

WHEN ALS IS LYME

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Examining the link between ALS and Neuroborreliosis



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“Activation of Microglia, the resident innate immune cells of the CNS, occurs in virtually all diseases of the CNS and was an early observation by 19th and 20th century biologists.”

~ Allitia B Dibernardo & Merit E Cudkowicz

“Facts do not cease to exist because they are ignored.”

~ Aldous Huxley

“If you are not your own doctor, you are a fool.”

~ Hippocrates - founder of Medicine

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Preface

Every few minutes, someone in the world dies of Amyotrophic Lateral Sclerosis. This book shows that most things we are told about ALS are incorrect:

- ALS is not just a motor neurone disease but affects the entire brain
- ALS is much more common in climates with endemic Ixodes ricinus ticks
- The more Lyme disease reported in a US state, the more ALS cases too
- The US's largest ALS cluster lies in the US's number one Lyme hotspot
- Other major ALS clusters are found in other major Lyme hotspots
- Almost all ALS patients test positive for Lyme, when tested properly
- Many ALS patients have the same reaction to antibiotics as a Lyme patient
- There are dozens of documented ALS cases that cured using antibiotics
- Antibiotic trials for ALS could never have succeeded - by design
- Proper Lyme diagnosis to rule out "ALS" is sabotaged for financial reasons

If you have been told that the chance that your ALS is Lyme-related is very small, then we hope that this book will persuade you to get tested for Lyme disease.

If you have been tested for Lyme disease, and the test came back negative, we hope that this book will persuade you to get properly tested: The only tests that has an acceptable rate of false-negatives is an antibiotic-provocative test using a test that has not been deliberately made useless. The likelihood that you have been tested correctly is vanishingly small.

If you have been taking antibiotics to treat Lyme disease and your ALS symptoms remained or worsened, we hope this book will persuade you to get proper antibiotic treatment - currently used doses are too low and treatment durations too short. Late-stage Neuroborreliosis is often incurable - but its ALS symptoms can be arrested and very slowly reversed.

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Acknowledgements

The trenchwork for this book has not been done by us - we are mere messengers, investigative journalists in the medical field.

We would like to thank all medical doctors, microbiologists, research scientists, medical journalists and patients whose material we used. Some of that material is included as "Fair use" for research purposes, with the aim of ultimately saving lives. The Copyrighted material we partially reproduced in Fair Use in this book is crucial to document a Lyme-ALS link and much is at risk to disappear soon, since the people involved have often died and hosting for their blogs and websites is therefore often not being paid any more, or at risk of being terminated in the near future.

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ALS-like illnesses soon bigger killer than cancer, says WHO

People tend to think of ALS as a very rare disease. Part of that is because there aren't many people with ALS. That is because they don't live very long. ALS is not rare at all.

"ALS is not considered a rare disease¹. Approximately 2,500 to 3,000 Canadians currently live with ALS. Two or three Canadians are diagnosed with ALS daily, while another two to three lose their battle with ALS every day. In Ontario, more than 1,000 people have ALS at any one time. While these numbers may seem insignificant, ALS is five times more prevalent than Huntington's disease, four times more prevalent than muscular dystrophy, and has a similar incidence rate as multiple sclerosis. The number of people with ALS is smaller than people with these other neurological diseases because people with ALS often die so rapidly.

According to Dr. Michael Strong, chief of neurology at the University Health Sciences Centre and research scientist at the Robarts Research Institute in London, Ontario, "ALS is clearly the most common cause of neurological death on an annual basis." **Furthermore, the World Health Organization (WHO) estimates that neurological diseases such as ALS will surpass cancer to become the second-leading cause of death in Canada by 2040."**

¹ <http://www.alsont.ca/about-als/>

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Patients are being misinformed about the nature of ALS

ALS is not a muscle disease

Many people are unaware of the fact that ALS always only affects the central nervous system (brain and spinal cord), never the muscles directly. In Germany, ALS is generally called a "Muskelkrankheit", a muscle disease.² In the Netherlands, ALS is called the same, a "spierziekte", a muscle disease.³ In Norway, ALS is called a "muskelsykdom", again a muscle disease.⁴

However, ALS has nothing whatsoever to do with muscles. **ALS is defined as a disease of the nerve cells in the brain and spinal cord**⁵ (that control voluntary muscle movement). As we will explain, there are two fundamental things provably wrong with this definition, but the basic premise is correct: Neurons are damaged.

What is the origin of this widespread misunderstanding, this misnomer of the disease as a "muscle disease"? Medical laypeople see the muscles withering and failing to respond to the commands from the brain, but neurologists know the underlying cause but are doing frustratingly little to dispel the fundamental misunderstandings caused by this widespread myth.

However, medical professionals are doing little to rectify this fundamental error in understanding the disease. Perhaps because focusing on the muscles distracts the attention from the brain? Distracting the attention from the brain makes life easier for doctors.

For imagine a patient with signs of early stage ALS. This patient, as many ALS patients do, remembers a tick bite one or more year prior. If ALS would be a "brain disease", the patient, having read about how Lyme disease can ravage the brain, would insist on getting tested for Lyme. But neurologists hate doing that, as we prove with hard data, published by the doctors themselves.

2 <http://www.google.com/search?q=muskelkrankheit+als>

3 <http://www.google.com/search?q=spierziekte+als>

4 <http://www.google.com/search?q=muskelsykdom+als>

5 <http://www.ncbi.nlm.nih.gov/pubmedhealth/PMH0001708/>

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ALS is not a motor neurone disease

Recent published research shows that the majority of ALS patients suffers from substantial neuronal damage above and beyond that to motor neurons - **redefining ALS as a generalized neurological syndrome affecting the entire brain**, with motor neurons affected in particular - and that could very well be coincidental, in the sense that when they are not particularly affected, the syndrome is called Multiple Sclerosis, Parkinsons or Alzheimer's.

In up to 15% of cases, ALS patients suffer brain damage to the non-motor neuron parts of their brains so severe that it leads to a dual diagnosis such as ALS-dementia and in an additional 50% of ALS patients, there is still so much brain damage to the rest of the brain that it can be detected in a brain MRI^{6 7}. **Research shows that at least two third of ALS patients suffer generalized brain damage in addition to motor neuron damage.**⁸

6 N. Sudharshan, C. Hanstock, B. Hui, T. Pyra, W. Johnston, S. Kalra. **Degeneration of the Mid-Cingulate Cortex in Amyotrophic Lateral Sclerosis Detected In Vivo with MR Spectroscopy.***American Journal of Neuroradiology*, 2010; 32 (2): 403 DOI: [10.3174/ajnr.A2289](https://doi.org/10.3174/ajnr.A2289)

7 U. Usman, C. Choi, R. Camicioli, P. Seres, M. Lynch, R. Sekhon, W. Johnston, S. Kalra. **Mesial Prefrontal Cortex Degeneration in Amyotrophic Lateral Sclerosis: A High-Field Proton MR Spectroscopy Study.***American Journal of Neuroradiology*, 2011; DOI: [10.3174/ajnr.A2590](https://doi.org/10.3174/ajnr.A2590)

8 <http://www.sciencedaily.com/releases/2011/09/110916131252.htm>

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ALS is not a disease

It may sound harsh but it is the truth: ALS is not a disease – and anyone claiming otherwise will fail to provide evidence to such.

ALS is most accurately defined as a set of “signs” – objectively quantifiable symptoms. In case of sporadic ALS, the underlying cause is unknown, making ALS not a disease but a symptom description. It is crucial to keep this in mind. It is unfair to patients when they are lead to believe that they are with 100% likelihood suffering from a disease without a cure, instead of a perhaps treatable symptom of an identifiable illness.

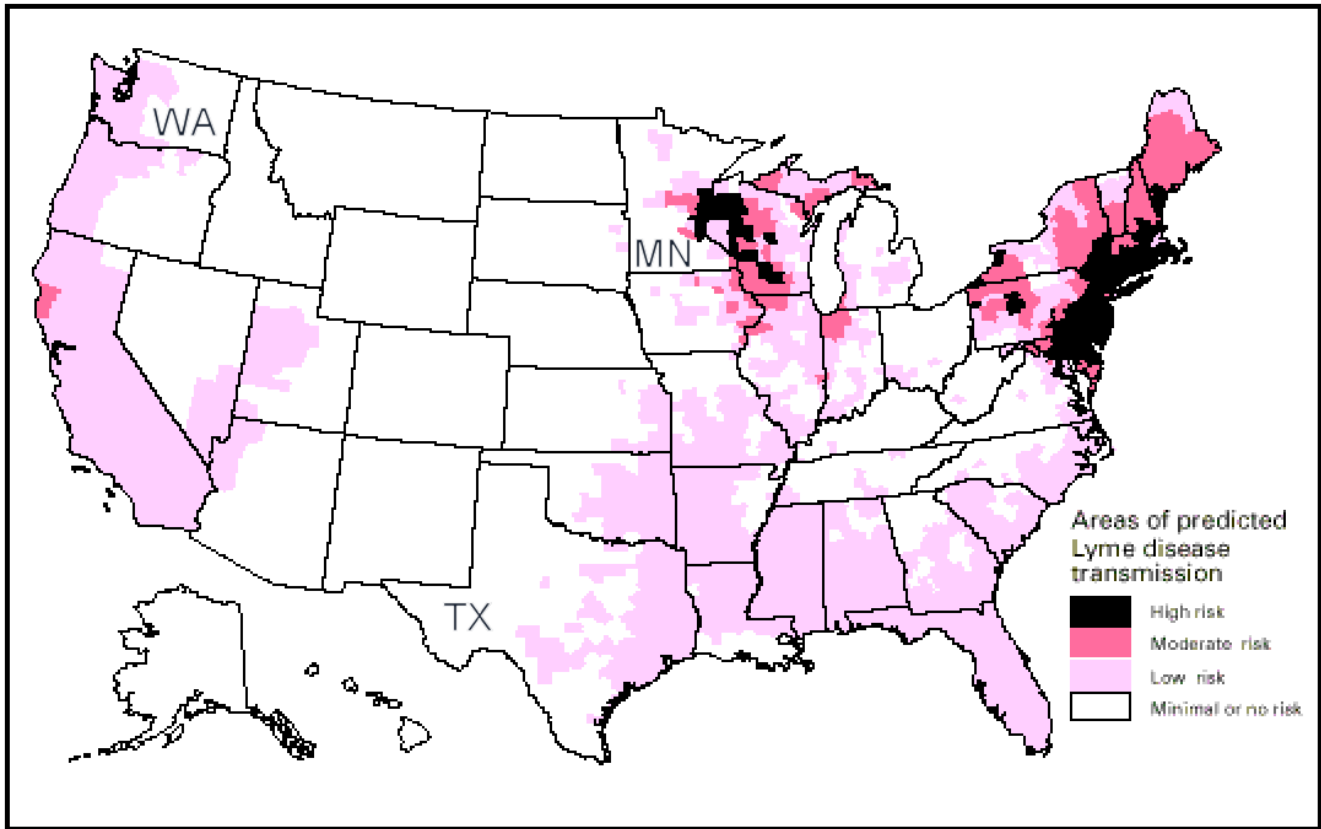
One of the reasons why people still believe that ALS is a disease is that ALS is also known as “Lou Gehrig’s disease”. When ALS got that name, it was not yet clear that ALS can be caused by a variety of factors, and that it is assumed to be a symptom of an unknown disease or -diseases. Even though there are countless cases of ALS being caused by Lyme disease, the medical establishment still believes that there is such a thing as “real ALS” and that it has an unknown cause or causes. So far, the only identified causes of Amyotrophic Lateral Sclerosis (Motor neuron damage) have been familial ALS and Lyme neuroborreliosis.

Patients should understand that ALS is a symptom, and that ALS has been cured many times with antibiotic treatment. Our book documents eleven ALS patients who cured their ALS with antibiotics. These people have been diagnosed by a neurologist as having ALS, but it turned out their ALS was a symptom of Lyme disease and antibiotics provided a cure.

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Circumstantial evidence linking Lyme to ALS

US states with more Lyme also have more ALS



It is hard to obtain detailed epidemiological statistics on ALS, but the relative risk of getting Lyme disease is available from the CDC:

We marked Washington, Minnesota and Texas because there was a study in Neurology where they gave the incidence of ALS for those states (number of new cases per year per 100,000 persons):

Minnesota 6.6

Washington 5.1

Texas 3.3

Source: Cronin S., Hardiman O., Traynor B.J, *Ethnic variation in the incidence of*

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ALS - a systematic review, Neurology 68, pp. 1002-1007, 2007⁹

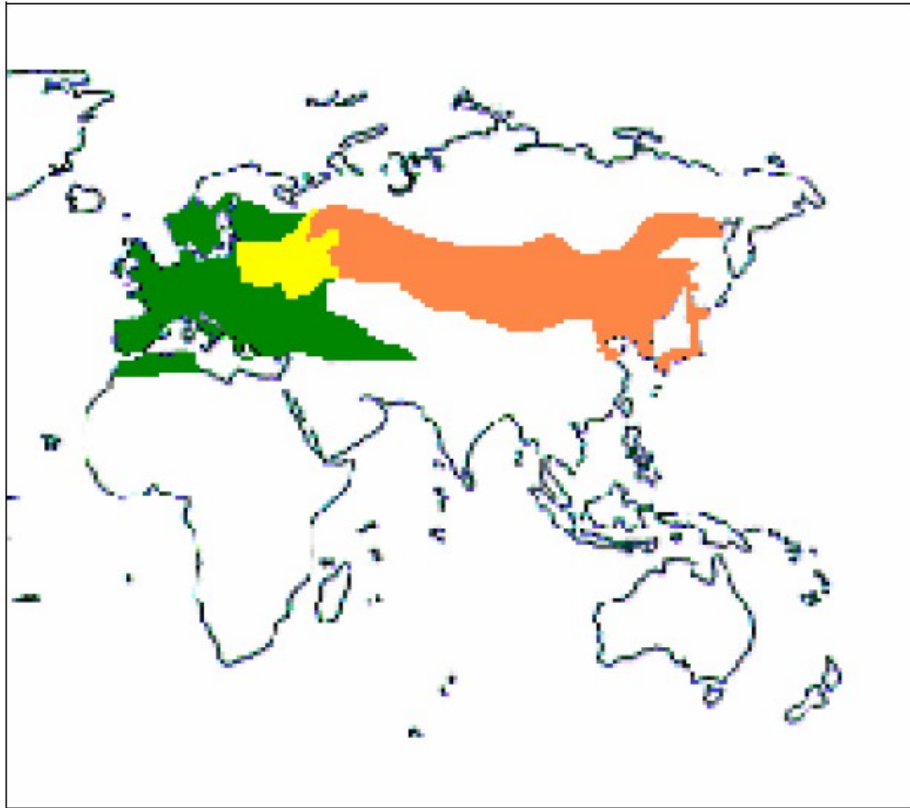
When we compare these figures with the CDC's Lyme-risk map, we see that Minnesota has a higher Lyme risk than Washington, and Washington a higher Lyme risk than Texas. This corresponds with an increasing ALS risk for those states. Coincidence? A Minnesotan is twice as likely as a Texan to get ALS. And that same Minnesotan is at least twice as likely to contract Lyme disease.

It is generally believed that there is no such thing as "ALS hot-pockets", that ALS is evenly distributed across a country. But this is obviously not true. **As the prevalence of Lyme disease goes up, so does the prevalence of ALS in the United States.**

⁹ <http://www.neurology24.com/lou-gehrigs-statistics.htm>

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The spread of Ixodes ricinus ticks is proportional to ALS incidence



Prevalence of the Ixodes ricinus tick (green and yellow)¹⁰

When we compare the WHO's map of the spread of the Ixodes Ricinus tick - a major vector for Lyme disease - in Europe and North Africa with the incidence of ALS in those areas, we again see a relationship: The more Ixodes ticks, the more ALS.

On the map we see that Tunisia, Israel, Libya and Iceland have few Ixodes ticks. This is due to their dry, resp. cold climate. Their respective incidencies for ALS are 0.4, 0.6, 0.9 and 0.8 per 100,000, whereas the incidencies for the Ixodes-endemic parts of Scandinavia and the UK are between 2.1 and 2.6. **We see the lowest ALS-incidence in countries with few Ixodes ticks, and an ALS-incidence of up to three times that in areas with a high prevalence of Ixodes ricinus¹¹.**

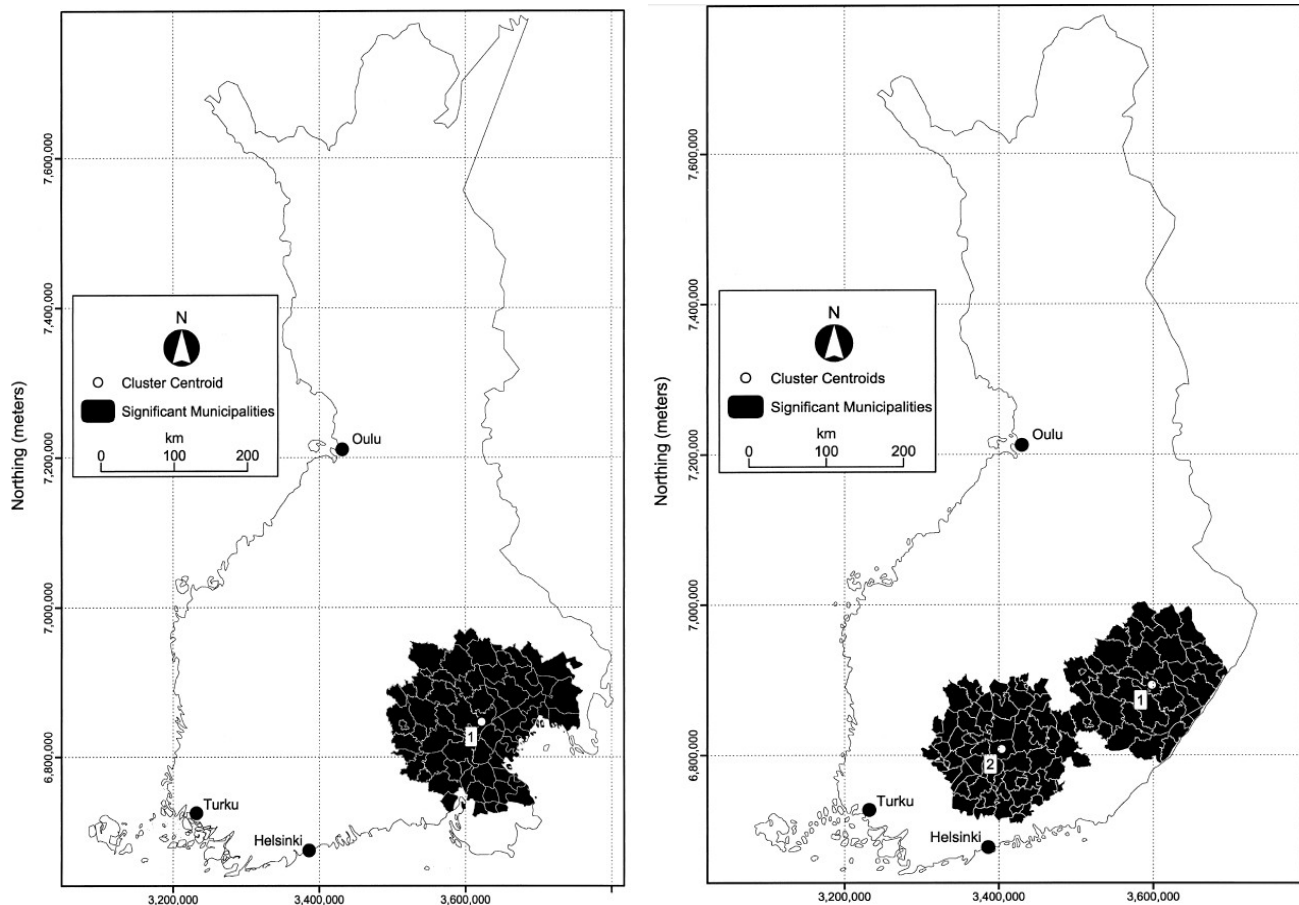
¹⁰ <http://www.unfictional.com/pdf/lyme-epidemiology-europe.pdf>

¹¹ <http://www.unfictional.com/pdf/ALS-epidemiology-EURALS.pdf>

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ALS clusters occur where Lyme disease is most prevalent

A study by Sabel et. al found Finnish ALS clusters related to place-of-birth and place-of-death. Those clusters were situated in the southeast of Finland, the area with the highest prevalence of ticks and Lyme disease. Cluster I in the rightmost picture (place of death) had double the ALS incidence as statistically expected¹²



Clustering of 1000 Finnish ALS cases 1985 - 1995

¹² <http://www.unfictional.com/pdf/als-cluster-finland.pdf>

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Enormous ALS clusters occur near grassy, wooded park areas

The largest ALS cluster to have been identified so far is amongst employees of Kelly Air Force Base, decommissioned in 2001. The families of the victims declared to investigators that a total of 140 employees died of ALS, but because the death certificates usually mentioned "aspiration pneumonia" or similar ALS-related causes of death, the health authorities refused to recognize those deaths as ALS, and the figure was therefore "spun down" to 39 deaths - still highly anomalous:¹³

Concern was raised after media reports of what appeared to be unusually high incidence of ALS among base workers who met at a support group sponsored by the San Antonio ALSA chapter. In 1999, the South Texas chapter of the ALSA reported a suspected cluster of cases to the Air Force and regional health authorities. The Air Force Institute for Environment, Safety, and Occupational Health Risk Analysis and the San Antonio Metropolitan Health District then launched an investigation into mortality from all causes at the base, including cancer and ALS.

An initial informal survey by the ALSA chapter found 39 workers and former workers with ties to Kelly who said they or a family member had been diagnosed with ALS or ALS-like symptoms. Ultimately, **Kelly families reported 140 deaths from ALS** but the researchers were unable to confirm most cases because ALS was not cited on the death certificates. Including these would have biased the study because national and state comparison figures were exclusively based on death certificate data, Dr. Armon explained.

A relative's take on the case:¹⁴

Well I have been doing research on ALS since 99 and I am 98% positive it is cause by the environment. My father passed from this back in Jan 01. Along with 112 people from his building at Kelly AFB in San Antonio. The numbers are high also in MS, Parkinson's, Alzh. There is a huge cluster of neurological diseases coming out of Kelly AFB. They closed Kelly down shortly after my

¹³ http://journals.lww.com/neurotodayonline/Fulltext/2003/02000/No_Excess_Als_Deaths_At_Kelly_Air_Force_Base..12.aspx

¹⁴ <http://www.godlikeproductions.com/forum1/message1449360/pg1> (spelling corrected)

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father passed and the first building they torn down was my Dad's.

Even though the health authorities fudged the numbers, the remaining 39 ALS deaths were still a big deal:¹⁵

Texas ALS Cluster Could Be 'Incredibly Important,' Experts Say

Renowned ALS researcher and clinician Hiroshi Mitsumoto says a recently identified cluster of 39 cases of ALS at Kelly Air Force Base in San Antonio, Texas, could be "incredibly important" in the quest to identify potential environmental and genetic factors underlying ALS.

Mitsumoto, who directs the Eleanor and Lou Gehrig MDA/ALS Center at Columbia-Presbyterian Medical Center in New York said that, while other ALS clusters have been identified over the years, "this is certainly, to my knowledge, a huge number, which is most unusual and which we cannot ignore.

"We need to investigate and not let this opportunity go by without finding out the cause. First, every effort should be made to see if those 39 patients are truly ALS or if other diseases have been included," he emphasized.

If the 39 current and former Kelly employees who are thought to have ALS do have the disease, Mitsumoto said, "My great hope is that the Defense Department will convene some kind of investigation team."

ALS, he says, doesn't have just one cause, "but multiple causes, all of which can lead to cell death in motor neurons."

