle with a characteristic bulls-eye appearance. This normally happens between 3 and 32 days after being bitten by an infected tick. Not everyone with Lyme disease gets the rash.
- There may also be fever, headaches, tiredness and joint pains.
- In later stages of Lyme disease the infection spreads through the bloodstream and can cause infection in the brain and membranes surrounding the brain (meningoencephalitis) and infection in or around the heart (endocarditis, myocarditis or pericarditis). The disease can also cause inflammation of joints and cause joint pain and long-term neurological involvement.

How is Lyme disease spread?
- Lyme disease is transmitted following the bite of a tick that is infected with the Borrelia bacterium.
- Only some species of ticks are capable of being infected by the Borrelia bacteria and only these infected ticks can pass the infection on to humans. This group of ticks is found in Asia, Europe and North America, but not in Australia.
- Ticks with Borrelia infection live in temperate forested areas of northern Asia and Europe (especially central and eastern Europe) and the United States (especially north-eastern, north central and Pacific coastal USA).
- In the 1990s, 12,000 ticks were collected from different parts of NSW and were tested for Borrelia bacteria. No evidence of Borrelia infection could be found in any of the ticks collected.
- In April 2011, NSW Health convened an expert panel with expertise in public health, epidemiology, infectious diseases, rickettsial diseases and entomology to provide advice on the current risk of Lyme disease in NSW. The panel concluded that although locally-acquired Lyme disease cannot be ruled out, there is little evidence that it occurs in Australia. The panel also noted that there was a continuing risk of overseas-acquired Lyme disease being imported into NSW.
- Lyme disease is not spread from person to person.

How is Lyme disease diagnosed?
- Lyme disease is diagnosed based on symptoms, physical findings (e.g., a characteristic rash), and the possibility of exposure to infected ticks. Laboratory testing is helpful in the later stages of disease.
- Diagnosis of any infectious disease requires a combination of clinical experience and assessment by the doctor and understanding of the lab tests and their limitations. Laboratory tests are rarely definitive and all tests have a proportion of results which are false positive (test indicates disease in someone without the disease) and false negative (test indicates that there is no disease in someone with the disease). When tests are done in places where a disease is rare or absent (for example, Lyme disease in Australia), many positive tests will be falsely positive.
- The tests to diagnose Lyme disease are technically complex and require specialist expertise. It is important for people who want to be tested to make sure the laboratory that performs the test has accreditation with the National Association of Testing Authorities (NATA).
- Lyme disease is most commonly diagnosed by a screening test called ELISA and this is then confirmed using a western blot test. Both of these tests detect antibodies that are produced by the immune system of someone with Lyme disease.
- Lyme disease can also be diagnosed by testing a sample of the skin lesion by nucleic acid testing (eg PCR) or culture. (See testing advise for clinicians for more information).
- Occasionally, tests performed in Australia for Lyme disease show evidence of an infection. When these cases have been followed up in the past, the cases have been found to have acquired the infection while overseas.
- Tests for Lyme disease should only be done by laboratories that have current accreditation with National Association of Testing Authorities (NATA).

How is Lyme disease treated?
- Most cases of Lyme disease can be treated successfully with a few weeks of antibiotics

Can ticks in NSW transmit infections?
- A species of paralysis tick called *Ixodes holocyclus* can be found along Australia’s east coast and can cause tick paralysis, tick typhus and allergic reactions.
- While there is little evidence that Lyme disease is caused by Australian ticks, there may be other infections carried by Australian ticks which may cause an infection which is similar to Lyme disease. These infections remain poorly characterised.

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**THE PARALYSIS TICK (I. HOLOCYCLUS) ON HUMANS**


Clinical and neurophysiological features of tick paralysis

P. J. Grattan-Smith 1,2; J. G. Morris 2,4; H. M. Johnston,5 ;C. Yiannikas 2,4 R.; Malik,6 ;R. Russel l3 ;R. A. Ouvrier 7

**Tick-transmitted diseases**

Many ticks worldwide are capable of transmitting viral, bacterial and protozoal diseases. In Australia *Ixodes holocyclus* is known to be capable of transmitting some of the bacterial diseases. In Australia, rickettsial spotted fever (*Rickettsia honei*), flinders island spotted fever (*Rickettsia australis*), and Australian form of Lyme disease (*Borrelia sp.*) are the major concerns although Q fever (*Coxiella burnetti*) is also a potential pathogen.