Health authorities were quick to dismiss the notion of an ALS cluster, and ALS patient advocacy sites reported:¹⁶

No ALS 'Cluster' Found at Kelly Air Force Base

When 39 cases of ALS were identified in 1999 among current and former employees at Kelly Air Force Base in San Antonio, area residents and experts wondered what that might mean.

¹⁵ http://www.als-mda.org/publications/als/als5_6.html#texascluster

¹⁶ http://www.als-mda.org/publications/als/als7_10.html

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But a thorough investigation, conducted by the Air Force Institute for Environment, Safety and Occupational Health Risk Analysis, and the San Antonio Metropolitan Health District, has failed to confirm the existence of an ALS “cluster” at the base.

The study results are published in the November issue of the Journal of Occupational and Environmental Medicine.

Case closed! The US military did a "thorough" investigation and concluded with: "There is nothing to see here". The families of the deceased said that 140 base workers died of ALS but the Air Force disagreed. Who is right?

Such wiping under the carpet of extreme ALS clusters amounts to the deliberate sabotage of ALS research. The military is understandably not eager to assume legal responsibility for the deaths of over a hundred people, so it is understandable that they want to make the issue go away, in case the cause turns out to be exposure to toxic chemicals. However from an epidemiological research point of view, this is yet another major setback in finding the cause - or at least one major cause - of ALS.

ALS patients' groups expressed dismay at the way the study was conducted.¹⁷

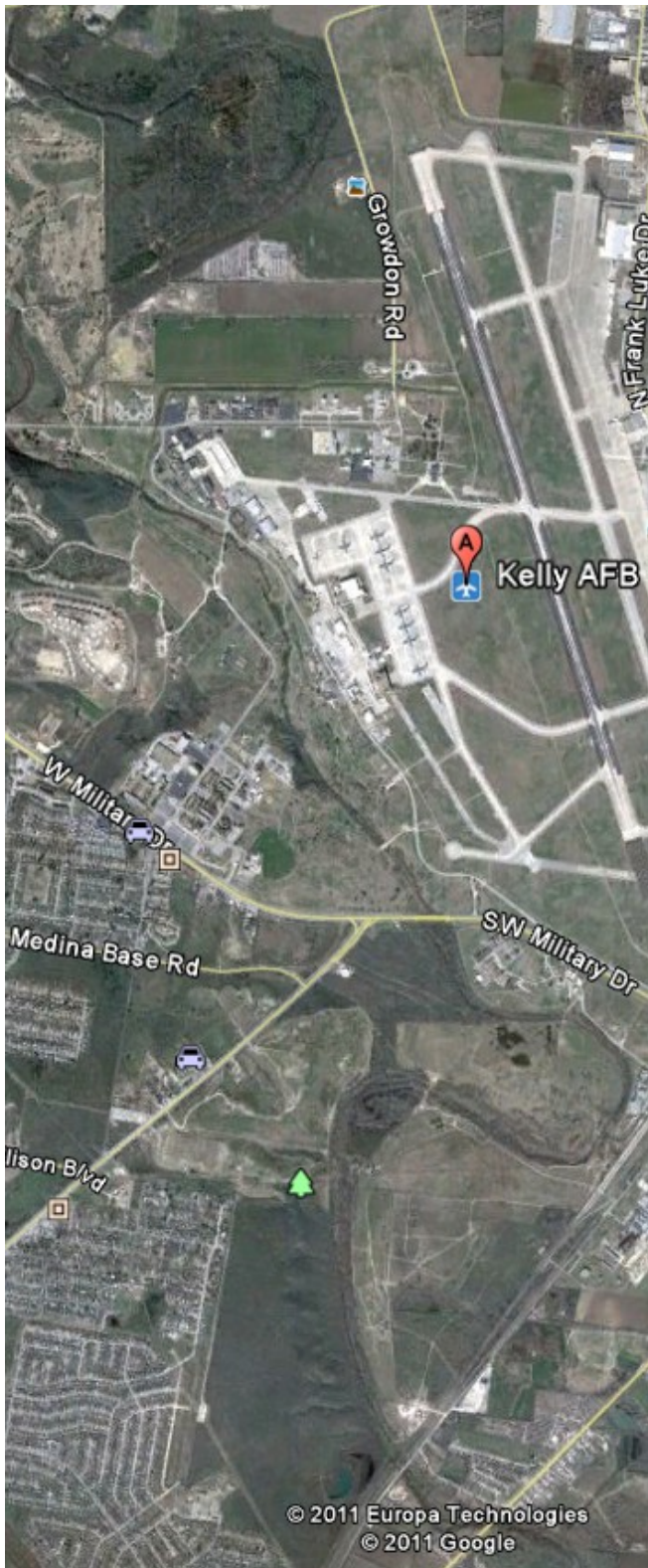
ALS Community Protests Kelly AFB Findings

A flurry of criticism followed the recent verdict that there's apparently no "ALS cluster" among workers at the former Kelly Air Force Base in San Antonio, Texas.

After results of the investigation were published in November in the Journal of Occupational and Environmental Medicine, some professionals and families in the ALS community expressed disappointment. Most of the criticisms centered on two points: that the study didn't include people living with ALS but only those who'd died of it; and that the study excluded all military personnel who had worked on the base, counting only civilians.

¹⁷ http://www.als-mda.org/publications/als/als8_2.html

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When we look at the former base, we see that its parking (upper left) is adjacent to a large wooded area through which a stream flows.

The entire base area is surrounded by and interspersed with grass lands and Pearsall Park, a large recreational area, is directly south of the base.¹⁸



¹⁸ <http://eventful.com/sanantonio/events/weekday-evening-meetup-pearsall-park-/E0-001-003245431-7>

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Frequent sliding with bare legs over grass increases risk of ALS sixfold

Italian soccer players have a six times greater chance of getting ALS than the average Italian¹⁹. This risk is unrelated to general physical activity or doping, because Italian basketball or volleyball²⁰ players have no increased risk. Neither is the risk related to just being outside a lot, because Italian cyclists also do not run an extra risk of ALS, according to Raffaele Guariniello, a Turin prosecutor who investigated the early deaths of more than 40 ex-soccer players from ALS²¹. His team could not yet identify a rugby player with ALS, but that was to be expected, since the number of rugby players in Italy is much less than the number of soccer players, so even if rugby players would run the same increased risk of ALS as soccer players, statistically, less than one would be expected to have died of the disease.

Some of the more well-known players who died are Adriano Lombardi, Stefano Borgonovo, Gianluca Signorini, Raffaello Guarinello, Giorgio Rognoni and Guido Vincenzi.

Borgonovo said in an interview that he thinks that a genetic flaw lead to his demise, but the evidence contradicts that. He did not suffer from familial ALS but from sporadic ALS - the type that is thought to occur randomly, and not six times more often amongst soccer players.

ALS does not just plague Italian soccer players: Playing soccer is just as risky for Brits: Three British men, carpet fitter George Pearce of Wash Common, electrician Graham Hodgetts of Thatcham and builder and carpenter Sam Brown of Kingsclere all played amateur soccer in the same league on the same soccer pitches at the Newbury and Basingstoke leagues from when they were teenagers until they were in their forties, and in later life died of ALS²². Statistically, this is unlikely in the extreme.

¹⁹ <http://www.unfictional.com/pdf/als-italian-soccer-players.pdf>

²⁰ http://www.timesonline.co.uk/tol/sport/football/european_football/article5114400.ece

²¹ <http://www.italymag.co.uk/italy/health/borgonovo-gehrig-shock>

²² <http://www.dailymail.co.uk/health/article-449144/Can-fit-raise-risk-motor-neurone-disease.html>

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It has already been established that merely "being fit" does not raise the risk of acquiring ALS as much as specifically spending a lot of time in nature or on grass. A study published in the American Academy of Neurology found that people who played university sports had a 1.7 times higher risk of developing the disease than those who did not. That is still several times less than the sixfold incidence of ALS amongst Italian soccer players.

There have been suggestions that heading a heavy ball could have initiated a cascade of brain damage leading to ALS, but there is no evidence in medical literature that boxers have a greater risk of developing ALS, making that hypothesis unlikely. And modern soccer balls are much lighter because they are coated to prevent rain water absorption - yet also in recent times, Italian soccer players continued to contract ALS at an alarming rate.

The "physical activity" hypothesis of ALS is based on solid data, but the conclusion that physical activity in general can contribute to causing ALS is unwarranted because it's still a tiny fraction of the physically active that go on to develop ALS. What should be looked at is the type of physical activity that results in the highest rates of ALS. Physically active people usually are active in many ways. They camp and hike as well as play tennis, swim and cycle. Clearly, some activities are much riskier than others. Soccer has been identified as the riskiest sport activity, so far.

What is unique about soccer is that players occasionally cause themselves tiny cuts and abrasions on the grass and dirt when they tackle an opponent. And even the shortest grass harbors ticks, since ticks normally dwell directly on the ground to avoid drying out, until they are ready for a blood meal - only then do they seek a suitable spot to attach to a host.

Countries where Lyme is very rare also have very low ALS incidence

Mexico has a 37 times lower ALS incidence than Finland²³, and Mexico also has an extremely much lower Lyme incidence as Finland.²⁴

²³ <http://www.unfictional.com/pdf/als-cluster-finland.pdf>

²⁴ <http://www.unfictional.com/pdf/lyme-mexico.pdf>

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Lyme hotspots are ALS hotspots

People who live around Mascoma Lake are 25 times more likely to get ALS²⁵ than other people in New Hampshire. When this fact became published by Neurologist Dr. Elijah Stommel of the Dartmouth-Hitchcock Medical Center, it caused quite a stir in the ALS research community. Mascoma Lake lies in New Hampshire. Let's look at the Lyme incidence in US states in 2008:

US States with the highest Lyme incidence in 2008

RANK	STATE	CASES PER 100,000	
1	New Hampshire	113	
2	Delaware	88	
3	Maine	66	
4	Vermont	58	
5	Pennsylvania	56	
6	Maryland	37	
7	New Jersey	32	
8	New York	29	
9	Minnesota	23	
10	Wisconsin	20	
11	Massachusetts	16	
12	Virginia	10	
13	California	0.6	

Based on MMWR Vol, 57 No. 53; Jan 9, 2009

Number of cases from ²⁶, using 2008 population table at ²⁷.

This shows us that people in New Hampshire run a very high risk of getting

²⁵ http://dartmed.dartmouth.edu/fall09/html/vs_als_cluster.php

²⁶ http://www.camlt.org/DL_web/988_lyme.html

²⁷ <http://www.infoplease.com/ipa/A0004986.html>

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Lyme disease - a chance of more than 0.1% every year, or roughly 10% over a lifetime, taken current trends into account for both life expectancy and reported Lyme cases.

Is it a coincidence that the United States' biggest ALS hotspot lies in the state where the risk of getting Lyme disease is highest? Is it a coincidence that other ALS clusters have been found²⁸ in Vermont and Maine, ranking resp. #3 and #4 in the US for Lyme disease incidence?

Adjacent to the south of Mascoma Lake lies the Enfield Wildlife Management Area:



²⁸ <http://www.vnews.com/06092009/5737158.htm>

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The Enfield Wildlife Management Area is a Lyme hotspot, hosting the white-tailed deer²⁹, a notorious reservoir³⁰ for Lyme borreliosis. The many ponds, marshes and open spaces make the Enfield Wildlife Area an endemic area for the ticks that transmit Lyme disease.

Much has been speculated about the cyanobacteria in the lake being to blame for the nine ALS deaths that occurred around it in recent times.

But what about the *Borrelia* bacteria from the deer, small rodents and songbirds from the adjacent forest? It seems strangely biased to peg the ALS cluster around the lake instead of around a major source of Lyme neuroborreliosis - a known cause of ALS. It seems that the researchers did not want to open any cans of worms and wisely steered clear of the highly politicized Lyme "controversy".

New Hampshire is indeed the number one Lyme hotspot in the United States, but until the summer of 2011, doctors were not allowed to treat Lyme disease with long-term antibiotics. If they did, they faced disciplinary action or even a revocation of their licence to practice:

N.H. doctors can now treat Lyme disease with long-term antibiotics³¹

DOVER — New Hampshire residents suffering from chronic Lyme disease will no longer have to worry about finding a doctor who will treat with long-term antibiotics.

A bill, HB 295, that states doctors are free to treat Lyme disease with long-term antibiotics and cannot be punished by the Board of Medicine because of such prescriptions was passed Thursday.

The bill's prime sponsor Gary Daniels, R-Milford, said the bill is an important step in helping both patients and doctors as it acknowledges chronic Lyme disease is a real ailment.

The text of the bill reads, "No licensee may be subject to disciplinary action solely for prescribing, administering, or dispensing long-term antibiotic therapy for a patient clinically diagnosed with Lyme disease, if diagnosis and

29 http://www.wildlife.state.nh.us/Wildlife/WMA/WMA_Enfield.htm

30 <http://www.unfictional.com/pdf/bb-white-tailed-deer-white-footed-mice.pdf>

31 http://www.fosters.com/apps/pbcs.dll/article?AID=/20110617/GJNEWS_01/706179908/-1/fosnews

WHEN ALS IS LYME

treatment has been documented and monitored in the physician's medical record for that patient."

Daniels said Thursday, "The problem we were encountering, and it seems to be a nationwide trend, is there are two standards for treatment and there seems to be favoritism over one standard that basically says there's no such thing as chronic Lyme disease."

He added that typically, if people have Lyme disease for longer than the four week average associated with the disease, they're instead treated for other diseases such as fibromyalgia or chronic fatigue syndrome.

"There can be such a thing as chronic Lyme disease and it can be treatable with long-term antibiotics," Daniels said.

He said doctors would often avoid treatment with long-term antibiotics due to fear of being brought up on charges by the Medical Board simply because it wasn't recognized as the appropriate treatment for Lyme disease. Patients would therefore have a hard time finding doctors in New Hampshire to treat the disease and would often seek help in other states.

"We're trying to set in place an environment where doctors are free to treat with long-term antibiotics and the bill says they can't be punished solely because they prescribe or administer long-term antibiotics," Daniels said.

The bill's language regarding long-term treatment with antibiotics is only in reference to Lyme disease.

Daniels said such a move just makes sense, especially when long-term antibiotics are already used to treat for many other diseases such as acne, and cancers.

New Hampshire, he said, has the highest incidence of Lyme disease per capita in the country, a statistic that played a large role in his sponsorship of the bill. He had been asked to bring the issue out into the open by a friend who suffered from Lyme disease for five years before she was diagnosed.

Daniels said the hope is Lyme disease and the instance of chronic Lyme disease will be better publicized and better understood because of this legislation.

WHEN ALS IS LYME

ALS clusters prove an environmental, not genetic cause

A French study found nine conjugal cases of ALS, of which at least eight cases were sporadic ALS³². **Statistically speaking, this was highly unlikely if ALS is directly or indirectly caused by genetical factors**, because the partners were not related to each other. What remains as a plausible cause is either a local environmental toxin or an infectious agent. The geographical spread is relatively wide. It seems more likely that an infectious disease such as Lyme disease is responsible than some mysterious toxin that only affects a few couples here & there. Lyme disease is highly endemic in France and the couples could have been exposed to ticks or other vectors in their immediate surroundings. From the study:

Subsequently, 2.5 couples were expected. If such results as a whole cannot strictly exclude a chance association, it must be stressed that for the département of Drôme, the estimated number of conjugal cases is 0.17 (estimated number each year x number of years of observation x population of Drôme in millions: $0.016 \times 24 \times 0.438$) or **20-fold less than observed herein**, and the estimated number of conjugal cases for the city of Valence is 0.024 ($0.016 \times 24 \times 0.064$), **which is 100-fold less** than in our observations.

A Clustering of Conjugal Amyotrophic Lateral Sclerosis in Southeastern France. Philippe Corcia, MD, PhD; Helene-Farnase Jafari-Schluep, MD; Dominique Lardillier, MD; Hassan Mazyad, MD; Pieric Giraud, MD; Pierre Clavelou, MD, PhD; Jean Pouget, MD; William Camu, MD, PhD.

Arch Neurol. 2003;60:553-557.

32 <http://archneur.ama-assn.org/cgi/content/full/60/4/553>

WHEN ALS IS LYME

Most ALS patients have classical symptoms of Neuroborreliosis

It has always been a persistent myth that ALS is a "motor neuron disease". This has recently been shown not to be true. In reality, most people with ALS suffer brain damage throughout the brain - it's only that the damage to motor neurons is much more quantifiable and life-threatening than damage to the rest of the brain. In up to 15% of cases, the miscellaneous brain damage is so severe that it leads to a dual diagnosis such as ALS-dementia, but in an additional 50% of ALS patients, there is still so much brain damage to the rest of the brain that it can be detected in a brain MRI^{33 34}. **Research shows that at least two third of ALS patients suffer generalized brain damage in addition to motor neuron damage.**³⁵ Generalized neurological damage to cognition and behavior is typical for Lyme neuroborreliosis - and this is exactly the damage done to at least two thirds of ALS patients - it's just that their paralysis overshadows it.

Sanjay Kalra, a researcher in the faculty's Division of Neurology and a practising neurologist, has published two papers in 2011 in the American Journal of Neuroradiology, providing evidence that ALS affects more than just the motor cortex, the part of the brain responsible for motor function.

"ALS was previously thought to be a disease restricted to the motor system causing only weakness," says Kalra, the principal investigator in both peer-reviewed papers. "But a significant proportion of people with ALS also have cognitive and behavioural changes."¹⁶

If ALS is a manifestation of Lyme neuroborreliosis, one would indeed expect CNS damage in ALS patients not to be limited to motor neurons.

33 N. Sudharshan, C. Hanstock, B. Hui, T. Pyra, W. Johnston, S. Kalra. Degeneration of the Mid-Cingulate Cortex in Amyotrophic Lateral Sclerosis Detected In Vivo with MR Spectroscopy. *American Journal of Neuroradiology*, 2010; 32 (2): 403 DOI: [10.3174/ajnr.A2289](https://doi.org/10.3174/ajnr.A2289)

34 U. Usman, C. Choi, R. Camicioli, P. Seres, M. Lynch, R. Sekhon, W. Johnston, S. Kalra. Mesial Prefrontal Cortex Degeneration in Amyotrophic Lateral Sclerosis: A High-Field Proton MR Spectroscopy Study. *American Journal of Neuroradiology*, 2011; DOI: [10.3174/ajnr.A2590](https://doi.org/10.3174/ajnr.A2590)

35 <http://www.sciencedaily.com/releases/2011/09/110916131252.htm>

WHEN ALS IS LYME

Hard evidence links Lyme to ALS

On ALS forums, it is almost common knowledge that "Everyone tests positive for Lyme" when the tests are sensitive enough, such as with the IGeneX test, that also includes bands 31 and 34, which are most specific for Borrelia and are excluded by other Lyme tests manufacturers. The below two ALS patients on als.net had positive Lyme tests:

Topic: interesting?? dr.matz	
RL Schafferr	Posted: Tuesday, March 15, 2011 12:57:55 PM
I agree, Fernando..antibiotics could speed up ALS as already proven in some..and they will never be a fix..Ceftriaxone, and others like it are a waste of time and resources This Lyme angle has been tried by many with ALS and it's a long shot at best that the two diseases are the same..all Lyme tests come back positive..mine did.. And I had 3 from reputable labs . But I didn't have Lyme ..	
Topic: interesting?? dr.matz	
GusGargoyle	Posted: Tuesday, March 15, 2011 7:36:50 AM
I do beleive that the cause is the cure. But you got to be out in time. I went 7 years with my Lyme before getting treatment and this is the bulbar kind. I think it is hard to get out of this trouble mostly because the muscles in the throat and face are so sensitive. Hope is what I got, but I am afraid that the process once initiated is hard to stop, I shall not lose faith until that time comes though.	

Posters "Bubba" and "Debbie", "PatMurray12" are also Lyme-positive on that forum, as well as many others. On other ALS forums we see the same situation:³⁶

10-24-2010, 09:46 PM

#1 (permalink)

[Degenhardt](#)
New Member (Say Hi)
Registered Member
Join Date: 2010
City: Fernandina Beach
State: Florida
Country: USA
Diagnosed: 00/0000
Posts: 22

Has any one else had lyme disease followed by ALS?

+1 0

In 2003-2004 I had **lyme** disease. I have heard from another person whose brother died of ALS that he had **lyme** prior to ALS. I do not have **lyme** now. I do have a new diagnosis of ALS.

Like Be the first of your friends to like this.

..and this one:³⁷

11-22-2010, 07:30 AM

#8 (permalink)

[davikingdawn](#)
New Member (Say Hi)
Registered Member
Join Date: 2010
City: Chico
State: CA
Country: US
Diagnosed: 00/0000
Posts: 4

Re: Misdiagnosing ALS

I had to be tested three times, before the **lyme** was found, it is very tricky to find. They finally found it on the Western Blot test. Make sure you find yourself a **lyme** literate doctor and ask for the Western Blot. I know that recovery depends on how quickly the **lyme** is found. I have read that 20% of **lyme** patients that go for a year without treatment, end up never recovering and die a death very similar to ALS. You need to understand **lyme** is a very "political " disease. Many doctors and ALL insurance companies would much rather make you an "ALS" patient than a "**Lyme** " patient , it is due to the financial drain of treatment for **lyme**, and no treatment (no cost) for ALS. I am on IV antibiotics now (Rocephin) and making tremendous recovery. But, I have to pay for it out of pocket as the Blue Cross policy I have will not pay for it....it is costing me \$3,000.00 a month for the IV antibiotics. If a person cannot afford this, I guess they just die....I think that is what the insurance companies hope for. Best wishes to all of you!

³⁶ <http://www.alsforums.com/forum/people-als-pals/13574-has-any-one-else-had-lyme-disease-followed-als.html#post143823>

³⁷ <http://www.alsforums.com/forum/als-healthcare-professionals/13709-misdiagnosing-als.html#post147135>

WHEN ALS IS LYME

..and a friend of this one:³⁸

06-14-2011, 01:54 PM

#50 (permalink)

IluvNY
New Member (Say Hi)
Registered Member
Join Date: 2011
City: montclair
State: nj
Country: US
Diagnosed: 05/2011
Posts: 6

Re: Misdiagnosing ALS


Hi all, a friend was diagnosed with ALS a few weeks ago. I was and still in shock considering the odds of him getting this fatal disease. Since he gave me the devastating news 3 days ago, I have been researching and simply trying to help, i feel the need to do something. After some research, i have questions and concerns. He shared his report with me from the Neurologist and after researching and seeing the info regarding ALS and Lyme disease, it caught my attention that it states in the neurologist's report that he tested positive for Lyme IgG. Is Lyme IgG the same as Lyme disease? If so, why was he not prescribed meds to treat the Lyme disease? His symptoms are weakness & numbness in hands, difficulty walking up and downstairs and balance issues. I'm not sure what other info is needed to offer advice/suggestions for my friend. Please let me know your thoughts re: the pos testing of lyme IgG and let me know what other information I can provide that may be helpful.

..and someone's nurse's friend's husband (very troubling..):³⁹

03-09-2010, 10:29 PM

#15 (permalink)

limegreenphysicist
Member
Registered Member


Join Date: 2010
City: Auburn
State: Alabama
Country: USA
Diagnosed: 00/0000
Posts: 243

Re: Lyme Testing?

Hey buddy,hi


I think they are concerned with my severe hyperflexia, muscle spasms, and weakness progressing in my leg. Even though, as the days go on, my symptoms are becoming more systematic rather than focal. One thing they noted about my hyperflexia is that is is symmetrical so hence the order for the cervical and thoracic spine MRI to check for MS lesions or compression. Also one of my doctors, was caught up in a sticky situation in which my nurses friends husband was diagnosed with **Lyme's** disease then ALS only to be re-diagnosed with Lymes. By the time they got back on the right track with **Lyme's** he was already too far gone and is not being treated for Lymes and is bedridden. Which scares me but hey one day at a time. I have 2 clean EMGs thus far! I get one every time I go to neuro again very thankful my neuro is my bffs hubby 😊 He is putting his bets in MS he has many ALS patients and he says he would know instantly when sticking me with the EMG if I had LMN damage.

This person has "Lyme and ALS":⁴⁰

02-20-2010, 01:40 PM

#8 (permalink)

Phil M
Senior Member
Registered Member


Join Date: 2008
City: Snellville
State: Ga
Country: US
Diagnosed: 5/2008
Posts: 632

Re: ALS/Lyme Help

I thought for sure I had Lymes when I started having symptoms. I had Lyme symptoms for years, and in the summer of 07, I got bit by 2 ticks, and the bite sites for both of them swelled up, and turned red. I'm not sure if there was a ring. Shortly after I had a really high fever that only lasted about 24 hours. Anyway, about 2 weeks later my speech started slurring. My doc did a lyme treatment, but only the 10 day oxy. Long story short. I end up seeing a neuro. He said lyme or no lyme, you still have ALS.

38 <http://www.alsforums.com/forum/als-healthcare-professionals/13709-misdiagnosing-als-4.html#post173995>

39 <http://www.alsforums.com/forum/do-i-have-als-als/10759-lyme-testing.html#post112700>

40 <http://www.alsforums.com/forum/do-i-have-als-als/10532-als-lyme-help.html#post110275>

WHEN ALS IS LYME

This woman was treated for Lyme in her teens and now has Lyme and ALS:⁴¹


10-08-2008, 08:19 PM

#1 (permalink)

elemkay80

New Member (Say Hi)

Registered Member



Join Date: 2008

City: Richmond

State: Virginia

Country: USA

Diagnosed: 10/2008

Posts: 29

🤔 Lyme vs. ALS - a bit confused!

+1 0

Ok, so, I spoke with my first neurologist today and got interesting news. My **Lyme** test is positive, but he said not to get my hopes, that he expected it would be (?).

I had a confirmed **Lyme** tick bite when I was about 10 or 11yo (I'm almost 28 now) when I was at summer camp. Had the bulls eye rash on my arm and everything. Went to my pediatrician about a week after returning from camp when we noticed this bite looked different from my multitude of other bug bites. I was put on antibiotics and that was the end of that. Never thought about it again, other than keeping in my head that it had happened. The rash went away and I never had a known symptom.

So now I'm being diagnosed with ALS 17 years later? Everything I'm reading is saying one can be misdiagnosed for another, but wouldn't that mean the bite would have had to have been more recent? I'm not reading anything that says antibodies would remain so many years later, especially if treated with antibiotics early enough. I guess a false-positive may be possible, but it's hard to imagine.

I don't know what I'm getting at really, just sort of trying to talk this out to organize my thoughts and see if anyone else has any ideas? I'm a little impatient in waiting for my 2nd opinion which is coming in November, and they'll probably give me more information, but I want to know now! I'm so not good at waiting. *sigh*

-Michelle

ALS patients who test positive for Lyme generally accept it as a coincidence or at best theorize that having been exposed to the bacterium may have predisposed them somehow towards ALS. "If ALS would be Lyme disease, the various antibiotics trials for ALS would have shown some success", they say - **totally ignoring the fact that those trials used doses and treatment periods that have been reported by Dr. David Martz to be wholly inadequate in clearing up his Lyme-ALS.**

Others say that since they never were bitten by a tick, they can't possibly have Lyme - even though 70% of diagnosed, CDC-reported Lyme patients do not remember a tick bite. It may very well be that Lyme can be transmitted by additional means. In 2009, a Florida lab found mosquitoes carrying the Lyme spirochete. Fleas are carriers as well. Lida Mattmann found *Borrelia* spirochetes in tears. *Borrelia* has been found in perspiration, the placenta, blood and semen. *Borrelia* bacteria have even been found in dust from Africa.

⁴¹ <http://www.alsforums.com/forum/general-discussion-about-als-mnd/6498-lyme-vs-als-bit-confused.html#post56334>

WHEN ALS IS LYME

Study found nine in ten ALS patients infected with Lyme bacteria

There have been several studies that found a strong link between Lyme disease and Amyotrophic Lateral Sclerosis. The Halperin paper is one. The Halperin paper found Lyme infection in nearly all ALS patients in their study group, but its authors dismissed this as a "coincidence" and even fudged the numbers in their summary to make the Lyme infection rate seem "only" 47%.

Not too long ago, scientific research papers were freely available online but nowadays, most are zealously guarded by a few publishing conglomerates that charge about the same for an article as you would expect to pay for the latest music DVD of a superstar. When you purchase, you give them your name, address, email address and IP address and have to agree not to reproduce the article. The whole system is designed to keep medical breakthroughs secret to the public in general and journalists in particular, and to intimidate and prosecute those who seek to uncover such secret knowledge.

No wonder, that the average person with ALS has no inkling of the fact that there is solid research, showing that ALS is strongly associated with Lyme disease.

We reproduce part of the Halperin paper here in "fair use":

“Immunologic Reactivity Against *Borrelia burgdorferi* in Patients With Motor Neuron Disease” by Halperin, et al., *Archives of Neurology*, May 1990, Volume 47, Number 5, pages 586-594.

Immunologic Reactivity Against *Borrelia burgdorferi* in Patients With Motor Neuron Disease

John J. Halperin, MD; Gary P. Kaplan, MD, PhD; Shari Brazinsky, MD; T. F. Tsai, MD; Teresa Cheng; Audrey Ironside, RPT; Priscilla Wu, MA; Joel Delfiner, MD; Marc Golightly, PhD; Robert H. Brown, MD; Raymond J. Dattwyler, MD; Benjamin J. Luft, MD

• Of 19 unselected patients with the diagnosis of amyotrophic lateral sclerosis (ALS) living in Suffolk County, New York (an area of high Lyme disease prevalence), 9 had serologic evidence of exposure to *Borrelia burgdorferi*; 4 of 38 matched controls were seropositive. Eight of 9 seropositive patients were male (8 of 12 male patients vs 2 of 24 controls). Rates of seropositivity were lower among patients with ALS from nonendemic areas. All patients had typical ALS; none had typical Lyme disease. Cerebrospinal fluid was examined in 24 ALS patients—3 (all with severe bulbar involvement) appeared to have intrathecal synthesis of anti-*B burgdorferi* antibody. Following therapy with antibiotics, 3 patients with predominantly lower motor neuron abnormalities appeared to improve, 3 with severe bulbar dysfunction deteriorated rapidly, and all others appeared unaffected. There appears to be a statistically significant association between ALS and immunoreactivity to *B burgdorferi*, at least among men living in hyperendemic areas.

(Arch Neurol. 1990;47:586-594)

Patients with Lyme borreliosis, a multisystem disease caused by infection with the tick-borne spirochete, *Borrelia burgdorferi*,^{1,3} frequently develop significant neurologic abnormalities.^{4,5} Peripheral neuropathies and radiculopathies often occur,⁸⁻¹⁰ producing symptoms lasting months to years if untreated. Fortunately, appropriate antibiotic treatment often leads to resolution of these problems. In contrast with Lyme borreliosis, amyotrophic lateral sclerosis (ALS) is a relatively uncommon, untreatable neurodegenerative disorder, the cause of which remains purely unknown.¹¹⁻¹² Several recent reports¹³⁻¹⁵ have considered the possibility of an association between Lyme disease and ALS, and a limited pilot study among our own patients with ALS suggested that a disproportionate number had immunologic evidence of exposure to *B burgdorferi*. In view of this finding, we undertook a more systematic, controlled study in an effort to address this issue.

PATIENTS AND METHODS

Study Design

Suffolk County, New York, consisting of the eastern two thirds of Long Island, is a mixed suburban-rural area with a high prevalence of Lyme borreliosis.¹⁶ Nassau County, New York, which occupies most of the western third of Long Island, is much more densely populated and developed, and has far fewer reported cases. To look for a possible association between Lyme borreliosis and ALS in a well-defined and unbiased population, we contacted all patients with the diagnosis of ALS, who were registered

with the Muscular Dystrophy Association (MDA) clinics in Suffolk and Nassau counties prior to the discussion in the popular press of a possible association between these two disorders. For each patient (Table 1), we obtained two control subjects matched for geographic area of residence, sex, and age (± 3 years in all but two patients, ± 5 years in the remaining two patients). Serum samples from all patients and controls were assayed (enzyme-linked immunosorbent assay [ELISA]) for anti-*B burgdorferi* antibodies. For comparison, serum samples were also obtained from patients carrying the diagnosis of ALS, who were followed in the MDA Clinic at Massachusetts General Hospital, Boston. Patients with the diagnosis of ALS who presented to the Suffolk County MDA Clinic (at University Hospital, Stony Brook, NY) following the assessment of this prospective cohort were also evaluated, but are considered separately. All patients in the prospectively defined cohort have now been followed either for at least 1 year, or until death.

Patients

All patients were evaluated by a neurologist, and underwent extensive testing, including neurologic examinations, radiologic or magnetic resonance imaging studies of the cervical spine, electromyographic studies, and blood tests including serologic tests for Lyme disease (ELISA) and syphilis (VDRL), and serum immunoelectrophoresis. All had signs and symptoms consistent with the diagnosis of ALS. Medical histories were reviewed for symptoms suggestive of Lyme borreliosis—tick bite, arthritis, meningitis, radicular pain, or erythema chronicum migrans (the pathognomonic cutaneous lesion of Lyme borreliosis). Cerebrospinal fluid (CSF) samples were obtained from 24 patients. (Fif-

Accepted for publication September 29, 1989.
From the Departments of Neurology (Drs Halperin, Kaplan, Brazinsky, and Delfiner and Mss Cheng and Wu), Pathology (Dr Golightly), Internal Medicine (Drs Dattwyler and Luft), and Physical Therapy (Ms Ironside), State University of New York, Stony Brook; Centers for Disease Control, Fort Collins, Colo (Dr Tsai); Department of Neurology, Nassau County Medical Center, East Meadow, NY (Dr Delfiner); and Department of Neurology, Massachusetts General Hospital, Boston (Dr Brown).

Reprint requests to Department of Neurology, HSC T12-020, State University of New York, Stony Brook, NY 11794 (Dr Halperin).

WHEN ALS IS LYME

21/24 were positive

teen of these patients were seropositive; 6 had evidence of cell-mediated immunity to *B burgdorferi*, but negative antibody titers.) Cerebrospinal fluid samples were examined for cell count; differential; concentrations of glucose, protein, and myelin basic protein; IgG index; Lyme titer; and oligoclonal bands, and samples were cultured for routine pathogens and for *B burgdorferi*.¹⁷ Lyme-seropositive patients received intravenous ceftriaxone (2 g/d for 14 days^{18,19}) to treat possible nervous system infection with *B burgdorferi*.

In each patient we attempted to determine if the earliest and most prominent difficulties were due to bulbar, upper motor neuron, or lower motor neuron dysfunction, and we attempted to provide an approximate quantification of the degree of involvement of each of these three elements when the patient was initially evaluated. For each of these three components of the disease, deficits were graded on a 0 to +++ scale: 0, normal; ±, mild symptoms but no objective abnormalities; +, perceptible appropriate findings on examination, but minimal functional limitation; ++, moderate functional limitation; and +++, severe functional limitation.

Group 1.—Twenty patients with the diagnosis of motor neuron disease (MND), living in Suffolk County, were identified. One could not be contacted, the other 19 agreed to participate (patients S1 through S19). All were seen in the Suffolk County MDA Clinic prior to their being aware of any possible association between Lyme borreliosis and MND. Each had a serologic test for Lyme disease (ELISA), and 13 had an assay of T-cell reactivity to *B burgdorferi*. Cerebrospinal fluid samples were obtained from 11 of 19 patients.

Group 2.—This case-control group consisted of 38 subjects, matched for sex, age, and area of residence. Each had a serologic test for Lyme disease (ELISA). Subjects were drawn from a combination of sources—friends and relatives of patients, hospital employees and their friends, and ambulatory patients followed at University Hospital, Stony Brook, NY, for problems that were neither neurologic nor rheumatologic. The seroprevalence in this group (10.5%) was comparable with that reported in several hyperendemic foci,^{20,21} and substantially more than the 5% to 6% seroprevalence rate in randomly selected specimens from healthy blood donors in Suffolk County (unpublished data).

Group 3.—Thirty-seven adult patients followed in the Suffolk County MDA/Neuromuscular Diseases Clinic with other neuromuscular diseases were randomly selected and underwent serologic testing for Lyme disease. This group included 1 patient with Guillain-Barré syndrome, 4 with post-polio syndrome, 4 with myasthenia gravis, 3 with familial neuropathies, 1 with chronic inflammatory demyelinating polyneuropathy, 12 with miscellaneous other neuropathies, 7 with muscular dystrophies, 1 with dermatomyositis, and 1 with Friedreich's ataxia. This group appeared to be a representative sample of the 82 adult patients (48 males and 34 females) followed at the same

Table 1.—Patient Characteristics*

	Group 1, Suffolk County, New York	Group 2, Control	Group 3, Other NMD	Group 4, Nassau County, New York	Group 5, Boston, Mass
N	19	38	37	14	19
M/F	12/7	24/14	21/16	5/9	11/8
Age, y					
Mean (SD)	59.2 (14.9)	58.3 (14.6)	46.7 (16.3)	59.6 (14.5)	61.2 (12.4)
Male	57.2 (16.4)	56.5 (16.1)	...	58.0 (13.9)	...
Female	62.7 (11.1)	61.4 (10.9)	...	60.6 (10.2)	...
No. of patients with positive Lyme titers (M, F)	9 (8,1)	4 (2,2)	1 (0,1)	3 (2,1)	1 (1,0)
No. of patients T-cell positive and titer negative (M, F)	2 (1,1)
Duration, mo (SD)					
Ab+	27.2 (50.9)
Ab+ excluding patient S10	15.4 (10.1)
Ab—	15.2 (7.5)

*NMD indicates neuromuscular diseases; Ab, antibody. Statistical comparisons for rate of seropositivity (Fisher's Exact Test) are as follows. Suffolk County patients vs controls: overall, $P = .0054$; males, $P = .0006$; females, not significant. Suffolk County patients vs Boston patients: $P = .0078$. Suffolk County patients vs other patients with NMD: $P = .0001$.

Table 2.—Clinical Characteristics of Patients Immunopositive for Lyme Borreliosis*

Patient No./Age, y/Sex						Duration, mo		Follow-up		
	UMN	LMN	Bulb	Sen	Onset Type	Before Evaluation	After Treat- ment	Condition	Duration, mo	
									After Onset	After Treat- ment
Seropositive										
S1/62/M	+	+	++	±	Bulb	9	16	Deceased	18	1
S4/82/M	±	++	—	—	LMN	17	29
S5/24/M	+	++	+	±	LMN	17	21	Static	36	14
S7/73/M	++	++	+++	±	Bulb	31	31	Improved	44	13
S10/70/M	+++	+	—	—	UMN	20 y	20 y	Static	21 y	22 y
S13/50/M	—	+++	+	—	LMN	48	48	Previously intubated†
S16/76/F	+	+++	+	+	LMN	30	30	Previously intubated†
S17/43/M	±	+	—	±	LMN	1	2	Improved	10	8
S19/65/M	—	+	—	—	LMN	6	6	Improved	18	12
S21/42/F	+	+	++	—	Bulb	3	3	Deceased	12	9
S23/66/M	?	+++	—	—	LMN	78	72	Previously intubated
N3/64/M	—	++	+	—	LMN	34	34	Deceased	43	10
N4/58/M	+	+++	±	—	LMN	42	42	Deceased	44	0.5
O1/66/F	++	+++	+	±	LMN	13	13	Deceased	18	4
O3/62/M	++	+	+++	±	Bulb	60	52	Worse	72	10
Seronegative, T-cell Positive										
S8/78/F	+++	++	++	+	UMN	19	32	Static	45	14
S9/53/M	++	+++	+	—	LMN	25	34	Intubated	36	3 d‡
S20/61/M	—	+	+	±	LMN	12	13	Worse	36	12
S24/55/M	+	+++	+++	—	Bulb	18	17	Deceased	21	4
S25/60/F	+	++	—	—	LMN	4	7	Worse	7	1
O2/65	LMN	?	?	Static	...	2

*UMN indicates upper motor neuron; LMN, lower motor neuron; Bulb, bulbar; and Sen, sensory. The column heading "Before Evaluation" indicates disease duration prior to our evaluation. Plus-minus sign indicates mild symptoms but no objective abnormalities; one plus sign, perceptible appropriate deficits on examination, but minimal functional limitation; two plus signs, moderate functional limitation; three plus signs, severe functional limitation; and question mark, unknown.

†Patient was intubated and followed up for a period greater than or equal to 12 months; patient was too physically limited to note a change.

‡Patient was intubated within 1 week of initiation of treatment; little change in patient's condition noted since then.

WHEN ALS IS LYME

The Halperin paper mentions in its first paragraph (green box) that nine out of nineteen ALS patients tested positive for Lyme. The notoriously unreliable ELISA test was used, a test with an unacceptably high proportion of false negatives. So that would be 47% of ALS patients testing positive for Lyme.

47% would be an astonishingly high number by itself, but the paper's authors - possibly afraid of the consequences to their careers if they were to pursue an "ALS is Lyme" angle, decided to "cook the books" and only casually mention, buried further in the paper (red boxes), that in fact it was 21 out of 24 ALS patients that tested Lyme-positive, making it 88%, or almost nine out of ten patients. Since the false-negative rate of the tests used is notoriously high, we are justified in concluding that most likely, every single ALS patient in their study was Lyme-positive.

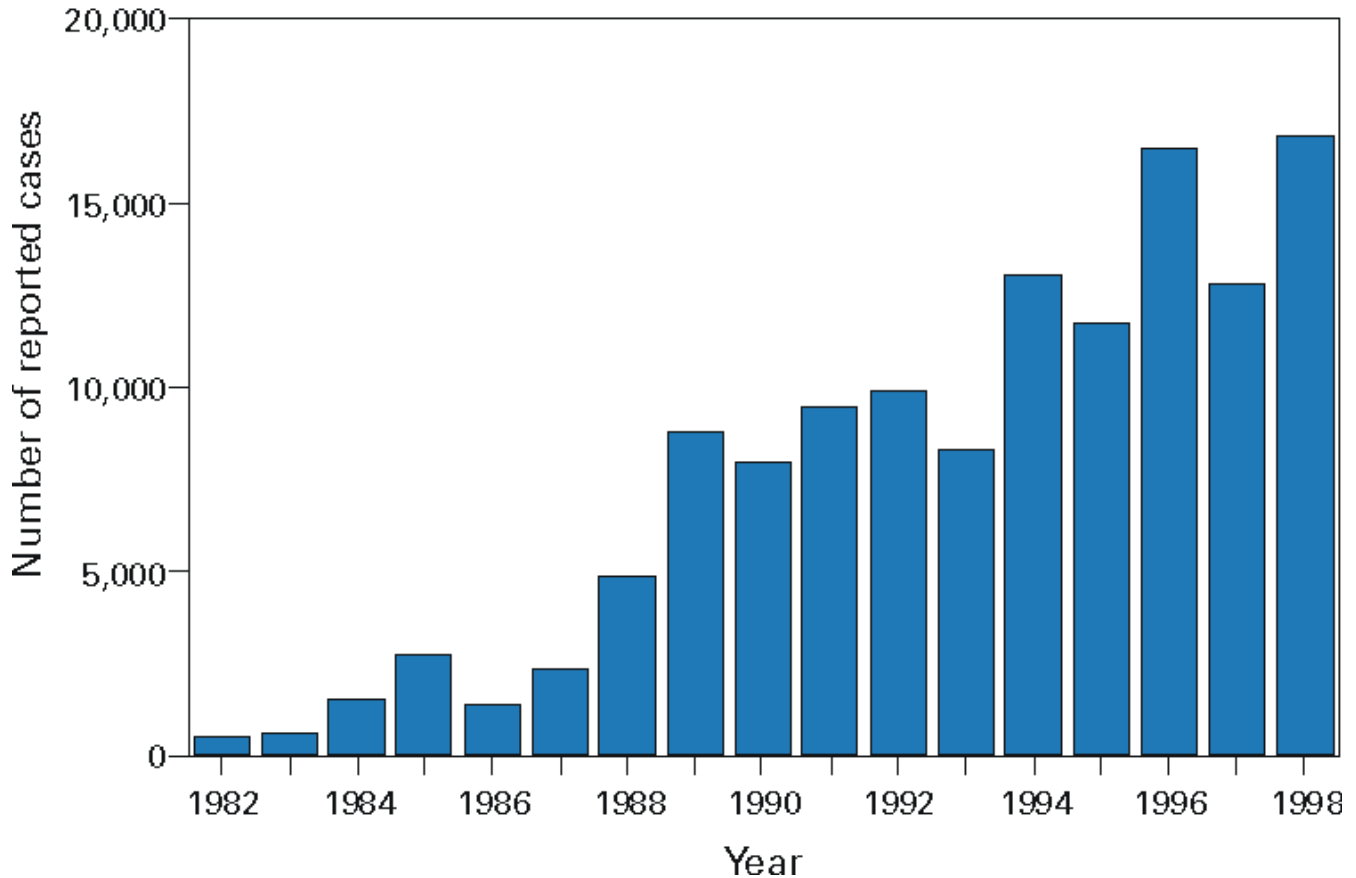
The authors decided to pretend that cell-mediated immunity to *Borrelia* did not count as "Lyme-positive", even though it is a certain indicator of internal exposure to the bacterium.⁴²

What was the likelihood, in 1990, the date of the study, of a person with ALS testing positive for Lyme disease? There were around 8,000 reported cases in 1990. IgM antibodies imply active Lyme infection, so we would expect to see only around 10,000 Americans testing positive for IgM antibodies. IgG antibodies last much longer, so we will take all reported cases from 1982 to 1990 into account, approx. 35,000 patients testing IgG positive. Reported cases are largely based on positive test outcomes rather than clinical diagnoses, so we do not have to correct for false positives. However we do have to correct for actual cases vs. reported cases. The most alarmist estimations now in 2011 are that there are ten times more Lyme cases than reported. Assuming that in 1990, Lyme infections occurred five times more often than reported to the CDC, we would expect $5 \times (8,000 + 35,000) = 215,000$ Americans to test positive for Lyme in 1990.

⁴² http://en.wikipedia.org/wiki/Cell-mediated_immunity

WHEN ALS IS LYME

FIGURE 1. Number of reported cases of Lyme disease — United States, 1982–1998



Source: CDC

In 1990, the US population was 249 million people. The percentage of people testing Lyme-positive would therefore be roughly estimated as $(215,000 / 249,000,000) * 100\% = 0.086\%$.

The mainstream medical establishment claims that chronic Lyme disease is very rare, and that Lyme disease certainly is generally not the cause of ALS. Therefore, according to their own statistics, if Lyme disease did not cause ALS, one would expect to see approx. 0.086% of ALS patients testing positive for Lyme disease. Instead, 87.5% of ALS patients tested positive in the Halperin paper. At least 21 out of 24 ALS patients had been infected with the *Borrelia spirochete*. False negatives are very common, false positives are not.

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Let that sink in for a moment. Instead of finding 0.086% Lyme-positive ALS patients, we find 87.5% Lyme-positive ALS patients in a large enough sample to be statistically significant. That is $87.5 / 0.086 = 1017$ times as many Lyme positive ALS patients as we expected to see. According to the Halperin study, American ALS patients have about a *thousand times* greater chance to be Lyme-positive than the average American. Not 1000 percent, mind you. 1000 times. That is a 100000% greater occurrence of being Lyme-positive. **A hundred thousand percent more.**

Critics may point out that in fact, about 1% of the US population tests positive for Lyme disease, instead of the statistics-based guesstimate of 0.086%. **That would still mean that the ALS patients from the Halperin paper were a hundred times more likely to be Lyme-positive than the general population.**

This means that which ever way you interpret the numbers, they state a solid case and that in the face of such overwhelming evidence, Lyme should be assumed the underlying cause of ALS. Absence of evidence is not evidence of absence, so a paper by the infamous anti-Lyme activist Dr. Steere who claims to have found no ALS patient testing positive for Lyme can not be considered counter-evidence, but merely evidence of ulterior motives.