**Australian borreliosis**

Whilst the offending *Borrelia* sp organism(s) has not been clearly characterised, a form of Australian human borreliosis does seem to occur. Incubation period ranges from 3 to 30 days after a bite from an infected tick. Often a lengthy elapsed time causes considerable confusion for diagnosticians. The clinical signs of human borreliosis in Australia are similar to the classic Lyme disease in America (first described in the USA in 1975, isolated in 1981). A wide variety of tissues may be invaded, and may lead to permanent injury to joints, heart, central nervous system and other internal organs. Early recognition and treatment with antibiotics over several weeks can halt the disease, but delay may result in a lifetime of chronic illness and fatigue. Differential diagnosis might include dengue fever, malaria, glandular fever, rickettsial spotted fever, epidemic poly-arthritis and rheumatoid arthritis. As yet there is no definitive test for borreliosis in Australia because the organisms are difficult to culture and hence to identify and characterise.

A six year old girl with a left-sided Bell's Palsy. An adult female paralysis tick *Ixodes holocyclus* had been embedded behind the left ear for several days. The left side of the face, and the left-sided peri-ocular muscles are paralysed (Moorhouse, 1981).

**Lyme borreliosis** (caused by *Borrelia sp* of some kind). If you experience any of the following symptoms after a few days, weeks or even months later, contact your doctor.

**Early infection:** flu-like symptoms, headache, fever, muscle or joint pain, unusual fatigue, swollen glands, conjunctivitis. A rash may occur at the site of the bite or elsewhere, variable in shape and colour. Many people do not get a rash. Skin irritation immediately following tick bite is not necessarily a sign of infection.

**Chronic infection:** Symptoms may include chronic fatigue, behavioural changes, severe headaches, neck problems, nerve inflammation, memory problems, eye problems, recurring rashes, intermittent or chronic disabling pain, arthritis, heart problems.
SYDNEY SUPPORT GROUP
Saturday 7th July 2012 –
Toongabbie Public School


Apologies: Marion A, Kim S, Elizabeth and Lloyd T.

Kim welcomed everyone to the meeting especially the new folks in the room. We then did a members’ update before the guest speaker arrived.

Jan: MVD 4 years ago and has had no more TN trouble except for a few balance problems. Is now having treatment for a disc bulge in her back and is taking Neurontin for pain control. Jan laughed at that she has come round a full circle.

Stewart: has Anaesthesia Dolorosa, and finds that nothing helps much with his pain. He is on Endep which gives him a good night sleep, and he finds distraction helps, while stress makes it worse.

Jocelyn: had a successful MVD 4 years ago – since then no pain.

Kim: Had TN for 8 years before she had an MVD then pain came back after 15 months. Is now taking Tegretol and has reduced 800gm to 600gm as she has lately been going to an acupuncturist.

Jane: (New) Fiona brought her mother Jane to the meeting. Jane has TN for 2 years and needs information. Recently she was admitted to hospital with the uncontrolled pain. She is now taking 100gm Tegretol twice a day plus Lyrica 75gm at night, but still gets break-through pain. She has tried Neurontin and Elipim which did not work for her. Has had a MRI which established there is a blood vessel compressing on the nerve.

Irene said that sometimes medication “don’t work” is because they have been incorrectly dosed or taken. And that perhaps the low dosage of Tegretol & being the normal Tegretol and taken 12 hours apart may have been the reason for the break through pain. It was suggested that Jane try the Tegretol CR. Being empowered with information, Jane could also learn when and how to titrate her TN medication.

Jeanette: Had TN for 18 years. In 2005 when due to have MVD suffered a stroke which released the vein/artery and she hasn’t had any pain since!

Ann: TN started in 1986. Had MVD in 1998 which lasted 1 year. Then had Radiofrequency (RF) lesioning which has taken the pain away but left ½ her face numb. She said with what she knows now, she would have had a 2nd MVD before the RF.

Brenda: (New) Brenda is profoundly deaf so her son Wally came along to sign for her. She had a wisdom tooth pulled 2 months ago which broke into 4 pieces. Afterwards pain started in her nose. GP put her on Tegretol 100mg then upped it to 200mg which makes her dizzy. She has suffered migraines all her life but they have lessened since TN. She is now on Lyrica 75mg twice a day with an extra 150mg at night. She is going to see a Neurologist on 25th July.

Irene: Brenda has brought a new dimension to the group. It was quickly noted that Wally has some difficulty signing the drugs name. To help overcome that the group will make small signboards with the all the different TN drugs name, so we can just hold up the signboard instead of trying to sign out the spelling. There may come a day we may have to make braille boards.