We stress that unlike Atkinson-Barr's testimony, the Halperin paper is not merely anecdotal evidence by someone who is not even a medical doctor - it is a peer-reviewed, published medical study conducted by a team of qualified medical scientists, using control groups and other safeguards.

And even though their conclusion is that Lyme has nothing to do with ALS because they consider their findings to be "coincidental", we think their findings speak for themselves. Nine out of ten ALS patients were infected with the Lyme spirochete, whereas only one in a hundred random people are. Coincidence?

WHEN ALS IS LYME

Then why do the authors of the paper say that all those ALS patients Lyme-exposure is purely coincidental and inconsequential?

Very simple: Its principal scientist and author, Dr. Halperin, has a horse in the race. He is an important stateholder for the position that chronic Lyme neuroborreliosis is "exceedingly rare". It would be an unmitigated disaster for Mr. Halperin if this position of his would turn out to be false, because it would make him at least morally responsible for countless deaths of people he misdiagnosed based on a misguided opinion or refused to give live-saving treatment based on a misguided belief it would not work.

If Dr. Halperin would draw the only possible conclusion from his own work, he would have to change his opinion and admit to a career of quackery, he would have admitted responsibility to many deaths of misdiagnosed patients.

In 1993, Halperin told the New York Times⁴³ he did not believe in Chronic Neuroborreliosis being a problem at all: *And yet doctors are unsure which, if any, of them actually has active Lyme. Many of the country's leading Lyme experts believe that the number is microscopic. "I think persistent infection occurs but it is very, very rare," said Dr. John J. Halperin, professor of neurology at North Shore University Hospital on Long Island. "There are a lot of people being labeled chronic Lyme with very little evidence of it. They don't have Lyme and so they won't respond to a zillion months of antibiotics."*

Mr. Halperin has made a career out of crusading against the rights of Lyme patients. We can only guess at his motives. Some people, when they realize that their mistakes have caused death and suffering, choose to go into denial. Others rent themselves out as "expert witness", paid by insurance companies and peers accused of malpractice. Halperin is one of those⁴⁴.

⁴³ <http://www.nytimes.com/1993/08/24/science/lyme-disease-does-it-really-linger.html?src=pm>

⁴⁴ <http://www.amjmed.com/article/S0002-9343%2812%2900875-3/abstract>

WHEN ALS IS LYME

At least 12% of bulbar ALS patients have Borrelia bacteria in their brain

From the first paragraph of the Halperin paper:

"Cerebrospinal fluid was examined in 24 ALS patients - 3 (all with severe bulbar involvement) appeared to have intrathecal synthesis of anti-B burgdorferi antibody."

What does this mean in layman's terms? From Wikipedia:

"Intrathecal is an adjective that refers to something introduced into or occurring in the space under the arachnoid membrane of the brain or spinal cord. For example, intrathecal immunoglobulin production means production of this substance in the spinal cord."

So those three ALS patients with severe bulbar involvement had *Borrelia burgdorferi* bacteria inside their brain or spinal cord, because beneath the membrane protecting those organs, their immune system was producing antibodies against those bacteria. Less than 1% of the general population tests positive for Lyme, so 12.5% positivity clearly suggests actual infection instead of a statistical anomaly. It is unclear what test was used, but the false-negative rate of most commonly used Western Blot tests is up to 70%, which would mean that around a third of bulbar ALS patients would in fact suffer from Lyme neuroborreliosis.

The first paragraph of the Halperin paper tells us that at least 12% of bulbar ALS patients have evidence of *Borrelia bspirochetes* in their brains: *"Cerebrospinal fluid was examined in 24 ALS patients - 3 (all with severe bulbar involvement) appeared to have intrathecal synthesis of anti-B burgdorferi antibody."*

Around 25% of ALS starts as "bulbar" ALS, and those patients often deteriorate faster and often die from aspiration pneumonia. "Bulbar" means that the "sclerosis" happens in the lower motor neurons in the brain stem. Damage there causes problems swallowing, speaking and chewing.

Damage to lower motor neurons is much more acutely dangerous than upper motor neuron damage, because upper motor neurons are part of a redundant system, where pairs of upper motor neurons in both brain halves send signals to lower motor neurons in the brain stem. Single-sided loss of

WHEN ALS IS LYME

upper motor neurons therefore cause mere muscle weakness, while loss of lower motor neurons result in paralysis.

WHEN ALS IS LYME

There are many anecdotal reports of antibiotics helping against ALS

It is rare to find reports in medical literature of cases of Borreliosis-related cases of motor neuron disease that cleared up with antibiotics, but they do exist.⁴⁵ Anecdotal evidence abounds however:


<http://www.als.net/forum/Default.aspx?g=posts&t=48016>

pdii Rank: Member Groups: Member Joined: 9/4/2008 Posts: 27 Location:	Posted: Thursday, February 18, 2010 8:36:16 PM I have to chime in here, my brother was progressing inexorably, got on ceftriaxone, and his progression slowed dramatically - it has not stopped, unfortunately. NOT the study, he got this prescription from a Lyme disease specialist, but his doctor has been impressed enough to continue it for 9 months now. Yes, single incident and anecdotal but dammit I am convinced my brother would be dead today if he hadn't started ceftriaxone. Maybe the glutamate route isn't the problem for everyone, but it's either that or Lyme, and the most recalcitrant Lyme buggers should be flushed out after 10 months of antibiotics. Back to top Like Tweet 0
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<http://www.als.net/forum/Default.aspx?g=posts&t=44041&p=6>

Neph Rank: Newbie Groups: Member Joined: 7/1/2008 Posts: 34 Location: Germany	Posted: Thursday, January 22, 2009 5:04:17 PM I'd suggest a close reading of what he wrote and how he came up with 150, it is explained in detail. I did never refer to any aggressive antibiotic treatment, but of effective treatment. My mother has been treated with various antibiotics since December because of infections/pneumonia. What can I say? Her fasciculations have disappeared completely. My mum can move her left leg again, which was impossible before. Of course, this is sporadic and especially a placebo effect, you will say, though my mum has never even heard about anything that has to do with antibiotics and a possible positive effect in ALS. From a scientific point of view, tests on and treatments of 150 PALS is of much more value than anything else, especially if research focuses on mice that mimick fALS. You are talking about unfortunate stories. To me the real unfortunate story is the huge number of PALS that die every year.
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<http://www.alsforums.com/forum/members/notals!-page2.html#vmessage12705>

	NotALS! 08-29-2010 12:40 AM - permalink I'm a new member so I hope I'm posting this question correctly. I was ALS diagnosisid with ALS few months ago but have been on ceftriaxone and am feeling much better. I've regained strength in my legs, swallowing has much improved but the muscle atrophy in my shoulders is about the same. Does this mean I don't have ALS? I'm afraid to go back to a nuero. Has anyone else gotten better on antibiotics and then relapsed?
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⁴⁵ <http://www.unfictional.com/pdf/generalized-als-lyme.pdf>

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<http://www.alsforums.com/forum/als-healthcare-professionals/13709-misdiagnosing-als.html#post146202>

11-15-2010, 07:40 AM

#5 (permalink)

[davikingdawn](#)

New Member (Say Hi)
Registered Member
Join Date: 2010
City: Chico
State: CA
Country: US
Diagnosed: 00/0000
Posts: 4

 **Re: Misdiagnosing ALS**

Thanks for the responses and I wish all of you the best outcome. I was bitten by a tick back in May and in June started having neurological symptoms, mostly with my speech, as I mentioned before, eventually the horrible joint pain started up, the hair loss, the loud ringing in my ears, the night sweats. These are the symptoms that have nothing to do with ALS. I saw a total of 12 doctors before someone decided to treat me for Lyme disease and he tested me twice and it came back negative twice, but he was forward thinking enough to start me on the antibiotics anyway. I have been on antibiotics for three weeks now and I am 90% back to normal. The only deficit I have now, is my speech is still a slight problem and I get tired easily. My doctor is planning three more weeks of the oral antibiotic and will see if that knocks it out of my nervous system and my speech clears. If I was not having progress, my MD would not be doing this. I THANK GOD above, that this has worked for me. Getting a diagnosis of ALS is devastating. Have any of you looked into stem cell treatment for ALS? I managed to get on the trial list for California starting next year, that is before I realized I have Lyme disease. Best wishes to all!

<http://www.alsforums.com/forum/do-i-have-als-als/10532-als-lyme-help.html#post103744>

01-05-2010, 10:14 AM

#1 (permalink)

[Palmbeach](#)

New Member (Say Hi)
Registered Member
Join Date: 2009
City: Middleburg Heights
State: Ohio
Country: Uni
Diagnosed: 05/2007
Posts: 8

 **ALS/Lyme Help**


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I have looked around this forum for some time now but never posted. My husband was diagnosed 6/07. He has been confined to bed and wheelchair for over a year now. He can no longer speak or swallow. He has been on feeding tube since June 2009. He lost use of his arms/hands, he can move his legs but cannot stand. His neck muscles seem to be getting weaker also. He seldom holds his head up in the wheelchair. Extra saliva and nasal congestion are bad. Have several meds with no luck and had allergic reactions to a few. Muscle contractions and leg stiffness have been getting worse lately and making it difficult to transfer him by myself. He also was ALS diagnosis with **Lyme** disease and is being treated for it also. He had improvement on IV Rocephin but when he was taken off it because of improvement he went downhill fast. Has anyone else been treated for **Lyme** disease mimicking ALS? I would appreciate any ones input. Thanks, Bev

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Many people with ALS have Lyme disease

There is overwhelming anecdotal evidence of people with a double diagnosis of ALS and Lyme, even though if the diseases were unrelated, there should be almost no people who suffer from both. Apart from the many people diagnosed with ALS + Lyme whose identity is known and whose stories are highlighted in this book, there are many others, such as these forum participants:

 **MedHelp Member's Question** A A A

 **Is it ALS or Lyme?**
by 2young2die197, Jun 06, 2010 11:26AM

I have a clinical diagnosis of early ALS and late stage neuro Lyme. I'm currently on Rocephin IV and have improved greatly. I still have muscle weakness in my arms. Since my symptoms have improved so much, does that mean I don't have ALS? I've heard of patients improving on this drug only to die from ALS after stopping. Can I ever really be sure the ALS won't return?

Share this:  Facebook  Twitter 

Posted: Monday, August 01, 2011 12:17:25 PM

Hi - My name is Darlene, 61 years old, and I started in the summer of 2008 with occasional hoarseness and slight difficulty swallowig supplements. My PCP ordered a Barium Swallow which was normal in October '08. The changes have been very gradual but by February '09 I was slurring quite often. I had an EMG in March which was "slightly abnormal" but I had also developed twitches throughout my upper body and my tongue and chin. I was given the ALS diagnosis and a second opinion confirmed that I was a "possible", lowest on the scale of probability. I also tested positive for Lyme around the same time and have been on several antibiotics. I did have some temporary improvement but it didn't last. My swallowing really got difficult late last year and in February of this year I had a feeding tube installed. I have still only gained 5 lbs. and realize I may have waited too late to have it put in. My speech is completely unusable and I manage with a little voice machine (Dynawrite, used that my brother found on eBay for \$14!) and writing things down. My limbs work well but my thumbs and index fingers have become stiff and painful. Also have had some trouble with balance recently. I still drive, do my own shopping and maintain a household of critters.

But how about "hard evidence"? Can Lyme disease really cause symptoms that mimic ALS so much? Yes. There are plenty of medical publications from

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all over the world that show that Lyme indeed can cause ALS:

German doctors found a Lyme case causing ALS ⁴⁶

(Letter to the editor by various MD's from the dept. of Neurology and clinical neurophysiology of the University of Kiel in Germany)

The article talks about a patient with both upper and lower motor neuron disease. He tested positive for Lyme disease, in spite of not recalling a tick bite. Two weeks of Doxycycline and five days of Cefotaxime did not help him, but after having been given two weeks of Ceftriaxone he steadily improved and his diagnosis was changed from ALS to Lyme.

German doctors found another Lyme case causing ALS

This German patient displayed all the clinical signs of ALS but she had Lyme disease instead:

ALS-like sequelae in chronic neuroborreliosis. PMID: 7610670

Hänsel Y, Ackerl M, Stanek G.

Neurologischen Abteilung des Kaiser-Franz-Josef-Spitals, Wien.

CSF investigation in a 61-year old female patient with clinical picture of motoneuron disease gave evidence for chronic infection with *Borrelia burgdorferi*. Improvement of clinical and CSF findings could be observed after antibiotic therapy. The diagnosis of amyotrophic lateral sclerosis which was initially suspected had to be revised and the disorder was interpreted as chronic neuroborreliosis.

So there is ample evidence for Lyme masquerading as "ALS" and wrong diagnoses being made because of that. This is because the acronym ALS

⁴⁶ <http://www.unfictional.com/pdf/Lyme-causing-als.pdf>

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describes a symptom - not an illness. Several conditions can cause the same symptoms. Familial ALS for example is caused by free radical damage to motor neurons, as a result of a genetical defect. Therefore, familial ALS is wholly unrelated to sporadic ALS, for which the "experts" claim there is no known cause. Many people were initially diagnosed with ALS. But when they sought a second opinion from an infectious disease specialist knowledgeable about Lyme, they obtained the correct diagnosis of Lyme neuroborreliosis, received antibiotic treatment and their "ALS" disappeared.

Indeed, it is possible for Lyme neuroborreliosis to cause ALS. Let that sink in. There are people that show no "typical Lyme" symptoms except "classical ALS" symptoms. **Many of those people test negative for Lyme but respond very favorably to antibiotic treatment.**

Swedish doctors found a Lyme case causing ALS

Acta Neurol Scand. 1988 Sep;78(3):181-4. PMID: 3227803
CNS-borreliosis selectively affecting central motor neurons.

Fredrikson S, Link H.

Department of Neurology, Karolinska Institutet, Huddinge Hospital, Stockholm, Sweden.

A patient is described having *Borrelia burgdorferi* spirochetal infection clinically affecting central motor neurons selectively and without any sensory impairment. Diagnosis was based on elevated B. burgdorferi IgG antibody titers in cerebrospinal fluid (CSF) and titer normalization at clinical recovery. This occurred promptly and was complete after penicillin treatment despite 14 months of progressive central nervous system (CNS) dysfunction, favouring the hypothesis of the presence of the organism within the CNS. CSF findings characteristic of neuroborreliosis were registered, including parallel occurrence of mononuclear pleocytosis, severe blood-brain barrier damage and marked CSF IgM index elevation of prolonged duration. Some earlier reports of CNS manifestations related to B. burgdorferi are reviewed.

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US doctors found a Lyme case causing ALS

Dr. Marz did research on ALS patients found that many of them tested positive for Lyme and responded favorably to antibiotic treatment. Approximately 75% of ALS cases start with predominantly upper motor neuron involvement, and such a patient has been fully cured of his Lyme-induced ALS through antibiotic treatment:⁴⁷

Motor neuron disease recovery associated with IV ceftriaxone and anti-Babesia therapy.

Acta Neurol Scand. 2007 Feb;115(2):129-31. PMID:17212618

Motor neuron disease recovery associated with IV ceftriaxone and anti-Babesia therapy.

Harvey WT, **Martz D.**

Source: Rocky Mountain Chronic Disease Specialists, L.L.C., North Circle Drive, Colorado Springs, CO 80909, USA.

This report summarizes what we believe to be the first verifiable case of a significant and progressive motor neuron disease (MND) consistent with amyotrophic lateral sclerosis that resolved during treatment with i.v. ceftriaxone plus oral atovaquone and mefloquine. The rationale for use of these antibiotics was (i) positive testing for *Borrelia burgdorferi* and (ii) red blood cell ring forms consistent with *Babesia* species infection. The patient has continued to be free of MND signs and symptoms for 15 months, although some symptoms consistent with disseminated *Borreliosis* remain.

The patient tested negative on all commonly done Lyme tests. His blood was negative for both IgM and IgG antibodies. His spinal fluid also contained no antibodies against *Borrelia*, neither was there any *Borrelia* DNA detectable with a PCR test. His doctors however managed to detect *Borrelia* DNA fragments in the patients urine, after having administered antibiotics for the explicit purpose of killing off some bacteria so that their fragments could be

⁴⁷ <http://www.unfictional.com/pdf/als-cured-with-antibiotics.pdf>

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observed in the urine. **This illustrates the importance of doing an antibiotic-provocative urine PCR in order to avoid false-negatives.** When your doctor tells you: "We did a lot of tests and you are not infected with Lyme disease" this testifies to absolutely nothing else but the incompetence of the doctor, when antibiotics have not been used to the detection of the *Borrelia* DNA. Accepting such malpractice could amount to a self-imposed death sentence, so by all means, move heaven and Earth to get *properly* diagnosed. Nearly all doctors will be unwilling to diagnose properly - fire them at the first sign of incompetence because your life is at stake. In case of ALS, it is essential to find a *real* specialist - someone aware of the proven medical fact that brain infections can cause ALS.

The patient also had a *Babesia* infection, but *Babesia* is not known to destroy neurons - it affects red blood cells. Treatment was given with an extraordinary high dosage of Ceftriaxone plus Atovaquone against the *Babesia*. It can not be ruled out that there was a synergistic effect in *Borrelia* killing-kinetics due to the Atovaquone, but there have been anecdotal reports of Ceftriaxone alone being able to improve ALS symptoms, so the main thing is to get treated with at least 4 g/day of Ceftriaxone.

Another hugely important aspect of this case is how long it took for the patient to improve substantially. Ten weeks before it became "unmistakable". Implying that even after a month on highly-dosed Ceftriaxone, there was very little improvement. Certainly not enough to be able to ascertain any kind of beneficial effect of the antibiotics. **The sad truth is that most trials done with antibiotics on ALS patients has been done with 2 g ceftriaxone/day over two weeks. If those cases were anything like this one, there would have been no improvement whatsoever.** And that is often the outcome in such trials, with most ALS patients. Or patients get worse, because instead of killing the bacteria, a below-MIC dose, insufficient to at least temporarily inactivate the bacteria, merely agitates them, trying to blindly swim away to safety, drilling through the neurons, synapses and myelin in their path.

A pity many patients in such trials were never given a real chance. It seems

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those trials were done to "prove a point" - to prove the opinion of insurance companies, who say that ALS should not be treated for many months with very expensive antibiotic treatment. Insurance companies claim that ALS patients are supposed to just die, instead of running up bills for hundreds of thousands of dollars in treatment with IV antibiotics. Note how the patient in the study, after his ALS symptoms disappeared, still needed many more months of treatment. It typically costs tens of thousands of dollars per month to receive intravenous treatment with antibiotics.

There are more documented cases where antibiotics improved ALS, such as this one:

Smith LG

Improvement of patient with amyotrophic lateral sclerosis given ceftriaxone
Lancet 1992;339:1417

However, here a lower dose of antibiotics was used over a shorter time and the patient was not cured.

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People who cured their Lyme-caused ALS by taking antibiotics

William B⁴⁸

I am a 39 year old male, living in Maryland, who has been in the landscaping industry for 15 years.

In July 04 I was diagnosed with ALS by a Neurologist at GW, based solely on a clinical exam of symptoms and a **negative Lyme Titer** (standard ELISA blood test); all of my other tests, MRI's etc. showed nothing. **My Dr. blatantly denied my numerous pleas (and my wife's), refused further testing & antibiotic treatment for Lyme Disease, and even though I told him I was treated 4 to 5 years ago for Lyme Disease.** The neurologist told me of my life expectancy, prescribed me Rilutek and sent me and my wife off with a death sentence.

After two weeks on Rilutek, my condition declined rapidly, making it more difficult to breathe, swallow, and talk.

In early August '04 I went to a different Dr. who believed strongly that my condition was caused by the Lyme bacteria. He immediately took me off Rilutek, started me on antibiotic treatment, and ordered a cultured Lyme disease test through IGenex Lab. **After 5 - 7 days on antibiotics I experienced dramatic improvement, particularly with breathing, swallowing, improved speech and energy level.** My test results came back positive on all accounts, for Lyme Disease (DNA, CDC, & Western Blot).

If I didn't pursue my own research, I would have died of "ALS" without the opportunity being offered, and literally discouraged by my Dr., to find out if the deadly bacteria, *Borrelia burgdorferi* was present and possibly treatable.

How many lives are threatened, people dying, and families destroyed because they trusted their doctor's diagnosis, and they trusted the standard protocol, approved by FDA and CDC, for Lyme disease testing? How many ALS patients are being treated with Rilutek without knowing for certain if the Lyme Bacteria is present? Could Rilutek have progressed my Lyme disease to near death? This is a tragedy and this is America's new threat.

⁴⁸ <http://www.drgregorybach.com/7433/7559.html>

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Dr. David Martz ⁴⁹

After having been given only a few more years to live, oncologist Dr. Martz wanted to be absolutely sure he did not have Lyme disease instead of ALS, so he got himself tested. All tests were negative - even the tests performed by Igenex, the recognized experts in Lyme testing. Igenex has been much maligned by the "Chronic Lyme does not exist" crowd, claiming that they somehow rig their tests to yield a lot of false positives. We debunked that here:¹²⁴ It turned out that also Igenex' Lyme tests suffer from false negatives because Dr. Martz' symptoms were indeed caused by Lyme disease and it took unorthodox measures to eventually obtain a positive test. Lyme tests are very often negative when a patient has the neurological symptoms of late-stage Lyme, because there is no immune response in the CNS, the brain and spinal cord being "immune-privileged". Any antibodies from the original infection are either long gone or bound to the spirochetes in the brain or spinal cord. But Martz did not give up: He took five days of antibiotics to "shake up the spirochetes" and let his urine again be tested by Igenex. They found antigens to dead bacterial fragments in his urine, he received antibiotic treatment and the rest is history. Dr. Martz made a spectacular recovery. Dr. Martz, being a medical doctor, then began to treat patients with a variety of severe incurable neurological syndromes with antibiotics, and some results are available.

Among the patients he saw were 150 with lethal motor neuron disease, 650 with variants of Chronic Fatigue Syndrome, and a cluster of 40 Lyme patients from Northeast rural Colorado.

⁴⁹ <http://www.spirochicks.com/2010/04/recovered-als-patient-dave-martz-talks.html>

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Now, for Martz's research findings. Regarding the success of antibiotics on his patients, a nurse in Martz's practice compiled "soft data" [unofficial] from 523 of the patient charts:

- "Straight Chronic Lyme" - 72% better; 25% stable; 4% worse
- **ALS - 26% better**; 52% stable; 22% worse
- MS - 52% better; 38% stable; 10% worse

His observations:

- **Negative Borrelia Burgdorferi (Lyme) was not predictive of antibiotics failure**
- Non neurological symptoms may improve before CNS symptoms
- 50% had definite Herxheimer (worsening of existing symptoms) requiring dose reduction
- Muscle groups never recovered function if they were flaccid, but weakened muscles DID regain
- No one has ever been 100% cured
- Bed-bound patients do not benefit functionally
- Early diagnosis with minimal function loss improves best
- Some patients initially improve, then decline, then improve at LOW doses – his patients start very slowly with Rx
- Start LOW and GO SLOW seems better than HIT HARD and HIGH with antibiotics
- Labs for ESR and CRP were usually normal
- **There were very few complications from high and long-term doses of antibiotics**

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The most exciting finding is that up to 30% of ALS patients did not deteriorate while on antibiotics.⁵⁰

We note that Dr. Marz did not just managed to get himself cured of ALS, but that **he managed to improve the symptoms of 39 ALS patients, or 26%.**

Of course, the above claims are far removed from being any kind of reliable scientific data, so we just have to take them at face value. Dr. Martz announced that he will publish his findings soon.

If antibiotics could make a quarter of ALS patients permanently better and stop their deterioration, how many lives would that save? Wouldn't you like to find out whether antibiotics would help you, if you had been diagnosed with ALS?

⁵⁰ <http://vimeo.com/2074932>

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Ter⁵¹

I was diagnosed Bulbar ALS on August 15, 2005. My lung expansion was limited to 42%. I had fasciculations everywhere, even in my lower body. At the time I was riding an exercise bike 45 minutes a day 6 days a week. My neuro-guy told me to get my affairs in order.

In October I had a test for Lyme disease which came back positive. Since then I've been working with Lyme docs. They determined I have Lyme and 2 co-infections (Bartonella and Erlichia). The Lyme docs say the ALS diagnosis was an error.

I've been on antibiotics for 3 months and the excessive drooling has subsided. The number of fascilations I have is much less. In March I started using a bi-pap at night. Since then I have gained 10 pounds and gotten a little of the bounce back in my step.

My speech has regained tonal quality. I ride the bike and lift light weights twice a week. I have some PT hand putty that I play with when I'm at the office.

I'm looking forward to a full recovery.

⁵¹ <http://www.als.net/forum/Default.aspx?g=posts&m=289536>

Sue Massie ⁵²

Mysterious Symptoms for Years

At 42, with six lovely children and a wonderful husband, I thought my life was over! I was very ill with migraines, slurred speech, difficulty swallowing, atrophy in my muscles, excruciating pain throughout my body, memory loss, light and noise sensitivity, etc. These are just a few of the symptoms I suffered from on and off over the years, and they were progressively getting worse.

Paralyzed From The Neck Down

I eventually became paralyzed from the neck down, and developed an ALS-like condition. My husband was also very ill with debilitating symptoms including “buggy” eyes, migraine headaches, rib pain, radiating jaw pain, chest compression, fatigue and a racing heart (intermittent). We spent years trying a number of neurologists, cardiologists (including Yale), and all kinds of specialists, only to be given a new diagnosis with each visit. These included TIAs, Grave’s disease, possible MS, and even stress. Finally a Lyme-literate neighbor suggested my husband might have Lyme disease. I thought it was a ridiculous idea because my dad was supposedly the first case diagnosed back in 1980 in Monmouth County, New Jersey, and he was just fine (or so I thought). Our neighbor handed me the list of symptoms, and my husband had just about every one of them! I asked for her doctor’s name and we saw him immediately. He diagnosed my husband with Lyme disease and treated him with long-term antibiotic therapy. Six months after his diagnosis, I was also tested and diagnosed, and I started treatment as well. Five out of six of our children have now been diagnosed with Lyme disease and had to be put on a special educational program to help them with their studies. (Lyme can often affect children and contribute to ADD, ADHD, memory problems, dyslexia, anger outbursts, fatigue, etc.)

102 ALS Patients Tested Positive For Lyme - In Treatment

I have talked with over 8,000 people with Lyme. 102 of these cases are ALS-diagnosed patients who were properly tested for Lyme and came up positive. I feel that people who are diagnosed with ALS/Lou Gehrig's disease, Multiple

⁵² http://www.leaparizona.com/my_story_sue_massie.htm

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Sclerosis, Alzheimer's, Lupus, Fibromyalgia, Chronic Fatigue and many other neurological and degenerative diseases, could actually have Lyme disease. To date, there is no definitive, 100% positive test for Lyme disease. However, patients should request (from a Lyme-literate doctor) to have a PCR test (to determine genetic material of *Borrelia*) or a Western Blot blood test (antibody assay) done by Igenex Labs in California. Most doctors are following the diagnostic protocol of doing a Lyme titre or ELISA test, which are not accurate. If a patient who has Lyme disease actually tests positive using the Lyme titre or ELISA test, their doctor would then order a Western Blot blood test. According to the Center for Disease Control, a patient must have a minimum of five bands (specific numbers and bands are how they read these tests), in order to be labeled positive for Lyme by Western Blot. Another important consideration is that Lyme antibodies must be present for a positive result, and if the patient has been taking steroids, Advil, Motrin, or other anti-inflammatories or antibiotics, this could cause a false-negative result. For this reason, patients should be clear of all OTC's and prescription medications for a minimum of six weeks before testing, but even this cannot guarantee an accurate result.

My Lab Results Would Have Been Considered Negative

After testing, I only had one band - number 4I, which is the "flagellin" (or tail) of the spirochete, specific for *Borrelia* bacteria (Lyme), so I would have been told that I was negative. Quite often, patients have to be diagnosed by symptoms alone. I know that most people reading this story probably know of someone who has been to various doctors, and is suffering without an adequate diagnosis, and people just label them as being a hypochondriac, etc.

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Rob Cutler⁵³

I watched a recent television interview with Jodi Adamson, who has Lyme disease, and felt compelled to share my thoughts on her plight. She has lived with Lyme disease for six years and no doctor could tell her what was wrong.

European doctors have known about Lyme disease for 100 years. Our public health system must catch up quickly. Lyme disease is misdiagnosed as ALS and other diseases. I am living proof that our public health needs to change how it tests for Lyme disease.

In late September, I was told by medical experts that I had ALS, also known as Lou Gehrig's Disease. I had to take a leave from my job. It placed undue stress on my family and friends. I had been having strange symptoms that would last three or four days, then go away.

I researched Lyme disease and found I had about 75 per cent of the common symptoms. **For four months, I tried to get neurological experts to listen, to no avail. I got my blood tested by public health. It came back negative.**

Research has shown that the testing method public health uses is not very accurate. Fortunately, I found an infectious disease doctor in North York and the people at the ALS clinic realized I wasn't going to believe them until I got to see him. The doctor drew my blood and it was sent to a reliable lab in New York State at my expense.

I just received the results, which came back positive for Lyme disease. I have been on an antibiotic for three weeks and some of the symptoms have ceased.

Our health-care system needs to be knowledgeable about the symptoms and treatment of Lyme disease. The Eliza test needs to be replaced with the much more research-proven Western Blot. Ontario Health Minister Deb Matthews needs to have public health revamp its policies around Lyme disease testing.

The parts of the Canadian health system that I dealt with almost cost me my life. Stop the pain and suffering of many Canadians and get the testing method right.

⁵³ <http://www.thespec.com/opinion/letters/article/544610--catch-up-on-lyme-disease>

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Pamela O'Kane

By Dorothy Kupcha Leland, published on September 5, 2010 (abridged):

Four years after she almost died from Lyme disease, Pamela O'Kane is one of three Sacramento women competing for Team USA at the World Triathlon Championships this week in Budapest, Hungary.

It's the second year in a row she's qualified for Team USA. The 49-year-old lecturer from CSU Sacramento hopes there will be many more such adventures to come.

It's a far cry from how things looked in the spring of 2006. With her kids grown, a job she liked, and a love of athletic pursuits like running, swimming, and biking, she should have been on top of the world. Instead, she started experiencing weird symptoms her doctors couldn't explain.

In the spring of 2006, Pamela O'Kane was a 45-year-old lecturer at Cal State Sacramento, happily training future teachers in the School of Education. Her own kids were grown, and she spent a lot of her non-work hours in athletic pursuits--running, swimming, biking. She often raced with her sisters Colleen and Denise. Life looked good.

Then, weird stuff started happening. She began losing weight and muscle mass. She developed facial pain, jaw problems, difficulty speaking. Then trouble walking, twitching in her face, tingling in her arms and legs. Her doctors variously thought it might be MS, Guillain-Barre, or ALS. They could do nothing to help and her condition continued to deteriorate. By early December, there was doubt she'd live to see Christmas.

Then, O'Kane developed an ear infection and was given antibiotics. The medicine cleared up more than her ear problem. Suddenly, her whole body felt better. Within a week, she took part in a 5K race. ("Didn't do very well," she said. "But I crossed the finish line.")

Although her physical progress didn't last, the incident provided an important clue. Her mother and sister researched on the internet. What medical condition mimics diseases such as MS and ALS, and can be affected by antibiotics? The answer they stumbled upon: Lyme disease.

In early 2007, O'Kane saw a Lyme specialist in the bay area, who began

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treating her not only for Lyme, but for several additional tick-borne diseases. It was not an easy road. There were episodes of muscle seizures, speech difficulties, and generally feeling horrible. There were ups and downs in finding the right medications--some would help for a while, and then seem to lose their effectiveness. She spent time in a hospital, and then needed to re-learn how to walk.

It would be tempting to assume that O’Kane’s health problems are behind her. Surprisingly, that’s not true. While Lyme treatment helped immensely, she still must cope with residual neurological difficulties.

“I make involuntary moaning sounds while I’m running--can’t help it,” she says. “And I often have lots of shaking and trembling. I get strange looks from the other athletes. But you know what? That’s their problem, not mine.”

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Bart Fenolio⁵⁴

A month ago, Bart Fenolio was told he had Lou Gehrig's disease and had two months to live. Doctors advised his wife, Heidi, to take him home and call a hospice.

But Fenolio is proving the doctors wrong. Instead of getting worse, he's growing stronger each day, thanks to antibiotics. That's because he doesn't have Lou Gehrig's disease, which isn't curable. He has Lyme disease, which is.

Lyme disease, a bacterial illness spread by ticks, is a poorly understood and strangely controversial illness that has been sweeping the country since it was discovered in Connecticut in the 1970s. While still rare in California, there were 28,921 confirmed cases and 6,277 probable cases in the United States in 2008, nearly twice as many as in 1994.

But Lyme experts suspect there could be 10 times that many. That's because when not treated immediately, Lyme can hide in the body for years and then attack, masquerading as anything from heart disease to arthritis to lupus. Folks might not even know they'd been bitten. And the tests for Lyme disease are notoriously unreliable.

Dr. Raphael Stricker, a Lyme disease expert in San Francisco, regularly sees patients who have been misdiagnosed with chronic fatigue syndrome or Parkinson's disease.

"I saw a new patient the other day who had weird symptoms and had gone to the Mayo Clinic for a complete work-up," Stricker told me. "All they could come up with was fibromyalgia," a syndrome characterized by chronic pain, fatigue and depression. Stricker learned that the woman had grown up on Cape Cod, where Lyme-carrying ticks are common.

"How could you miss that little tidbit of her history?" he wondered.

Bitten in Morgan Hill

Fenolio, 69, knows just how he contracted the disease. Six years ago a healthy and hearty Fenolio was playing with his dog Cody near a percolation pond in Morgan Hill and was bitten by a tick. When a circular rash appeared around the bite, he went to the doctor. A Lyme test came back negative, and he

⁵⁴ <http://www.nwfpf.org/News.aspx?Item=3202>

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forgot all about it.

Three years later he retired from the tropical fish store —Dolphin Pet Village — he and his sister owned in Campbell. He and his wife moved to San Diego to be near their grandchildren and to enjoy playing lots of golf.

But his golf game slowly deteriorated. He couldn't seem to grip the club. Then, during a vacation in Hawaii, he was too weak to climb out of the pool. His doctor told him he was just getting old. His wife wasn't buying it.

"I said, 'This is not old age. My husband is disintegrating before my eyes, and something's going on.' "

Their Kaiser Permanente internist referred them to a neurologist, who diagnosed Lou Gehrig's disease. Then Fenolio's son remembered the tick bite.

Fighting for treatment

A laboratory that specializes in Lyme tests confirmed his suspicion, and a Lyme specialist in Redwood City prescribed a long-term course of antibiotics. But the ordeal wasn't over. Although Fenolio began to improve on antibiotics, his wife told me, Kaiser doctors wanted to discontinue them.

That's because the Infectious Disease Society of America still recommends against extended treatment using antibiotics, and it casts doubt on whether chronic Lyme disease exists at all, despite thousands of documented cases. Because of the IDSA's position, health insurers generally refuse to cover long-term antibiotics. In most states, though not in California, doctors can lose their licenses just for treating chronic Lyme.

Dr. Sara Cody of the Santa Clara County Health Department cautioned that Lyme disease is rare here, and Fenolio's case doesn't prove that there's rampant misdiagnosis going on.

"What he is experiencing is tragic but not common," she said.

Dr. Jonathan Blum, an infectious disease specialist at Kaiser Permanente Santa Clara, wouldn't discuss Fenolio's case. He confirmed that Kaiser follows the IDSA protocols.

"Long-term antibiotics can cause significant side effects," he said, "and should be used only if they are going to help the patient."

Fenolio's family is convinced that the antibiotics are helping. Today he is in a

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San Jose nursing home, improving each day. He knows there will be setbacks, but his wife hopes he'll be strong enough to go home in a couple of months.

"I just wouldn't want anyone else to go through this nightmare," she said. "If I had one of those diseases and was told there was no cure, I would definitely want to be tested for Lyme."

July 30, 2010

Bart Fenolio still tires easily, needs a walker to get around and has occasional memory lapses. He knows it could take years for him to overcome the devastating effects of chronic Lyme disease.

But for a 70-year-old guy who had been told he was dying of Lou Gehrig's disease and couldn't breathe on his own or pick up a cell phone when I saw him six months ago, Fenolio looks pretty darn good. And he is counting his blessings.

"I'm just so lucky to have a family and so many friends who could advocate for me," he told me Thursday as he prepared to leave the San Jose nursing home where he has been recovering since December. "I've lost a lot of muscle mass — I've got no buns — but I'm finally on the road to recovery."

In January, I told the story of the former San Jose State track star, classic-car nut and golfer who contracted Lyme disease from a tick bite but was misdiagnosed with amyotrophic lateral sclerosis, an incurable illness. The longtime owner of a Campbell tropical fish store was bitten by the tick while walking his dog in Morgan Hill seven years ago and began to develop symptoms two years ago.

Only through the persistence of Heidi, his wife of 46 years, and their scientist son and social-worker daughter was Bart given a series of Lyme tests that came back positive. He says his doctors at Kaiser Permanente insisted he had ALS even though he began to improve with antibiotics, and he left Kaiser to get the treatment he needed.

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Fast-growing disease

Lyme disease, a bacterial illness spread by ticks, is the fastest-growing infectious disease in the country, with 28,921 confirmed cases and 6,277 probable cases reported in 2008.

First identified in Connecticut in the 1970s, it still is not understood. Tests for Lyme are notoriously unreliable, and it can masquerade as Parkinson's disease or arthritis.

After I wrote about Fenolio's case, I was inundated with inquiries from people with similar stories and those who suspected they might have the disease. I have been referring them to the California Lyme Disease Association at www.lymedisease.org.

Since then the Fenolios, who retired to San Diego four years ago but came back to the Bay Area to find a Lyme specialist, have been living here, Bart in a nursing home and Heidi with friends in Saratoga. They have become strong advocates for Lyme education.

"We have met so many people who were misdiagnosed," Heidi said. "There's so much of it out there," Bart added, "but people don't realize it."

Legislation that would provide more money for research and testing for Lyme is inching its way through Congress. But advocates for patients with chronic Lyme suffered a blow in April when a federal review panel refused to endorse the use of long-term antibiotics, saying that treatment posed too great a risk of side effects.

The panel's decision infuriated Bart and Heidi because they are convinced that the antibiotics he has been taking since October saved his life.

"Why withhold treatment when you've already got a death sentence?" Heidi asked. She is confident that someday Lyme will get the attention it deserves. But for now, patients have to fight for treatment.

"I always say we're just five years too early," she said.

The disease has taken its toll on the family financially as well as physically. Medicare has picked up much of the cost of Bart's treatment, but the Fenolios have spent about \$100,000 of their savings.

"It's been a real journey," Heidi said, shaking her head. "I don't even know how to describe it. This week especially has been just horrible for him because he

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can't wait to get home."

At last, Bart's wait is over. This morning, he and Heidi are scheduled to fly to San Diego, where their daughter will meet them in Bart's 1939 Ford woodie wagon. A procession of woodies driven by members of the San Diego Woodie club will escort them home. Then Bart will be able to sit in his backyard by his koi pond, feel the ocean breezes, enjoy Heidi's veal scaloppine and sleep in his own bed for the first time in a year.

"It will be so good to get him home," Heidi said. "Just to be able to hold his hand during the night, I can't even tell you."

Bart died on December 16, 2010 from blood clots in legs and lungs.

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Tom Coffey⁵⁵

For months no one knew what was happening to Tom Coffey. In the spring of 2001 the then 34-year-old radio dispatcher was struck by high blood pressure and double vision. By summer's end he was suffering from facial palsy, crushing fatigue and joint pain so intense he walked with an old man's shuffle. Medical visits turned up nothing. By October his weight had plummeted 105 lbs., to 202.

"My doctor was at the end of his rope," says Coffey. "He kept referring me to different people."

When he awoke unable to swallow his saliva, Coffey rushed to a hospital near his Frederick, Md., home and was given blood tests and brain scans. Doctors returned with a terrifying diagnosis: ALS, or Lou Gehrig's disease, a degenerative illness likely to kill him within six months.

"Tom's dad said, 'I always thought he'd be burying me. Now I'll be burying him,'" says Coffey's wife, Tricia, 35. Hooked to a feeding tube, Coffey waited to die.

But a relative who thought his symptoms might have another cause suggested a trip to Dr. Greg Bach, a suburban Philadelphia Lyme disease specialist. The doctor found something everyone else had missed--a "bulls-eye" rash beneath his patient's hair. Coffey was suffering not from ALS but from a severe case of Lyme, which is spread to humans by tick bites.

For most people, Lyme manifests in a rash and flulike symptoms easily treated with antibiotics. Left undiagnosed, however, it can invade the nervous system.

"I always thought Lyme was no big deal," says Coffey, who rebounded after taking medication. "But it damn near killed me."

In fact Lyme is rarely fatal, but as Americans head outdoors during the peak infection months of May to July, experts warn that it can be devastating--and that the threat is growing. In the past decade the disease has spread from the Northeast to every state except Montana.

Last year the Centers for Disease Control reported 17,000 cases--more than double the number in 1990--but researchers like Joseph Piesman of the CDC say the actual incidence may be about 10 times higher.

⁵⁵ <http://www.angelfire.com/planet/lymedisease/7/TomC.html>

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The illness is also the subject of a growing debate. While most doctors believe that *Borrelia burgdorferi*, the tick-borne spirochete that causes Lyme, is quickly killed by medication, many patients complain of arthritis, irregular heartbeat, memory loss and motor-skill problems long after they have undergone the standard two-to-four-week treatment regimen. That has led some researchers to conclude that Lyme can return as a chronic illness in perhaps 10 percent of those thought to be cured.

"Lyme is much more serious than the public recognizes," says Dr. Brian Fallon, director of Columbia University's Lyme Disease Research Center. "People can have severe cognitive problems for the rest of their lives."

The medical establishment, however, remains unconvinced, and a few doctors have been penalized for their treatment of recurrent Lyme.

Pat Smith, head of the Lyme Disease Association, a nonprofit group calling for greater research on the disease, thinks pressure not to recognize the chronic form comes from insurance companies: "They don't want to pay."

No one disputes that late-stage Lyme is little understood. Tests are often effective only in early-stage infection, and while sufferers usually get a rash, they don't always notice it. Worse, many Lyme symptoms mimic other diseases: fibromyalgia, multiple sclerosis, even mental illness. That means other diseases may be mistakenly treated as Lyme--the crux of mainstream objections to the chronic-disease theory.

"People who test negatively for Lyme are still being given antibiotics to see what happens," says Dr. Gary Wormser, head of infectious diseases at New York Medical College.

But Fallon argues that a longer course of medication, given intravenously, may be needed for some patients. Coffey's doctor agrees.

"Half the patients I see have been inadequately treated," says Bach. "These people thought they were cured by being treated only a little."

Coffey, now 37, is taking no chances: He has remained on antibiotics for 14 months to be sure that his Lyme is really gone.

Still, he's grateful to have escaped his death sentence.

"I'm starting over again," he says. "From here on out, the rest is gravy."

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More about Tom Coffey in this interesting forum posting:⁵⁶

I came across this forum and post tonight while doing additional research for a patient who was recently diagnosed with ALS prior to learning they actually had Lyme disease and coinfections.

This patient is one of more than at least 200 people who have contacted me over the past few years who were told they had ALS after being exposed to ticks in Lyme endemic areas. A number of these patients came to me several years back from clinical trials for ALS which were being conducted at some well known universities. The patients or their families were not convinced the problems (symptoms) were actually stemming from ALS and had suspected it might be from Lyme disease and had contacted me for more tick borne disease information.

Since there seems to be some questions about the ALS/Lyme connection, I thought I'd register here and share some information that may assist some of the people reading or posting here.

I have had the pleasure of meeting Tom, have learned about his situation and watched his progress unfold.

Tom was originally diagnosed with ALS by several doctors at top universities. He had digressed to the point he was wheelchair bound, unable to sit up without restraints, had severe ALS symptoms and had a feeding tube. He was unable to speak or hold his head upright by the time his family took him to a doctor who works with people in the advanced stages of Lyme disease. His original doctors told the family there was nothing that could be done for Tom at that time.

When Tom first arrived at the Lyme doctor's office he had recently been sent home (from Johns Hopkins I believe) with approximately 2 weeks to live. Suspecting Lyme disease was the reason Tom's health had originally declined, his family wheeled him into the doctors office where he was given an exam and a history was taken. His doctor admitted there was little hope and was fairly sure nothing could be done to help him.

But he was tested for Lyme and tick borne coinfections and other conditions at the families request and the blood was sent out to various labs. Tom was

⁵⁶ <http://www.als.net/forum/Default.aspx?g=posts&t=44041&p=5>

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sent home with a clinical diagnosis of Lyme disease due to his history and symptoms after being given an injection of antibiotics for the condition and the family was told not to expect much due to the advanced stages Tom had reached by then.

By the way, Igenex Lab, the one some claim has "too many positive results" (rumors spread to discredit the competition for financial reasons, to discredit labs which could prove an improper original diagnosis and jeopardize physicians licenses and reputations who misdiagnosed patients) actually has a more specific test than other labs due to the fact it is able to detect more than one strain of *Borrelia*. Unlike the standard commercial labs which are often used and approved by insurance companies due to the cheaper prices and political reasons, Igenex tests detect exposure to both *Borrelia burgdorferi* (Lyme) and the more recently discovered *Borrelia lonestari* (STARI or Master's Disease). Since there are over 300 known strains of *Borrelia* that are missed by all commercial tests, Igenex is more accurate than most but it is not able to detect all strains of *Borrelia* or all cases of Lyme or Lyme-like diseases.

Eventually Tom's lab results arrived confirming the diagnosis and I honestly can't remember what co-infections were present so I won't speculate about them now. The doctor continued the treatment and Tom started to slowly improve after going through a time period where his symptoms were worse (typical with people infected by spirochetal bacteria such as Lyme- called a Herxheimer reaction).

All of this was documented in records and luckily on film, which I have seen. Tom made progress slowly but as expected due to the advanced condition.

Fast forward (sorry I am fading here)...Tom eventually was able to speak, walk and actually returned to his old job (and he looked so good at that time it still brings tears to my eyes to think about it). A while later I heard Tom and his wife were expecting a baby and starting a family.

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Dave Marr⁵⁷

I was dx'd definite ALS 3/00 and tested positive for Lyme on ELISA and Western Blot 2/01. On 4/18/01 I began an aggressive therapy for Lyme.

I started a six week course of IV Rocephin followed with oral Flagyl 500 mg tid and oral doxycycline 100 mg bid. My ALS symptoms started to diminish at day four of IV Rocephin with spasticity, cramping and vertigo disappearing. Within another three days clonus in ankles and jaw became almost non-existent as well as speech problems and swallowing difficulties.