John: (New) TN since August 2009. GP diagnosed TN and sent him to a Neurologist who put him on Tegretol which he couldn’t take so tried Lyrica which made him feel like a zombie. Pain was so bad in April he couldn’t eat, clean teeth etc and so decided to have an MVD. He has been free of pain since, but has double vision so wears a patch over the eye. He is hoping this will resolve soon.
Our speaker for the day was Margaret Spicer – Bowen Therapist
Margaret has been working as a Bowen Therapist for 18 years. Margaret explained that Bowen Therapy is a holistic and multidimensional, soft tissue approach to healing. This therapy is based on Tom Bowen, the founder’s work. Tom lived and practiced in Geelong, Victoria. He theorised that the underlying cause or source of many musculoskeletal, neurological, neuromuscular and other health or pain problems could be found in the soft tissue or fascia.

(Fascia is a specific type of connective tissue that forms a three dimensional web surrounding every tissue in the body. Fascia is the body organiser embracing all nerves, bones, arteries, veins and muscles. Therefore fascial dysfunction can affect every structure, muscle, nerve and organ in the body. Fascia has become the subject of increasing research and recent studies have shown it to be the largest sensory organ in the body. It is rich in a range of receptors that powerfully influence the body’s neuro-muscular physiology in many and complex ways. Bowen Therapy, through specific soft tissue or fascial release and integration techniques, stimulate specific receptors that enable the body itself to correct dysfunctions and restore homeostasis (balance) on a holistic level. Through treating the cause rather than the symptoms Bowen Therapy has consistently shown it can have profound and permanent healing and pain relief outcomes. This relief is experienced by many people who present with a wide range of painful conditions, even where other modalities or treatments have resulted in transient or little improvement.

**Conditions Commonly Treated**
There is a very wide range of acute and chronic conditions for which Bowen Therapy has provided outcomes ranging from long-term rapid remission to significant improvement in presenting problems, mobility, physiology, pain, stress and general well-being. Conditions that can respond well include:

- Acute and chronic pain with musculo-skeletal or neurological origins
- fibromyalgia,
- back pain
- lumbago,
- sciatica,
- chronic fatigue syndrome,
- neck pain,
- arm pain and carpal tunnel syndrome
- TMJ syndrome,
- shoulder pain (frozen shoulder),
- leg and foot pain (plantar fasciitis),
- emotional depression and stress,
- asthma,
- sporting and other trauma injuries.

**The Benefits**
The principal benefit of Bowen Therapy is the often rapid and long term or permanent remission from pain, reduced stress, greater mobility and improved physical and emotional quality and enjoyment of life. Additional benefits can include increased energy, improvement in the immune system, rebalancing of the body, improved circulation, lymphatic drainage and detoxification. Further and important benefits of Bowen Therapy are that it has few contraindications. It is very gentle, relaxing and non-invasive involving no manipulation, and is therefore ideal for everyone including children and the aged.) ~ excerpt from Bowen Therapists Federation Inc.

Margaret also said it is important for the skin to be well hydrated in order for the fascial to release for deep relaxation. Pain can also be caused from injuries received years before in other parts of the body, which you have forgotten but the brain has not, especially if from being unconscious due to an accident or having teeth pulled etc.

Margaret also treats cancer patients in between their cancer treatments which helps them relax as patients are often tense from being in pain.

Kim thanked Margaret for sharing Bowen Therapy with us and spending her Saturday with us. Kim presented Margaret with the book “Insight” as a token of our appreciation.

Please make a note - 3 November 2012 – city (venue to be confirmed)
Invited guest speaker is Prof Phyllis Butow from University of Sydney (Australia), on the topic “Doctor-Patient Communication: The Key to Patient Care and Adherence.” All are welcome.
Frank noted that after payment for the hall today, we have $227.60 and he proposed that $100 be donated towards next year’s conference. The motion was unanimously supported.

After the meeting we enjoyed all the lovely food that everyone had brought to share, while catching up with a cup of coffee. Welcome back Henry our raffle man. Glad to see sore foot and all is well.

Reminder: September’s meeting will see us mark our 12th Anniversary. Please come along and celebrate the occasion.

Irene.

TOWNSVILLE SUPPORTGROUP
Saturday 30 June 2012

**Present:** Sera A, Peter A, Joy K, Jill S, Mary C, Sue M, Loris K.

Apologies from Troy and Nancy as they are away in Toowoomba.