Prior to IV Rocephin the spasticity and cramping kept me on my butt. My diaphragm would cramp violently if I sneezed. I also fell a lot prior to IV and managed face plants most of the time. I have not fallen once since abx. I also changed the oil and filter in my two cars and hand washed them.

During the six weeks of IV Rocephin my ALS symptoms were mostly gone. I was left with the muscle wasting that had occurred since onset.

There has been no new wasting as far as I can tell.

Being a smart-ass, I felt there was no need to combine oral flagyl and doxycycline, so I laid off the doxy and started with only the flagyl. Within four days of just oral flagyl my symptoms all returned, spasticity, cramping and clonus. I started doxy and within 24 hours those symptoms started to disappear.

It is now three months since the start of abx. I have no spasticity, cramping or clonus. My reflexes fall within normal range, they had become quite hyper. My FVC went from 68% prior to abx to 88% now. I have minor foot-drop in the left ankle and the right has none. Prior to abx I had AFO's for both feet as dorsiflexion was gone. My leg strength is increasing as I am now able to walk two miles/day and continue to shave time from the miles. I could not walk one mile prior to abx.

Basically, what I am left with is a pure lower motoneuron syndrome that has left me with atrophy. My left hand, arm, both shoulders, my neck and left leg have significant loss of muscle. My right leg has rebounded the best as it was not too affected. My right hand and arm are affected but they are also rebounding nicely.

⁵⁷ <http://www.angelfire.com/planet/lymedisease/7/DaveM.html>

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Kari Mayne⁵⁸

I contracted Lyme in February of 1988 - thirteen years ago. I was not diagnosed with lyme until August 1999, after being sick for 11 years (and having a stillborn child - lyme of course.) Before I was finally diagnosed with lyme I exhibited all the classic ALS symptoms, and had lost an uncle to ALS. Thus - the doctors thought they had an ALS diagnosis nailed down tight. Only through my stubbornness and refusal to accept the ALS dx did I research the internet and find an llmd. My first visit, even without labs, the doc - God bless him - said I had the worst case of lyme he had ever seen at that time. Subsequent lab tests ELISA and Western Blot both positive.

The symptoms which are now COMPLETELY GONE are:

- no more severe muscle cramps
- no more fasciculations
- no more numbness in face/tongue/forehead
- I can swallow without difficulty now
- no more slurring of speech
- no more limping and severe weakness in legs
- I can now pick up pennies off the floor - fine motor skills are back in hands
- I can lift my eyebrows now
- I don't choke on liquids
- Reynaud's syndrome is better
- No more intense heel/bottom of foot pain
- No more running into walls, doors because of poor balance or legs "giving out from under me"
- I can raise my arms to shampoo my hair
- No more pain in joints or numbness in limbs

(We snipped the non-ALS specific symptoms)

58 <http://www.lymeneteurope.org/forum/viewtopic.php?f=10&t=150>

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Pat Pepper^{59 60}

Pat Pepper says she was never initially treated for any Lyme infection. Suffering from a sore throat and intense headaches, the blonde former mayor of West Palm Beach who now divides her time between Miami and New York was growing progressively weaker. Pepper consulted more than 30 leading neurologists and internists. All the doctors told her she had incurable, fatal amyotrophic lateral sclerosis (Lou Gehrig's disease) and had only two to five years to live; after all, she'd had seven negative Lyme tests.

Pepper eventually took more expensive, more exacting tests that looked for the presence of spirochete DNA or the outer membranes shed by spirochetes. These tests immediately revealed rampant borrelia infection. (Animal inoculation, which is injecting an animal with fluid from the Lyme victim, then analyzing its tissues in an autopsy or biopsy, is another method.) Pat Pepper was finally wheeled into Dr. Burrascano's office, nearly quadriplegic.

Burrascano warns patients that their symptoms may get much worse during his treatment before they get better because spirochetes put up a nasty fight. Spirochetes in the dormant phase of their reproductive cycle (ranging from ten months to fourteen years) survive the antibiotics, which is why it takes many courses to obliterate them all, says Burrascano. Though his treatment usually takes from one to six months, it was a year before the spirochetes completely disappeared from Pepper's blood tests.

"Since July of '98, when I first saw Dr. Burrascano, my health, for the first time in seven years, has been improving," says Pepper, who recently graduated from her wheelchair to a walker.

Pat's own story can be heard in an audio interview here:

<http://www.unfictional.com/mp3/pat-pepper.mp3>

59 <http://www.unfictional.com/mp3/pat-pepper.mp3>

60 <http://nymag.com/nymetro/health/columns/bodypolitic/2225/>

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Lyme-positive people who died of ALS

Jon William Davis

Jon wrote the following story in March 2009 of how he was initially diagnosed with ALS, but how instead he turned out to have Lyme plus several coinfections. The story shows how his ALS slowly stabilized and even partially improved with antibiotics, and remained stable for years. (abridged):

“Dad? Dad, what’s wrong? Say something!”

I couldn’t make a sound. A quadriplegic, I couldn’t even move. My son Nathan and I had just done a maneuver we had done dozens of times before to clear my lungs, but this time it didn’t work. Mucus plugged my windpipe and it wouldn’t budge. I was suffocating.

At the same time, my hospice nurse, Kimberly, was arriving to draw a blood sample for monitoring my blood thinner. Nathan and the nurse tried to do the maneuver again to open my windpipe, with no luck. They could see my face changing color before their eyes, turning pale and then gray.

They called for Nancy, my wife, who immediately grabbed the phone and dialed 9-1-1. I could feel my lungs starving for air and my vision fading to black. I could hear everyone calling out my name in panic as they did chest compressions to try to get the air to move. I had become completely non-responsive, with an erratic, faltering heartbeat.

Fortunately, the emergency squad made it to my house within minutes. When the emergency medical team got into my room, Nathan picked me up under my arms to put me on the gurney, which dislodged the plug and allowed me to breathe. I was rushed to the emergency room, where the doctor recognized me from several earlier visits to the E.R.

He stated to Nancy that I had a progressive disease and would not likely survive, and asked if she wanted him to give me a dose of morphine to make me “more comfortable”. Nancy knew that I was in respiratory failure and that a shot of morphine would further suppress my breathing.

“Comfortable” would mean dying. Nancy got angry and told the emergency room doctor that if she wanted to put her husband to sleep she would have taken me to a veterinarian.

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She told him to either put in an airway, or to give her a butter knife and a straw and she would do it herself.

Just then my respiratory doctor arrived. He ordered the ER doctor to put in an airway. Then he dragged Nancy to an adjoining room. I was in the ICU for the next two weeks.

When diagnosed with amyotrophic lateral sclerosis (ALS, or Lou Gehrig's Disease) back in April 2002, the neurologist told me I could expect to die and had given me three to five years to live.

Three months after this news I was diagnosed with Lyme Disease and five co-infections. I believe the Lyme Disease caused the ALS.

At the time of this ER visit I had developed double pneumonia and had almost died right on schedule with his prediction. What he didn't know about me was my drive and determination not to lay down and die.

I knew it was likely that I had got Lyme disease from a tick bite while deer hunting in December 2000 in southeast Ohio.

By 2003 my hunting season was very different. I needed help from Nathan to get into my cammo and boots. Getting on the four-wheeler was almost impossible, too. My friend Dale Smith went to the south edge of the property we were hunting on, and Nathan went to a stand to the north.

I was sitting on the footrest of my Polaris looking down into a ravine. I had sat there for about 45 minutes when I heard rustling in the leaves and saw a doe come into the ravine. I had my 20 gauge propped up on the front rack looking at the deer less than 40 yards away. Excited to have a slam-dunk shot opportunity... ..I was too weak to bring my gun up because of nerve damage from the Lyme Disease. I was put on intensive antibiotic therapy in July of 2002 and have been taking it ever since, with no end in sight.

My health was declining and spiraling out of control. My six-foot frame that once weighed 175 pounds, withered to 110 pounds in January of 2006. That is when I could no longer swallow solid food. I had a feeding tube installed just two months before my near death experience.

My respiratory doctor and surgeon, who had been reluctant to do a tracheotomy, were amazed that I was rebounding and gaining strength. After ten days the doctors went forward and put the trache in.

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A year later my weight climbed up to 215 pounds. The only regret I have is not getting the feeding tube and trachea sooner. They are tools that could have helped me rebound sooner.

With his guidance and products I have regained even more strength. Also my speech has improved significantly even while on a vent. Although I am still too weak to move my arms or legs I can wiggle some of my fingers and toes.

This doesn't sound like much of a life but I am determined to fight through this nightmare. Other victims of this disease that I have spoken with through the years have passed away from pneumonia, diaphragm failure, or they simply lost their will to live.

In my opinion, Lyme Disease is an epidemic affecting up to five hundred thousand people each year in the United States.

The main problems with LD are improper diagnosis and insufficient treatment, allowing a resurgence of the disease. Lyme can be misdiagnosed as over one hundred different conditions.

It is recommended that symptoms and environmental risks be used for diagnosis, with blood tests for confirmation, since blood work is unreliable on its own.

One reason for this unreliability is that the blood tests are not sensitive to local strains of the *Borrelia Burgdorferi* bacterium. U.S. blood tests use Lab Strain B-31, which is not even found in nature.

Carriers of these infections can be any blood-feeding insect, including ticks, mosquitoes, fleas, and biting flies.

In my opinion there are many important treatment options for Lyme Disease, such as pharmaceutical antibiotics, herbal supplements and alternative treatments like oxygen therapy.

Doctors in several states have suffered legal sanctions for prescribing therapy for Lyme Disease patients who had been misdiagnosed by conventional medical professionals.

An investigation by the office of the Connecticut Attorney General brought to light several problems with the writing of the Infectious Diseases Society of America's (IDSA) 2006 Lyme Disease guidelines.

These problems included committee chairmen and members with financial

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and professional conflicts of interest in the treatment of Lyme Disease. The IDSA findings were compromised by committee members and the chairmen who also served on Lyme disease committees for another medical association.

These same members helped the second association write guidelines that were used to reinforce the guidelines of the IDSA committee.

Lyme Disease patients also face a challenge from the Center for Disease Control. That group is tightening, rather than broadening, the criteria for a diagnosis of LD. This means that potential cases of Lyme Disease are not reported to county health departments.

It is now March of 2009. Seven years into this disease, my condition has stabilized and slightly improved since that trip to the ICU in September of 2007.

On November 16, 2010, Jon wrote that antibiotics were reversing some of the ALS symptoms (abridged):

I was diagnosed with ALS in April 2002. I was also diagnosed with Lyme Disease in July of 2002. I've been taking antibiotics ever since. I went from 175 lbs. to 110 lbs. in January 2006. I couldn't eat or drink at that time. I received a feeding tube on my 46th. birthday.

In March 2006 I nearly suffocated from mucous in my lungs. I spent two weeks in ICU and received a trach and was put on a vent after 10 days and regaining some strength. On Sept. 2007 I went to the ICU with pneumonia and two blood clots in my lungs. The good news was, I weighed 215 lbs. when I was admitted.

Back in 2005 we were told by a hospice caseworker that the feeding tube and vent would be pointless as I had a terminal disease. I'm glad we didn't listen to her. I can communicate (speak) even though I'm on the vent, which surprised my lung doctors. I'm using a Pulmonetics turbine vent and a Bovina trach. Through the years I have been using herbal supplements and holistic tinctures along with multiple combinations of antibiotics, including IV antibiotics.

At the end of 2005 I couldn't move a muscle. I was basically a quadriplegic.

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But I am now able to move some fingers and toes, and can feel other muscles beginning to respond throughout my body. I am also able to eat solid foods, including steak, and drink water and swallow pills again, for over a year now. Several years ago I was on the health boards regularly, but my advice was not received well on this ALS topic board. Just trying to reach out to others to seek a Lyme-literate medical doctor (LLMD) to help you fight back from this diagnosis. Check the Lyme Disease panel to find a LLMD near you. I have been to two in Pennsylvania, and one in Wichita, Kansas. There are many others around the country. Many of us have not survived but I am improving, thank God.

Jon William Davis was 51 years old when he died on March 4th, 2011 at his residence in Pleasant Hill, Ohio.

Charles McPhee

Charles is an example how antibiotics failed in his case of Lyme-positive ALS. We were unable to ascertain whether Charles received 4 gram/day of Ceftriaxone instead of 2, which would likely lead to treatment failure. However we could establish that Charles had bulbar ALS and difficulty swallowing before he was treated with antibiotics. Charles initially experimented with Curcumin and other substances that will not help Lyme disease much. Charles serves as a sad example of either "too far progressed neuroborreliosis for successful treatment" or "purely coincidental that he had both an ALS-diagnosis and a positive Lyme test". Charles used to interpret people's dreams at dreamdoctor.com.

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*Tony Hofer*⁶¹

Tony wrote this on December 14, 2006 (abridged - spelling corrected - emphasis ours):

Early on, after diagnosis of ALS my speech started to slur, to the point where I found myself having to repeat myself. **We'll believe it or not with the help of just oral antibiotics my speech cleared to 100% normal.** After a while say a few months, the slurring returned. **We then switched to a different but similar antibiotic and speech again cleared. We had my speech clear 3 times.**

Now we have mentioned this to the ALS experts at Northwestern university here in Chicago and **they were astonished.** but, because none of this was officially documented, its as if it never happened.

It is my contention that I indeed have Lyme disease that at the time could have been cured had the folks at RUSH PRESBYTERIAN hospital not refused to test for it!

Tony Hofer of Chicago Illinois died at age 43 on June 7, 2007.

⁶¹ <https://www.inspire.com/janet4/journal/lyme-disease/>

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Brian Pierson

Canadian Brian Pierson wrote his story on December 31, 1998. He describes how he finally went from an ALS diagnosis to a Lyme diagnosis, and how some symptoms improves on antibiotics:

My name is Brian Pierson. I am 46 years old and married with three children - wife Karen , Crystal 20 yrs., Aaron 16 yrs., and Christine 14 yrs. I live in Lethbridge, Alberta Canada. I have been a motorcycle dealer for 30 years and have raced off road motorcycles since I was 15 years old; most of the time, at an elite level. Having won numerous Canadian, Provincial, Montana Championships for motocross, Hill climbing, dirt track, trials, Hare scrambles and X-country motorcycle racing, I have been in good physical condition with some very strange ups and downs over the last 7 years.

I've always had a fairly good diet, drank alcohol mostly hard liquor (rum) on weekends and sampled a few cigarettes throughout my life but was never an everyday smoker. I have had knee injuries, broken right leg, severe burns twice, two concussions and a dislocated right shoulder. I have always had a good heart, blood pressure, muscle tone, lungs, and no allergies.

Now that I've had 7 months to reflect, soul search, watch my condition, and argue with doctors, I do believe my story started in 1987. In 1985/86 I was in the best physical condition I was ever in. I started building a new house out on the prairie, and I lost 35 lbs. of body weight for no good reason. I was working hard (long hours), but I also ate well; however, my physical condition started deteriorating. For years I had a mysterious itch affecting my legs and arms that would drive me nuts for a week or so a couple times a year, and then go away. It would come and go for as long as I can remember.

I'd have night sweats, sometimes so wet that I would need a life preserver, which would come and go. I suffered from a very stiff neck, so stiff I couldn't turn my head (mostly left side and it would come and go). I had a lot of migratory joint pain of knees and wrists. In July of 1991 my right knee became so painful that it put me into emergency screaming in pain (from walking to collapsing in one day). The first ER doc to look at me said take two tylenol. I came unglued, as this was the worst pain I have ever experienced.

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I screamed, yelled and tore the curtains off the wall. Finally an orthopedic surgeon came in and looked at me; twenty minutes later I was in the OR being operated on. They flushed my knee with something and kept me in the hospital, bedridden, for two weeks. I was treated with Ancef, 1 gram IV and Gentamycin, 80 mg. IV every 8 hours for 7 days; then Penicillin Pefk 500 mg., 2 tablets every 6 hours, for a total of 336 tablets.

I took it easy through the winter, and started to ride and train in the spring. I felt excellent, strong, and came back to win two more Canadian Championships for off road motorcycle racing. Actually, I never lost a race I entered in 1992. Throughout 93, 94, and 95, my condition seemed OK except for the migratory joint pains that would alternate between knees and wrists. Thinking it was do to aging and injuries. I never sought medical attention and just lived through it. February 1996, I dislocated my right shoulder in a racing accident and took a severe body pounding.

Now to the hospital, where they had to put me out, and four try's later, got my shoulder back in place. They used some kind of new drug that puts you out, but when you awake you can walk out of the hospital right away. I drank alcohol that night and sat in a lot of diesel fumes while they were dismantling the track (indoor race). I don't know if this had anything to do with my condition now but thought I would mention it.

Within a week I could lift my arm over my head and actually felt quite good. Then, two weeks later I woke up in extreme pain (right shoulder), which went away in a few days. About a month after the dislocation, my right arm muscles started twitching (fasciculation's) and slowly spread to my left arm; right shoulder never did come back to strength. My left leg would start shaking on occasion. Overall, body stiffness got worse, and with joint pains. No matter what I did I was always stiff, sometimes barely able to get off the floor. Sometime during this period, my ears started ringing (especially the left one. This symptom also comes and goes.

I was also under a lot of stress business wise through this period. I had two complete physicals and both times was pronounced fit. The doctor said the twitching must be a pinched nerve. Overall physical condition and balance got

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worse. I developed a very itchy and sore anus which I sought medical attention for. The doctor said I had a very rough looking rash and gave me some ointment to put on it. This rash and soreness would come and go for the last few years. I got weaker and couldn't stand for any length of time. I didn't train because I just felt run down and tired most of the time. My X-family doctor once thought I might have CFS (chronic fatigue syndrome), but wouldn't come right out and make a diagnosis.

The summer of 1997, my balance was off more and I was still getting weaker. I started to drink more and smoke more. The only time I found some relief was when I was partially drunk. Hangovers were getting worse and always seemed to come on the day after. At around 5:00 pm. on October '97, I crashed at high speed and took another severe body slam with the ground. By December of '97, writing was becoming difficult and the ringing in my ears was getting louder, along with my balance going downhill.

In May of 1998, I had a complete physical with a new doctor. I showed him the twitching in my arms and that my right hand index finger movement was slowing down. He checked my vitals and told me to get more exercise, and didn't seem to think much about the muscle twitches or the slowing down of the finger. May long weekend, I went from riding, to not being able to put my fingers into a glove, in one day. The weather turned cold and wet, which seized up my right fingers to the point of uselessness.

My index finger started curling in when I rode my bike and brain to fingers reflexes became so slow that I could no longer ride my motorcycle properly. One other strange thing was if I pointed my index finger out and tried to move my arm, I couldn't, but if I made a fist my arm would move normally. I also started to walk spastically. My legs felt like springboards but I couldn't move quickly or run. Coffee started seizing me up and making my fingers twitch. A small amount of alcohol had the same affect to the point where one night I had two beers with the guys and could barely move or open a door with keys.

I mentioned to my wife that I thought I'd found out what was wrong with me - allergic to alcohol! Something was definitely wrong so back to my X-family doctor, who said again, maybe CFS, and then referred me to an internal

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doctor; nice fellow that thumped on me with a rubber hammer for awhile and I just about kicked him over. My reflexes were very brisk and my legs would shake when he cranked up on my foot. He said he thought he knew what was wrong but would have to look in his books; then he referred me to a neurologist.

I went to see the neurologist on June 11, 1998. This guy was a total jerk. The examine was about 10 minutes long, where he too, thumped on me and flashed a red light on my skin in the dark. He then told me I had ALS! We sat down, and he did a family tree. I asked what ALS was (I'd only heard the name before and knew that a baseball player died from it). He told me two years to live - no cure, no drugs would help, no hope, see ya!!!! The whole meeting with this guy was about 30 minutes long. I was shocked! What was ALS? Needing more information I bought a computer, and internet time, then proceeded to search for information. What I found was not very encouraging. My sister Cheryl was also searching for me (thank god for her), and found someone that had ALS and was experimenting with supplements, with what looked like some success. I quit drinking and smoking totally and started taking supplements. Within a couple of weeks I felt a lot better; energy came back, I could stand and keep going all day long, but my hands did not work any better.

I started exercising regularly, because I felt good knowing I was getting stronger. Weights were getting easier and I started lifting them over my head with my right arm which I hadn't done in a long time. This didn't make sense with ALS, so I sought out a second opinion. I got myself into an ALS clinic in a large city, where they are doing experimentation with new drugs. Thinking these doctors should know for sure, I was examined in the middle of July. The neurologist said I showed signs of both upper and lower motor neuron disease, but needed an MRI and an EMG, because of a questionable neck injury. The MRI showed that my brain and cervical cord appeared normal; slight posterior disc bulges at c4-5 and c5-6. EMG showed some nerve degeneration in the arms, mouth, tongue, throat - all normal.

I was also under high stress at this time, because my son had had a severe racing accident. In the middle of July I still felt fairly good and was continuing to exercise when I started to get what I will call lightning strikes. These feel

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like someone is sticking you with an open end electric current. Had one so intense I thought one of my toes blew off! I was also developing another very strange symptom. When I got excited my body would seize up, my legs wouldn't work and I could barely move. As soon as I calmed down, a period of a minute or so, away I would go again.

Sometimes my legs would shake too. For years I would get very wet - nightsweats on the odd occasion, but they seemed to be getting worse and more frequent now. I had all the regular blood work done; aluminum test, mercurytest, IgG and all the subclasses, but nothing showed much from normal. I had a Lyme disease test done because I read somewhere on the net that I should. It turned out to be an Elisa test; *Borrelia burgdorferi*-negative. The only thing I tested positive here for was Epstein Barr VCAIgG. The doctors didn't seem to think this was a problem.

In August, I saw the neurologist at the ALS clinic again for review. He went over my MRI and EMG and then said I had probable limb onset ALS. I asked him about some of the weird symptoms I was having and he said he didn't know what it meant. He gave me three bottles of riluzole and sent me home. I took the riluzole for four days and then stopped; thought it made me weaker. A heat wave hit in my area with temperatures running from 80 to 95 degrees. This really changed things for me. If I stayed out in the heat for any length of time I would get next to very violent. I would get so tired by mid - afternoon that I had to go sleep; I felt tired and run down all the time.

I quit exercising because I had lost all my energy and drive. I started getting forgetful. I would forget where I put things, or walk into a room and wonder what I was doing there. My thoughts would collect in a few minutes. I also was getting worse brain fog; hard to concentrate, and it was like I was spaced out(half drunk). I had this problem for a few years but never thought much about it. For me, it was like driving down the road, with everything in slow motion and hard to concentrate.

Since the ALS doctor couldn't answer my questions, and after many hours research and communicating with different ALS patients and support groups, I needed more answers. Back to the internet - CFS close on symptoms, but it didn't answer for my neurological problems. Then I read an article in the ALS

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Digest (#518 Borrelia infection masquerading as ALS). This got me thinking; I had two Elisa tests done previously (both negative) but I knew nothing of the symptoms of Lyme disease.

I contacted the person that wrote this article, and she put me in touch with a person who had had Lyme disease since 1986 - a two hours drive from me. He was a real nice fellow whom I had a long chat with. He then sent me a stack of information and a Lyme symptom check list. THERE I WAS. I COULD GO DOWN THE LIST AND CHECK OFF JUST ABOUT EVERYTHING; ESPECIALLY IN THE NEUROLOGICAL LIST. Finally some answers for my symptoms!

Here I was living on the prairie. I have three horses that I constantly pick ticks off of with my bare fingers, and squash. I have a history of being bitten in my youth, and my mom took them off with a hot needle most of the time. I remember a more recent possible bite on my chest just below the hairline. I am an avid hunter and have been in known Lyme disease areas. It started to make sense to me so I searched for all the Lyme disease information I could find. The more I found the more convinced I became. ALS pals thought I had very strange symptoms but people with Lyme (some that have had positive tests) were so similar to me, it was scary; right down to the EMG tests and the way my hands and arms are.