Sera: Welcome everybody and thank you for coming and welcome to the new member. Sera is having a miserable time at the moment. She is in a lot of pain sometimes very excruciating and other times not as bad. Cold it is setting it off. At times the pain is so unbearable I yell. A lot of trouble eating and brushing teeth. On 6 Panadol in the day and 2 Panadeine Forte at night so I can sleep, as well as the rest of the TN medication and the Myasthenia Gravis (MG) medication, which makes me swollen in the face. Once the MG is under control, then the steroid medication can be reduced. MG affects mostly women under 25 and men over 60. I go to their support group as well.

**Loris:** (new) Had a very bad bout - couldn’t eat, touch her lip and brush teeth. Was told the operation only works if the pain is on top of the head. Told about another operation where a nerve is cut and that causes numbness. Her son, who is a doctor, doesn’t recommend that one.

**Sera** said she heard at the conference that botox can help.

**Loris:** Asked if anyone knew what sets it off for a long time.

Sue said Cold weather and breezes and viruses do that to her pain.

Jill is not having pain at the moment but when it comes it is continuous and affects both sides of the eyes area.

**Sera:** If you can get referred to the pain clinic, you can get the drugs at the PBS rate.

**Mary:** has TMJ not TN, it slices down all one side of her body and 2 sciatic nerves. She takes Endep for a good night sleep. Had a very bad bladder infection as well, an infection of the lining of the bladder.

**Sue:** Generally not too bad on medication but feeling it because of the cold winds in Mt Isa at the moment. Has to rug up with a scarf. Still plays up after a virus. Was on up to 6 Panadol Osteo for arthritis as well but Dr told her it was not good (blood test results) so she is down to 2 Panadol Osteo and 2 Nurofen most days as she cannot cope with more Nurofen. When asked if the drugs made her dopey, she said that she tries not to drive early in the day or after the evening tablets. She has no tablets in the middle of the day by choice. Yes they do make you dopey, so she doesn’t want to take any more or she wouldn’t be able to drive. B12 helps prevent the requiring of more medication.

**Jill:** Can get sub-lingual B12 spray at Calanna now.

Sue: Explained to Loris why the B12 was recommended.

Loris and Sera want to try the spray, which is cheaper.

Peter, Sue and Sera discussed the relationship between MG, Gabapentin, Lyrica and TN and the doctor’s reaction. He said that the results had not been able to be replicated.

Jill: Asked Sue again about the connection between no hayfever when on Lyrica and/or Epilim.

Loris asked about membership and was given a copy of the newsletter. She also asked about how it was advertised.

Sera: Thanked everyone for being there and said that we will break for afternoon tea and the next meeting is TBA depending on Sue’s visit.

*Sue & Sera*
Correspondence Corner

Dannielle O: I’m not able to attend meetings but I thought I’d share some progress that I’m having with a new medication.
4 weeks ago, my doctor prescribed me Cymbalta and I have had a great deal of success with it, I literally have NOT had any pain since I’ve been on it. It’s truly amazing!
It’s an anti-depressant and an anti-anxiety and used for patients with chronic pain. I have noticed a remarkable decrease in my anxiety and depression levels and I physically feel the BEST I have felt in years! My only side effects are insomnia to which I’ve been given sleeping tablets for. I thought I’d share my experience if it will help others.

Frank M: Norma is not well. Her knees are giving her lots of trouble. She is scheduled for right knee replacement on 21st August. A rough time ahead I am afraid.

Hobart Support Group:
- next meeting is on Saturday 25th August Glenorchy library, to be there at 1.50pm for a 2pm start.

Laughter is the Best Medicine

Exercise For Real Life
The doctor told me “Physical exercise is good for you.” I know that I should do it, but my body is out of shape, so I have worked out this easy daily program I can do anywhere:
Monday:
Beat around the bush.
Jump to conclusions.
Climb the walls.
Wade through paperwork.
Tuesday:
Drag my heels.
Push my luck.
Make mountains out of mole hills.
Hit the nail on the head.
Wednesday:
Bend over backwards.
Jump on the band wagon.
Balance the books.
Run around in circles.
Thursday:
Toot my own horn.
Climb the ladder of success.
Pull out the stops.
Add fuel to the fire.
Friday:
Open a can of worms.
Put my foot in my mouth.
Start the ball rolling.
Go over the edge.
Saturday:
Pick up the pieces.
Whew! What a workout!