Loaded with this new information it was off to my X-family doctor for help. To make a long story short, he said too experimental, too expensive, might lose my license, negative test results, hope the other guy helps; that's why he is my X-family doctor. Devastated, I took my documents in search of a doctor that would help me. I got lucky and found a great GP doctor in a small town close to me, that listened and wanted to help. He didn't know much about Lyme disease and he had just lost someone he knew to ALS Bulbar, so we started treating the symptoms instead of waiting for a positive Lyme test to come back. Here in Canada our test procedures are barbaric, and for the most part, doctors are very reluctant to say there is Lyme disease here.

I can send to the United States for tests but would have to pay for everything myself. The neurologists handed out a diagnosis of ALS way too easy, without checking all the other possibilities. On September 1, 1998, I started treatment with 2 grams of IV Ceftriaxone (Rocephin) per day, scheduled to finish in

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January 1999. I have constantly done supplements and messed around with different tones and doses. I have had regular blood work done monitoring my liver function and so far all normal. **Improvements so far are: stiff neck is gone (took two months), right index finger doesn't lock up my arm anymore, had a second EMG done three months later and it showed nothing getting worse, short term memory loss seems better, and my brain fog has finally lifted enough so that I could sit down and write this; and I am still moving.**

What's not better: **I have gotten noticeably weaker shortly after starting IV therapy, experienced fairly regular HERXHEIMER REACTIONS,**(but the intensity seems to be getting less as the months go by) feel sick, weak, tired and out of balance, but I do get the odd hours or days of feeling a lot better, where I actually think I'm on the road to recovery; however, overall body stiffness and joint pain still moves around. My right knee hurts like its been injured again. Here it is December 31, 1998, and I can't wait for 1999 to begin. 1998 was a very rough year for my family and '99 can only get better. Here's wishing you all a HAPPY NEW YEAR and better health for everyone in 1999. Keep the faith.

Brian Pierson
Lethbridge Alberta, Canada

Brian died in May 2000.

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*Luther Conant*⁶²

June 13, 2000. Luther receives his ALS diagnosis.

By now things were growing increasingly difficult at work. When my coworkers and clients saw me limping up the hall, I'd catch that look of pity in their eyes which as any cripple will tell you, you soon come to truly loathe. It didn't bode well for my continuing employment, either. People don't like to pity the people who work for them. It makes them feel guilty when they ask you to do unnecessary bullshit.

I was deeply depressed at having to wait so long to find out anything. In addition, I'd about had it with Dr.Y's office staff, who had the same attitude of, "Listen bud, I'm doing you a favor just by talking to you," that I'd come to expect from late night Seven Eleven clerks. That's when my brilliant wife told me that I ought to see the neurologist who she'd been seeing, a doctor she liked and respected. So, I called up and got an appointment with Dr. R.

He turned out to be the antithesis of Dr.Y, a quiet gentle man who seemed to genuinely care about how I was doing, as a person, and not just as a medical mystery to be logically explored. The fact that he had books on Zen and the Art of Healing, and Native American Medicine on his shelves further bolstered my opinion of him. He put me through most of Dr. Y's tests, but with sensitivity to the possibility of causing pain, which I very much appreciated. Then he did something new. He had me hike up my gown and do a muscle watch. I was fairly horrified to see what he called faciculations, random groups of muscles twitching all throughout both legs and calves. It was like watching the movements of animal herds from an airplane, muscle groups shifting and moving around as though trying to avoid unseen predators. I hadn't been aware of them before, but, like the edit in the middle of the Beatles' "I am the Walrus," once you hear it you'll always hear it. Now, I could feel my legs twitching all the time. Dr. R also looked at my hands and I was even more disturbed to realize they were twitching, too. I'd had hopes that, if I couldn't continue as a video editor, I could continue working on my third novel. I was suddenly terrified that might become impossible.

When I mentioned MS to Dr. R, he excused himself from the examination

⁶² <http://lutheroutloud.com>

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room, and returned a few minutes later with several large medical texts. In these he proceeded to show me the stages of progression for MS and how they weren't anything like what I was going through. I had sort of come to accept the idea of MS, it's nasty, but you do get to live and communicate for a fairly long time, and I was suddenly cast back into limbo again. I've always thought that the idea of limbo, a place where unbaptised babies go because they haven't the requirements to make it through the pearly gates, but they haven't had a chance to screw up bad enough to warrant burning in eternal pools of fire, was on rather shaky theological ground, more appropriate to secret societies like the Illuminati, the KKK, or the Moral Majority than a loving God. Consequently, I found my stay there spiritually and emotionally debilitating. A royal pain in the ass. Believe me, not knowing is almost always worse than knowing.

My legs continued to get weaker until finally, in February, I took a spectacular fall at work, in front of a crowd of both employees and clients. After that I found everyone to be so overly solicitous, and worried about my every move, that I could no longer function as a trusted professional. In addition, the Baclofen was making me so sleepy that it took a strong cup of coffee and 10 to 12 iced teas just to stay awake during the day. And I knew that I wasn't giving my clients the quality of work that I'd come to expect from myself. So, in the last week of February, I told them I had to take medical leave. I think they were relieved, but were kind enough to try and not show it.

Luckily they had a good long term disability plan, which everyone assured me would kick in in 30 days, with no hassle on my part. The insurance management group would take care of everything. Naturally, the insurance management group was about as adept at its' job as you might expect most mid level corporate employees to be: it's now June and I have yet to see a penny. I ended up doing their job for them, making all the calls and contacts myself. I have some hope of eventually seeing some money.

Then Dr. R ordered a few more tests, which I don't remember as particularly nasty, checked me again, and dropped the bombshell. He wanted me to go see a Dr. M at the Pennsylvania Hospital ALS clinic. ALS, which I had read about when I had researched neurological disorders, is also known as Lou Gehring's disease. It's a rather nasty affliction where the only drug available may prolong your life for up to 3 months! Whoop-de-do! Most people live an average of 2 to 3 years after diagnosis.

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Luckily, the ALS clinic is composed of humane people and they have "Rule out sessions" every Friday. No waiting for months here. So the next Friday, I went in, confident of being ruled out.

Dr. M turned out to be my idea of the perfect physician, kind, gentle and thorough, a true healer. This made it a lot harder when he said, "I'm afraid that it looks like ALS to me." There was no chance of thinking "What does this asshole know," as he'd studied ALS for 23 years, and he decidedly was not an asshole.

My wife and I left the hospital shaken and in shock.

June 13, 2000. Luther tests negative for Lyme disease.

While all of this was going on, there had been another development that I hadn't paid much attention to. The mother of one of my students in First Day School, (See GOD STUFF-Quakers), had given me some papers on Lyme disease. I had been tested for Lyme early on, so I hadn't paid them a whole lot of attention, but with ALS suddenly looming on the horizon like Spiney Norman hunting the Piranha Brothers, (See Monty Python. It's not here, but see it!) I decided to check out Lyme. Imagine my excitement to find out that not only are Lyme tests notoriously imprecise, but the damn disease has been known to mimic both MS and ALS! (See LINKS for more Lyme info.) Naturally, out of the 300 strands of Lyme found worldwide, (200 in the US) the one that mimics ALS is found mostly in Europe, but it had shown up in Land of the Free and the home of the growing-less-than-free-by-the-minute, and as friends pointed out, I've never done anything the normal way in my life.

So I printed out all of the stuff which I found and dropped it off with Dr. R, and asked him to look it over. He did, but again told me that I had been tested for Lyme and the results were negative. I let this crush my hopes like Charlie Manson's parole board, for about a week, until it was time to go back to the ALS clinic.

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June 28, 2000. Luther tests positive for Lyme

Dear Friends,

I went to the Doctor who specializes in Lyme disease today. She told me that a third test had come back positive, so they will begin treating me for Lyme disease. This involves having a tube or shunt inserted into my body so that I can receive antibiotics intravenously three times a day. She said it would take about a week to get the insurance company to okay it. In the meantime, I'll be taking large doses of antibiotic orally.

When I asked her what she thought the progression of the disease had been, she told me she thought that the ALS had been triggered by the Lyme spirochetes, and that the IV therapy would stop it's progression. She held out a faint hope that I might regain some of the muscle control I have lost. Needless to say, halting the disease where it is, will be just fine with me!

The harder news is, that I'll be in pretty bad shape, fevers, aches, etc. as the treatment begins. In fact the first oral dose I took today put me to sleep for 4 hours. I asked how long this could last, and she told me her last patient had to wait four months to see any improvement. So, although the news is great, I'm going to be pretty much out of commission for a while. And ultimately, we won't know if the doctor is correct or not, until we see some improvements, or lack thereof.

So, that's where things stand. Thank you all for your concern and prayers, and know that I'm grateful beyond words for all the kindness we have received,

With love,

Luther

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Luther reports a Jarisch-Herxheimer reaction - a sign that he has serious Lyme neuroborreliosis.

Dear Friends,

So far the IV antibiotics are kicking my ass, I got mugged by a kitten yesterday, little cocksucker took my wallet and keys, but couldn't reach the pedals and so hopefully is starving to death in the van. This is the first time out of bed in 2 days. But, I'm hoping for the future.

Dear Friends,

Today was Gwen's High School graduation and everything went beautifully except that I had to sleep a lot. These IV antibiotics are a lot like having Joe Frazier give you a full body massage. Everything hurts. But, since that's a good sign, that the detestable little parasites are kicking the bucket and dumping their toxic stools in my bloodstream, I can put up with it. For every ache or spasm I imagine an entire herd of spirochetes dropping into one of Dante's flaming pits of the seventh level and the thought pleases me immensely. I've been reading a lot of Twain if you haven't noticed.

April 16, 2001. Luther has deteriorated badly

In the last chapter, I said I was going to try to get through the grieving process as quickly and efficiently as possible. However, I've found the process itself is not amenable to that approach. It's something that needs to be worked on in small portions every day.

I learned this through reading "Morrie in his Own Words". Morrie said that he did a little grieving in the morning each day, in order to get it over with. I'm just dealing with it whenever it arises. So far, it hasn't been too overpowering a process. Just something you do, and then move on. There's been so much to mourn, lately. Which leads, I guess, to a description of where things stand right now.

I've lost control of both hands. Now, my voice is giving out on me. My loudest

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voice is what I used to call a "stage whisper". This makes even talking on the telephone difficult. In addition, I've been bedeviled by a number of flu-like physical ailments. I've spent a couple of days vomiting, which, when you can't lift your head, is a particularly unappealing process. It's also hard on my poor wife, who can deal with anything except puke. Luckily, during both attacks, my brother was visiting from Massachusetts and was able to take over some of the basin-holding duties. Chills and hot flashes accompany the vomiting, as well as a strange sensation I find easier to describe than to categorize. It feels as though a pressure wave pushes up my body from my legs through my head, making each area feel as swollen as my hands, almost to the bursting point.

This pressureWave is usually accompanied by chills and is particularly nasty. It started off visiting once every few days, but has lately been stopping in every day and on a few days, twice. My hope is that all of these - pressureWave, nausea, chills and hot flashes - are symptoms of some secondary infection, and that, if we can ever find it, the doctors can cure the infection and they will go away. They just don't feel like part of ALS, somehow. The doctors have agreed to have a look

So last Friday, I went into the hospital for various tests, including x-rays of my lungs, a CAT scan of my sinuses, and an array of blood and urine tests, of which the last-named proved the most difficult. The hospital was packed to the walls. Not a bed to be had. Accident victims and cardiacs were stacked against the walls - carefully, mind you. They pushed my gurney into an office full of busy schedulers, slipped a urinal beneath my schwanz-Stucker and told me, "You go right ahead. No one will notice." Needless to say, I found this impossible. So they pushed me into a quiet hallway. The moment I was alone, however, huge automatic doors whooshed open behind me. I discovered I was in an entrance to the ER, surrounded by people walking in and out, pushing emergency patients. I never did succeed in filling the cup. Of course, the ambulance ride home instead of struggling to get it out saw me struggling to keep it in. It's a world of contrasts, what can I tell ya.

I eventually sent them some urine via carrier pigeon.

So far, I've heard nothing about any results. And so, I remain hopeful.

Everything had actually come to a head the Wednesday before. My memories of that day are very fragmented, and I've had to rely on what a lot of people have told me to put it together. I remember that I woke up choking on a huge mouthful of phlegm. Once that had cleared, things got very strange. Lynne

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tells me that I wasn't very coherent. At some point, I asked to have a table removed from my chest. I am aware of the fact that I seemed to be in the very first apartment she and I had rented together 25 years before. Sometimes, I could even open my eyes and see it. But on reflection I would realize I couldn't possibly be there.

It was all very confusing. I would keep orienting myself as to where I was, only to find that I had slid back to that earlier apartment, later. At the same time, I had the distinct impression of being able to see the life force within my body. It was like a glowing blue gas, and my body seemed to be made of a thin crystalline structure containing it. The gas seemed to be escaping somewhere, growing thinner as I watched, and I could feel my hold on this reality slipping away. I was convinced that I was dying, and announced this to everyone in the room.

It felt like something I had no control over, and that at any moment I would just drift away. My brother wanted to know if I wanted them to call 911. I refused, wanting to die in my own bed. I also refused any pain medication. I wanted to keep my thinking as clear as possible, you see. Naturally, I was scaring the piss out of everyone in the house, and even some people who called in the middle, whom I blithely told, "I'm dying. Goodbye."

When I told my daughter, she just said, "NO!" and left the room. I hadn't expected that. When I told my son, he explained in an adamant voice, that talking about dying was pure bullshit and that I could stay or go but it was my choice even if I didn't understand how to work it right then. I remember laughing with Lynne after they had left the room, saying that of all the reactions I had expected, not even being believed was one I had never considered. The fact that I could laugh even when I believed I was dying gave me great pleasure at the time, and even more now, thinking back on it.

Another thing, which was very disconcerting, was that I kept hearing voices and sounds that were inaudible to everyone else in the room. I wish I could remember more about these voices now, but I can't. When I was talking to Brian, I had the distinct impression that he was holding my hand. I thanked him for it. He told me that no one was holding my hand, but I continued to feel the gentle soothing pressure for at least another hour. Whether this was contact with spirit guides or someone departed or just imagination, I have no idea.

It took until late afternoon for me to realize that I was over some hump, and

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wouldn't be dying that day. I'm still convinced that I could indeed have let go and slipped away, except for my children's insistence that I not. Anyway, I'm glad it didn't happen. But I feel tremendously sorry for the hell I put my family and friends through. It was a very odd day. Trying to remember it is like trying to grab smoke with my fingertips. The scariest part is, I have no idea what precipitated this event or how to deal with it if it should come again.

Since that day, I've felt more disconnected from my life than ever before. I find it hard to indulge in trivial chitchat anymore. During particularly painful days I've taken to disconnecting from my body and floating in some soft dark fantasy world, the details of which elude me when I'm not there.

All in all, I guess it was good to have a dry run for the day when death really arrives. We found several holes in our support system, including the fact that no nurse or doctor would come to the house to check on me. Thanks again to those wonderful folks who create insurance regulations. I also discovered that I have a lot more to talk with my son and daughter about, to get them ready, or as ready as they can ever be, for the ultimate ending.

Quite frankly, I never thought death gave you a dry run. But as in everything that has happened to me lately, I stand back in awe, amazement and appreciation.

I'm wondering if the feelings of disconnection that I'm currently experiencing are really a holdover from that day, or are symptoms of whatever infection feels like it's raging through my body. It's hard to be present when every muscle is screaming for your attention.

The other day I was struck by a hurt dating from around 7th grade. And I didn't want to let it go. Still. But eventually I talked myself into letting it go, and I did, and it really felt better. I was struck with how much energy I'd spent over the years holding on to this trivial piece of shit. If nothing else, I will continue going over my past and discovering more old hurts I can discharge through forgiveness. I don't know that that will keep me around any longer, but it'll certainly take what time I have left and make it a lot more pleasant.

Love,
Luther

Luther Chittenden Conant III died on June 7, 2001.

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Dean Chioles⁶³

I was diagnosed with sporadic ALS sometime in October of 1993. As I recall my first sign of the symptoms was weakness in my hands that occurred sometime in 1989, at the age of 23.

I was a guitar player since the age of nine and was in several bands including, "Abomination" (speed metal) which released two full length CDs and toured Europe. The loss of strength was affecting my playing so I became very concerned. I saw a chiropractor to see if he could determine the cause of the problem. A grip strength test was performed and the average for a man 23 and 5'6 was 110-120 lbs. I was only able to squeeze approximately 60 and 65 lbs. After X-rays of my spine and my neck, his diagnosis was that the nerves going down my arms were pinched, causing the weakness. He warned me that if this was not treated that the muscles in my arms would eventually begin to atrophy. He used ultrasound as well as spinal manipulation for treatment. After five months 90% of my strength had returned and I felt great and I discontinued the treatments.

In the Spring of 1993 I was working as a sound engineer when the drummer from "Abomination" and I joined "Body Bag" (no love songs here). The feebleness in my hands returned so I found another chiropractor. He thought the problem was similar to the previous diagnosis and began treatments by manipulating my spine and neck. In the meantime my guitar playing ability was diminishing. I would squeeze a racquetball for 30 minutes while riding to band practice, but it still did not seem to help my playing. My fingers were sluggish, weak, playing rhythms was more difficult and my solos were becoming less melodic.

In late August 1993 I was leaving a White Sox game with some friends and while walking down a ramp my legs were very stiff. I was walking slowly and was having a difficult time when my friend asked me, "Why are you walking so funny?", "I don't know, maybe it's because I'm not stretching out before I exercise on my cross country skier", I replied. The next few months were very confusing. Running became difficult, I wanted to sprint but I was limited to some weird demented jog. After riding my mountain bike I could not get my leg over the bar to get off, so I would just fall. The next few weeks I was real jumpy. I would jump at loud noises, even if I knew they were coming. I was asking myself, "What the hell is wrong with me?"

⁶³ <http://www.shoptown.com/Dean/ALS2Lyme.htm>

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My hands were becoming weaker and more symptoms appeared throughout my body. I was unstable walking in confined spaces and started losing my balance. I fell a couple of times from either tripping over something small and not being able to recover, or just falling to my knees from dragging my left foot. I was trying to figure this one out. Was it the Nordic Track? Around the same time when I would lie in bed ready to go to sleep I could feel my muscles all over my body twitching a little bit. I thought it was weird, but it really did not bother me at first because it did not happen all the time.

I told my chiropractor what has been happening and he referred me to a chiropractic neurologist. I went to see the Doctor and after a physical, x-rays, EMG and two M.R.I.s his diagnosis was ALS. "What's that?" I asked, "Amyotrophic Lateral Sclerosis," he replied, explained what it was and that there was no cure. Right then I knew I would have to find a way to beat this thing. He recommended I go see a neurologist at Loyola Medical Hospital. That evening I looked up ALS in our Mayo Clinic encyclopedia. It explains a little bit about the disease, that it is terminal and the average life expectancy after diagnosis was 2-5 years! It's like the story of the guy asking, "Well Doc, how long do I have to live?" The rest of the evening my parents and I were in shock and could not believe the diagnosis.

I went to Loyola Medical Hospital with hope and high expectations that the previous Doctor's diagnosis was incorrect. The neurologist's assumption was that it was most likely ALS but wanted to run tests to rule out any other afflictions. He did extensive blood tests, checked me for Aids that came out negative and performed an EMG. I had to wait six weeks for the results of the tests and during this time I was convincing myself that it has to be something curable and that I would be all right. All tests came back negative. He then scheduled me for a muscle and nerve biopsy and explained that if there was a degeneration in the muscle cells that it would be something other than ALS. If there was a degeneration in the nerve cells it would pretty much narrow it down to ALS. Unlike other diseases there is no specific test for ALS, several tests are performed to rule out any or all other afflictions, as I have mentioned before. The results showed a degeneration of the nerve. The last test was a spinal tap which might reveal the presence of bacteria or any other pathogens in the spinal fluid. Again the results were negative. I am now curious if antibodies were found in my spinal fluid since there is evidence ALS may be linked to a virus.

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At this point my guitar playing was worse and my hands were so weak I could hardly hold the pick. I had to quit Body Bag and playing was too difficult, frustrating and depressing to continue so I eventually had to give up my instrument. That was over three years ago.

1998

It is becoming more and more apparent that ALS (Amyotrophic Lateral Sclerosis) and many other neurological degenerative diseases are actually quite often misdiagnosed Lyme disease. There is no definitive test for ALS, diagnoses is based upon symptoms and by ruling out other treatable afflictions. There is no known cause or cure for ALS. so the question I and others often ponder is how can I be absolutely positive that I have ALS and not some other treatable illness?

I was diagnosed as having ALS by some of the top neurologists in Chicago. **After culturing my blood for four weeks by a research lab in Michigan, the results were positive in finding Borrelia Burgdorferi, the bacteria responsible for LYME DISEASE !!! I am not the only one, I know of thirty seven (37) other PALS who also have tested positive for Lyme.**

Dean died on May 17, 2001.

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Michael Cunningham

Michael was diagnosed with ALS and shortly afterwards with Lyme disease. He fought to get antibiotic treatment, but did not manage to get prolonged treatment. His autopsy results show that he died of Lyme neuroborreliosis. ALS patients' brains should be routinely examined for spirochetes, using dark field microscopy. The fact that this is never done shows how biased and dogmatic the medical field is - how nothing has changed, how the Medicine still is dominated by quacks.

Mike and Kim's story. Friday, November 24, 2000.⁶⁴

Hello everyone, so very nice to meet all of you. There are many blessings, in this nightmare, we have seen. Some of the very best friends, we could ever hope for, or dream of, we have met through all of this. We all will stick together, and we all will get through this experience, as confusing, and aggravating as it is, and in the end, we will be well, and have beautiful life long friendships, I just KNOW IT!!

Now, to introduce ourselves. My name is Kim, and my husband's name is Mike. We have four beautiful children aged 1 1/2 - 6 1/2. WOW, yes, I know!!

Mike's problems started back around September of 1999, with his right leg not working correctly. We originally thought, along with the doctors that this was probably a back problem, or neck problem, and from there Mike had all of the MRI's done, plus saw a back surgeon. We then found out, when they could find nothing there, and sent Mike off to a Neurologist, that something else had to be wrong. Along with this Mike also had some fasciculations (muscle twitching) in his shoulder, he noticed a little loss of strength, etc.

So, to make a very long story short, we sat in the Neurologist's office on the day of April 4th 2000, and heard her say that Mike had ALS, better known as Lou Gehrig's Disease, and he could have anywhere from about 1-5 yrs to live. OH MY GOD!!!! Needless to say, our worlds fell apart, we cried, no longer slept, couldn't eat, and the list goes on. Please note that the only tests that Mike had to diagnose his ALS, was 2 different EMG's, (which I heard were a hellish experience) and a basic blood test, to rule out some infections, or

⁶⁴ <http://www.angelfire.com/planet/lymedisease/2/Cunningham.html>

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whatever. The MRI's were done to rule out the pinched nerves and back problems, etc.

SO, then, after losing it, and crying out to God for several nights. I decided to ask Him to **SHOW ME WHAT I NEEDED TO KNOW TO HELP MIKE GET WELL.** I refused to believe that there was nothing that could be done, and that we all should just live with it. Then I met some wonderful people on the net, who suggested that Mike be tested for Lyme Disease. I read and heard Pat Pepper's story, plus several others. I met my now best friends, Terry and Matt from New York, who's story is almost identical to Mike's, and off we went to hunt for a Lyme Literate doctor.

Now to shorten things up a little more. Mike had a complete blood panel for Lyme done, the Elisa, the Western Blot, the tests that check for the co-infections, etc. He came back equivocal on two bands. Band number's 41, and 23. OH YES, I thought, FINALLY, now we can get some kind of treatment to stop this nightmare. Mike also has taken a LUAT test, which showed 2 out of 3 positive. Good enough for me.