The second day of a diet
The second day of a diet is always easier than the first. By the second day you’re off it. ~ Jackie Gleason
Diet pills
“I’m prescribing these pills for you,” said the doctor to the overweight patient, who tipped the scales at about three hundred pounds.
“I don’t want you to swallow them. Just spill them on the floor twice a day and pick them up, one at a time....”

PUNNY DIET FACTS

A diet is a weigh of life.
It's not the minutes spent at the table that put on weight, it’s the seconds.
It's something most of us do religiously: We eat what we want and pray we don't gain weight.
The problem with curbing our appetites is that most of us do it at the drive in window of McDonalds.
The most fattening thing you can put in an ice cream sundae is a spoon.
The biggest drawback to fasting for seven days is that it makes one weak.
Sweets are the destiny that shapes our ends.
Diets are for people who are thick and tired of it.
The toughest part of a diet isn’t watching what you eat. It’s watching what other people eat.
Diets are for women who not only kept their girlish figure but doubled it.
A diet is when you have to go to some length to change your width.
Many women reduce and reduce, yet still never manage to become a bargain.
The best way to lose weight is by skipping ... snacks and desert.
Most people gain weight by having intimate dinners for two...alone.
People go to Weight Watchers to learn their lessens.
A diet is the modern-day meal in which a family counts its calories instead of its blessings.
A diet is what you go on when not only can’t you fit into the store's dresses, you can't fit into the dressing room.
One guideline applies to fat and thin people alike: If you're thin, don't eat fast. If you're fat, don't eat - FAST.
## 2012 Meeting Dates

<table>
<thead>
<tr>
<th>State</th>
<th>Group</th>
<th>Date &amp; Time</th>
<th>Venue</th>
<th>Group Leader/s</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACT</td>
<td>Canberra</td>
<td>TBA</td>
<td>Barbara Byrne Room Labour Club, Belconnen</td>
<td>Jan Goleby 02 6254 6640</td>
</tr>
<tr>
<td>NSW</td>
<td>Sydney</td>
<td>1st September 1:30 – 4:00 pm</td>
<td>Toongabbie Public School Cnr Fitzwilliam &amp; Binalong Rds</td>
<td>Kim Koh 02 97431279 Kim Smith</td>
</tr>
<tr>
<td></td>
<td>Sydney CBD</td>
<td>4th August 10am 12:30pm</td>
<td>St. James Parish Hall, Level ONE, 169 Phillip St. Sydney CBD</td>
<td>Irene Wood 0413 363 143</td>
</tr>
<tr>
<td>QLD</td>
<td>Brisbane</td>
<td>11th August 1.30 -4.00pm</td>
<td>30 Ridley Road BRIDGEMAN DOWN</td>
<td>Leonie Gall 0407 55 44 07 Tony MacPherson 07 3822 2286</td>
</tr>
<tr>
<td></td>
<td>Sunshine Coast</td>
<td>22nd Sept 1:00 PM</td>
<td>Kawana Library, Nanyima Street, Buddina</td>
<td>Jean Williams 07 54911978</td>
</tr>
<tr>
<td></td>
<td>Townsville</td>
<td>TBA 1.00 – 4:00pm</td>
<td>Carville Senior’s Villa 35 – 37 Diprose St PIMLICO</td>
<td>Sue Macey; Sera Ansell 07 47516415</td>
</tr>
<tr>
<td>S.A</td>
<td>Adelaide</td>
<td>30th Sept 2:00 – 4:00 pm</td>
<td>Burnside Town Hall Civic Centre Cnr Portrush/Greenhill Rd</td>
<td>Graham/ Liz Boyer 08 8392 2781</td>
</tr>
<tr>
<td>TAS</td>
<td>Hobart</td>
<td>25th August 2:00 – 4:00 pm</td>
<td>Glenorchy Library Enter via Barry and Cadell Streets</td>
<td>Helen Tyzack 03 6245 0429 Ros Wilkinson 03 6234 7989</td>
</tr>
<tr>
<td>VIC</td>
<td>Melbourne</td>
<td>11th August 1.30 – 4:00pm</td>
<td>Maroondah Federation Estate, 32 Greenwood Ave RINGWOOD</td>
<td>Evelyn Diradjki 03 9802 6034</td>
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Contact: TNA Australia P O BOX 1611, CASTLE HILL, NSW 1765 Australia Tel: 02 4579 6226; Email: tna_sydney@yahoo.com or irene.wood@tnaustralia.org.au