Now is where, my story comes into play here. I got sick FIRST!!!! I have been very ill, and totally abnormal, you might as well say, for 4 1/2 years. I have a very long list of symptoms from anxiety, to sweating, to pain, to vision problems, the list goes on. I was diagnosed with Probably MS, and also Fibromyalgia, and all the other BS diagnosis' that goes along with this game. After hearing about ME, the doctors said "We need to test your WIFE". Ok, I figured, why not, I've had everything else done. Needless to say, I have now found out, after showing up on three bands myself, numbers 41,23, and 66 that I too have LYME DISEASE, and not that other crap.

Now, here is an update, and facts about us, that may be beneficial to all of you. **Mike was started on Doxy, and took that for 6 weeks. After stopping the Doxy, Mike noticed a BIG decline, and could no longer work.**

Then Mike was put on IV Rocephin, for 2 1/2 months, and lost that, due to the pic line closing up, and some reactions, which the doctor feared could be allergic reaction. Now Mike is trying the Bicillin shots, plus Flagyl, Zithromax, Diflucan (for yeast). Now then, I ask, "why such a rapid decline, and worsening SO FAST?" Mike went from walking with a limp, in April, when he was first diagnosed, to now, hardly being able to move. He has to use a wheelchair, plus his walker, to get around the house. His condition continues to decline rapidly. **He noticed a BIG decline, when he stopped the Rocephin.**

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That weekend, after that, he questioned whether he would make it through the weekend alive or not. The doctors that diagnosed Mike with the ALS, say that he probably has the rapid progression, and that they can't deny the Lyme, but they feel that the treatment will not have any effect of the ALS. WE DO NOT AGREE. Why would he have such a huge reaction to the antibiotics, if it wasn't linking into the ALS diagnosis?? Why would the antibiotics have any effect like that at all. We honestly can not say what will happen, but we are in a fight to save Mike's life, and anybody else, we are blessed enough to help along the way. We believe that Mike has shown the signs, that the doc is looking for, to point to LYME. We believe that if others have improved, or are well now, then Mike can TOO. So can all of you!!! I will give you all a list of Mike's symptoms and mine, so you can compare, but I find it very interesting that we have so many alike. I will keep you closely updated on me, and what I go through, so we all can compare, and hopefully see that most of this mess is LYME and not ALS.

Michael Cunningham was a staunch member of the ALS/Lyme group & a loving father and husband. His autopsy report just came back and his entry on the ALS/Lyme page has just been updated, as below:

Michael F. Cunningham died December 24th 2001. **He was diagnosed on April 4th 2000 with ALS and then shortly after with chronic Lyme disease and both co-infections.** He fought to get treatment. He fought with doctors, to try to make them see, to understand, to believe this. Now after his death, and having an autopsy done, we have the proof we need, to show that Mike's death was due to Lyme disease! **His postmortem diagnosis was Lyme disease, not ALS.** Nowhere in the autopsy report does it mention ALS - only Lyme disease. Mike's samples and organs will be kept for research and study, in the hope of helping to save lives. God bless you all, and thank you so much for your continued support and your prayers.

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Vincent Sota

Copyright Times Publishing Co. Jul 6, 2002⁶⁵

Vincent Sota fought big fires, but it was a tiny tick that rendered him a quadriplegic. Sota, a former Pasco County Fire Rescue driver engineer and emergency medical technician, died Thursday. He was 47. He started at the county in 1991 and worked in the Hudson, Seven Springs and Crystal Springs stations.

His wife, Mary, has been fighting to draw attention to his affliction for more than two years. **Vincent Sota was diagnosed in 1999 with amyotrophic lateral sclerosis**, a fatal nerve disease that renders its victims unable to walk or even talk. It is commonly known as Lou Gehrig's disease.

Mary Sota, 40, refused to believe the diagnosis. Her research led her to believe he had Lyme disease, which is caused by bacteria contracted from ticks and mimics many symptoms of ALS. Mary Sota said he caught it from a deer tick in Florida.

Detected early, Lyme disease is treatable, **but it took 15 doctors before Vincent Sota was diagnosed with Lyme disease.**

Every time his story got out, Mary Sota said she received e-mails and calls from people across the United States who suffered similar fates. They gave her hope, she said. Last year was the couple's 11th anniversary.

"He owes me 39 more," she told the St. Petersburg Times in December. "I'm not letting him go until I get 39 more years."

At that point, Vincent Sota was looking better. His 220-pound frame, which had withered at one point to 131 pounds, was starting to get bigger again.

He was home from the hospital. He was confined to a bed and couldn't speak, but he communicated with his wife by blinking. His children - David is 7 and Emiko two years older - climbed into his bed to kiss him.

His wife slept by his side on an air mattress - but never for more than four hours at a time. She needed to monitor the machinery that helped him breathe and eat. "I would love," she said, "to hear his voice again."

⁶⁵ <http://www.angelfire.com/planet/lymedisease/4/Sota.html>

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Brian Hill

"Brainhell" lived in the US and was the father of two young children. He died in early 2008. From his blog:⁶⁶

I was diagnosed with ALS on January 13, 2004. At the time, my blood test for Lyme disease was in the 'indeterminate' range -- halfway between negative and positive. A year later it was still indeterminate. These tests were done by Quest, a reputable laboratory. The experts who confirmed my diagnosis also tested me for various obscure, rare conditions, but reluctantly settled on ALS as a diagnosis. In my view, the experts were negligently incurious about the Lyme test results. These days, I walk with difficulty and my speech is slurred. I have lost a lot of functionality. However, I was still curious about the 'indeterminate' Lyme result, having heard that Lyme is a great imitator. With the help of various people on the internet, I located another lab, IgeneX, and had my blood tested for Lyme by them. IgeneX is controversial. They use a different methodology for analyzing the test results, based on published, peer-reviewed science -- but not generally accepted by doctors or insurance companies. IgeneX has even been investigated by the state of New York and, I think, the federal government, for improper conduct. In casual conversation, an expert at my ALS clinic had warned me that IgeneX is totally disreputable. My results from IgeneX were positive for Lyme disease. After quite a bit of searching, I managed, with the help of The International Lyme and Associated Diseases Society (<http://www.ilads.org>) to locate a 'Lyme-literate' doctor. He put me on a twice-daily dose of 100 mg oral minocycline, with the plan of getting me on two grams of daily IV ceftriaxone (brand name: Rocephin), shifting after 5 weeks to twice-daily infusions four days a week with three days off. The infusions might continue for 15 months, depending on my responsiveness. This is tremendously expensive, and you can see why insurance companies might not look favorably on it. The drug is no cure for ALS. Certainly it did not save Dee Chiplock, now deceased from familial (genetic) ALS, despite her daily infusions of four grams or more. It may, however, have prolonged her life, since she did last longer after diagnosis than other members of her family tree who were afflicted. Since January 10, 2005, I have been testing the grip strength in my hands daily, and recording the data in a spreadsheet. I use an instrument called a hand dynamometer. There have

⁶⁶ <http://brainhell.blogspot.com/2006/03/als-lyme-here-is-post-i-recently-made.html>

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been some periods where I did not collect this data, but generally the graphs have progressed downward in an inexorable trend. I had a left grip of 52 pounds in February, 2005, and by January 2006 it was at 19 pounds. My right side, which is less impaired than my left, declined from 100 pounds down to 65 in the same period. I was curious to see what effect, if any, the ceftriaxone would have on my grip strength. I began taking the minocycline orally while I scheduled with a vascular surgeon to place a "port" for IV drugs on my chest near the shoulder. But in the two weeks before the port installation, I began to notice a new trend in the graph of my grip strength. It was leveling off, perhaps even climbing, apparently as a result of the oral minocycline. By the time I had the IV port installed on March 2, the trend was clearly upward. Between January 19 and March 8, my left grip has improved from 19 pounds to 30 pounds, and my right grip (on the less-impaired side) from 65 pounds to 75 pounds. In my view, this result supports the notion that I may have ALS-like symptoms brought on by Lyme disease. I do not know what the future holds for me, whether I may recover from my symptoms and begin to walk and talk normally again, or whether I will suffer some kind of complications, or relapse. I don't claim that all ALS is Lyme, or even to know what causes my disability. But here is my message to those of you with ALS: Get a blood test for Lyme. If the test results are 'indeterminate' or even show a positive result for just a few markers, find a 'Lyme-literate' doctor and begin moderate, cautious treatment for possible Lyme.

Note how "BH" confuses the cause for familial ALS with that of sporadic ALS when he says that antibiotics certainly are no cure for ALS, because someone with familial ALS was treated with antibiotics and died. It is to be expected that antibiotics have little to no effect on the course of familial ALS, since its cause is genetical, whereas sporadic ALS has a suspected infectious etiology.

BH was later put on an insufficient dose of Ceftriaxone (2 grams a day as opposed to 4 grams a day or more) and he mentions that he had to pay it out of his own pocket:⁶⁷

⁶⁷ http://brainhell.blogspot.com/2005_01_01_brainhell_archive.html#Nature

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When I went to the pharmacy with the prescription, the pharmacist said that my company's drug plan does not cover injectables. The cost to me for one kit would be \$520.

However there is the possibility that if the doctor administers the drug, the cost may be covered under the "physician's benefit." That means that they cover what the doctor does to you in the office. So I called the able office assistant and she said that on Monday she will have the new office assistant call my insurer and see if we can get it covered that way.

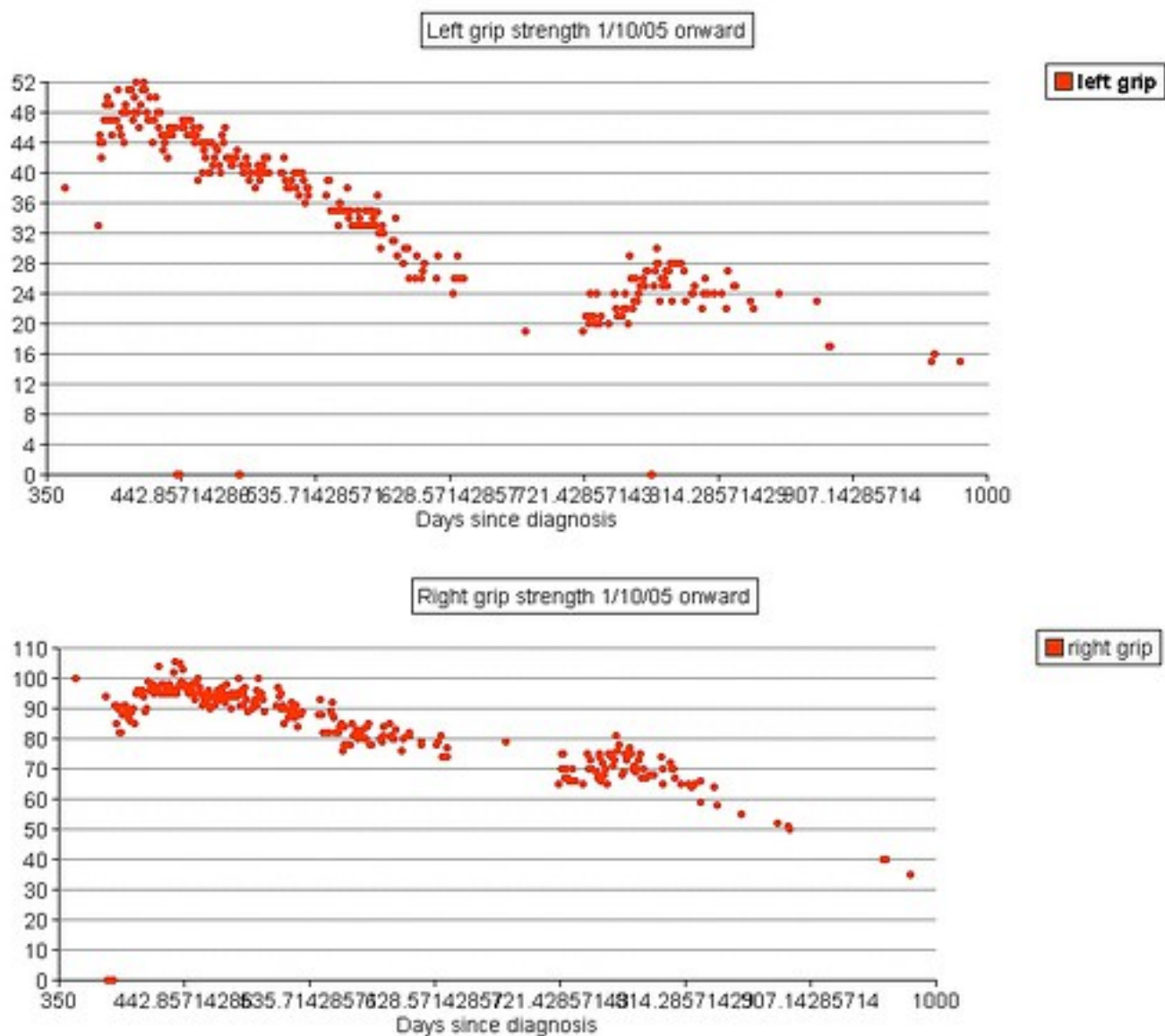
Those of you in other industrialized countries are no doubt recoiling in horror. Well, this is America. Sick people with no money are thrown overboard.

If it becomes the case that the drug will be covered on the condition that the doctor injects it, I won't mind paying a 10-minute visit to him three days of the month. And he'll be able to test my grip strength.

Brian mentions that due to their high cost, he'll likely only use the antibiotics one a week - which would of course lead to total treatment failure, especially at such a low dose.

On Friday, September 29, 2006 he writes that the following is a graph of his grip strength, and that the temporary increase was due to taking Minocycline for a while. For some reason he calls his LLMD (Lyme Literate Medical Doctor) "Dr. Quack", even though the Dr. is going to prescribe Minocycline again. Ceftriaxone did not work for him but Minocycline obviously did.

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Brian's last words on his blog were:

Wednesday, January 23, 2008

wife, i love you.

children, i love you.

He died on February 2, 2008.

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29 more people who died of Lyme-caused ALS

Peter Anthony Banducci

Lonnie Benedict

William “Billy” Boesché

Ross Boynton

Scott Brazil

Terry Cain

Phil Chapman

Michael Coers

Vickie Crawford

Katherine Alderson Crowe

Carole A. Van Doorn

Michael Keith “Mike” Gregory

Kenneth Hagen

Hawkes-Koons

Alexandria Hermstad

Michael Hinsberger

Brian Hirsch

Robert J. Hoffmann

Barry Horton

Michael G. Linebaugh

Balsorah Lamar Miller

Ed Parker

John Douglas Powell

Priscilla Moulton Shafer

Roberta J. Sorbello-Luongo

[Lori Hall Steele](#) (of the documentary "Under the Eightball")

Mike Thomas

Steven F. Wells

William James Zell

I wrote a separate article, available online, where I provide references.⁶⁸

⁶⁸ <http://unfictional.com/lyme/dying-of-lyme-disease-case-fatality-rate-nearly-100/>

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"Lymies" with ALS who are still alive at the time of writing

Travis Mitchell

Travis is a young man from Kentucky. This was posted by his mother Michele Mitchell on January 1, 2006:

When my son, Travis, was first diagnosed with sporadic ALS, at age 22, in November 2004, his symptoms were "steamrolling", by that I mean, between September 2004 and January 2005, he went from twitching in his upper arms to unable to walk unassisted. The greatest extent of his mobility problems stem from uncontrollable spasticity. He is taking Baclofen, oral, 20mg 3 times daily. He is still able to chew and swallow well, but his speech is declining (up and down) and I have noticed incidents of excessive saliva starting this November.

Travis was also diagnosed with Lyme disease in April 2005. Two positive tests, two separate labs. He also tested positive for Bartonella Hensalae and Babesia. He began Ceftriaxone IV (Rocephin) May 19th, prescribed by his neurologist. **Within three weeks, the fasciculations were reduced almost to the point of imperceptibility whereas previously they had been violent, continual and everywhere; arms, hands, legs, torso, tongue.** By the fifth week he looked better than he had in years; skin - tone and color, eyes - clarity and alertness, hair - texture, sheen. He also seemed to regain some of his energy/stamina.

He continued to do well into July, when we began some additional oral antibiotics, as well as a 21 day course of Mepron (Atovaquone) and a few weeks afterwards a rather large dose of Lariam. After the Mepron course, Travis stated that he could think more clearly than he had in years. The Lariam seemed to positively affect his cognitive abilities as well. However they were each severe in reaction physically. Each time he begins an oral antibiotic, he reacts, in varying degrees, with exacerbated ALS symptoms as well as flu like symptoms. Along with the Mepron and Lariam, Ketek and Tinadizole seem to affect him the most severely. Within an hour of a 400 mg dose of either, he cannot speak at all. Later the fever, sweats, achy joints, clammy, agitated (cranky) and malaise. With each day and each dose these reactions abate.

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Since July, he has continued to decline, albeit at a snails pace compared to "pre-treatment" speed. He has been on Ceftriaxone since May with only one three week interruption. During which the first week was same, second week the fasciculations started ramping back up and by the third week they were back with their old fury. We re-started the Ceftriaxone and by the third week they were calmed again.

Early on in this process of "pulsing" different oral antibiotics, Travis would "rebound" in between, but since late September of 2005, the health improvements we have seen with the ceftriaxone have become diminished. I believe this is due partly to lack of exercise (from not feeling good because of the flu like effects), and nutrition absorption problems (due to oral antibiotics effect on GI). Extra focus on these two areas over the last month, has shown slow improvement in health once again, but I hesitate to say that my belief has been proved out, as yet.

On January 14, 2010 Travis' mother wrote this:

I've posted before that **when my son Travis was on Rocephin, the neurologist chronicled improvement in his symptoms for 6 consecutive months.** He gained weight, looked better, felt better, his fasciculations lessened greatly, reflex, strength, ...everything. However, when we switched to Ceftriaxone, the improvements stopped. Interestingly, while on rocephin Travis was also taking Ursodiol. With Ceftriaxone the doctor said Ursodiol was not necessary after all.

I have asked before if the Ursodiol might have been a factor. I believe that fats and sterols are implicated in (at least our case) ALS progression in light of the finding that high cholesterol levels are linked with slower progression. You can bet I will be asking our family physician for an off label prescription for Ursodiol again now!

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Will ⁶⁹

February 2, 2008:

Some of you might already know that I've been fighting a mystery illness that started in August of 2007. It took 6 months for the "regular" doctors to give me a diagnosis of ALS. Even though I have health insurance I still had to pay out close to \$6,000 of my own cash to pay for 4 MRI's, 2 EMG's, 4 Blood tests. It took 6 months because the doctors all needed vacations. Meanwhile I worked my business, Barker's Buckles to the bone during November and December while I was getting more and more deep into the illness. For months, I had been telling the doctors I thought I was bit by a Tick in late June. They kept telling me I didn't have Lyme Disease giving me reasons that I could fact check on the internet. They where so wrong and misinformed about Lyme. In the end the big shot UW doctor told me I had ALS, drew a graph with 18 months and 10 years, and pointed that I was in the middle and likely to live 4-6 more years. This was Jan. 10th.

A week later, I saw a Lyme Disease Specialist and without hesitation she thought that it was very clear that I had Lyme Disease. In fact when I asked the UW doctor about my Lyme symptoms not related to ALS, all he could come up with was "You have anxiety and depression". I was put on Antibiotics that evening, now month 7 of being infected. There is so much that I'm leaving out. I've lost faith in Healthcare in America. They where all too busy to listen, too busy checking off what to test at the blood lab. They tested my electrolytes 4 times.

Often I would need to pitch in and fork out \$30 for each blood test since my insurance never paid the whole cost. Where they just generating business for the lab? The EMG studies where to verify that I had muscle twitches. Yeah, I know I have muscle twitches. The bills totaled \$4,000 for the 2 tests. My insurance paid about 51% and I paid the other 49%. They pretend that the test shows so much information about the muscles twitches like "it seems to come from your brain or your spinal cord". Good, I'm glad they don't come from my knee or an ugly dog or a rusty town like Detroit, Michigan. What clowns! Snake Oil Hockers, Hocus Pocus Bullshit! This isn't Mexico is it?

I'm waiting for the results of a blood test sent to an independant California lab.

⁶⁹ <http://www.myspace.com/mookiewill/blog>

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If you ever think you got bit by a tick, avoid the regular doctors and the CDC controled labs. Seems the goverment wants to create the illusion that Lyme Disease is still rare. Its bigger than AIDS. Otherwise they will waste your time, money and might destroy your life.

I'm 2 weeks into Antibiotic treatment and I'm nervous. All I have is a clinical diagnosis of ALS and a clinical diagnosis of Lyme Disease. A positive Lyme test will clear the air and help me move forward.

In December, I put my eBay store on "vacation" because I couldn't keep up anymore. On December 18th, my Amazon store was suspended for a week because I fell behind on shipments. They held my money for almost 4 weeks.

I estimate I worked both November and December to pay all my medical bills. By December it was clear that my walking was getting bad and I had a clear limp and had problems going down stairs. I lost my balance several times at home and fell on my ass. My body would wake me up 4-5 times a night.

Sometimes in sweats, others with chills and tremors. I had lost 12 lbs in 3 weeks at one point in October. October was a giant blur to me. I had weeks of mental confusion, disorientation, balance problems, vision changes, ringing of the ear. Just about every medical worker I came in contact with would ask me if I was diabetic or if I had a thyroid issue. By November it seemed to clear up some, but I still had twitches, sleep distrubances, and now walking was becoming an issue. By December friends noticed that my speech was slurred some days. I would often get stuck on words or forget the next word. I started to make mistakes in my business. "Temp" closed my Amazon store a few weeks ago.

I never wanted to be a Lyme Disease Advocate, I just had to become one to save my ass. The one that got me was smaller than a poppy seed. My crime? I sat on a rock wall at a camp west of Gig Harbor for 5-10 minutes. Rested my arm on the top of the wall. The bite was on my right wrist. One morning in the shower, I picked off the tick thinking it was a tiny cat scratch. The bruise showed up weeks later. It took a few weeks for me to put it all together and suspect Lyme Disease. It took the doctors 6 months to get me a misdiagnosis.

Wish me luck!

I've been faking it for so long, but I can't hide it anymore.

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Might be on Antibiotics for several more months. God damn ticks!

Nobody ever suspects the Tick!

March 1, 2008:

Lyme update: **Tested positive for Lyme Disease and also tested positive for a co-infection Babesiosis.** A side effect of Babesiosis is death. It is a blood parasite similar to Malaria. Both come from the bite of a tick. Lyme is a bacteria.

My doctor is more concerned with Babesiosis right now. Starting a new rx tonight Mepron and Zithromax, for the next 3 months.

December 30, 2008

Today I walk with a walker, sometimes have a hard time getting up from chairs, and bed. One Michigan Dr. says it looks like MS. My grip test is 30 L-60 R. I'm in Physical Therapy, and I have a PICC line inserted in my left bicep, the line going to the area above my heart. I'm on IV Rocephin twice a day. I have Neurological Lyme - looks like ALS or MS. My Lyme MD here in Michigan told me he thought Lyme was the cause of diseases like ALS, MS, Parkinsons. The first time he saw me he asked me to pray with him. I passed. Now he says because my symptoms seem stable - it mitigates against the ALS.

I'm still running Barker's Buckles with the help of Mom & Dad. My customers don't know I'm sick. I don't have much pain. My legs and back are stiff in the morning, but feel almost normal after a shower. My nurse Lisa visits every Tuesday.

Hope everyone is doing fine. What a crazy year!

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*Pat Murray*⁷⁰

I was diagnosed with bulbar onset ALS in September of 2009, exactly one year and one month following a tick bite misdiagnosed by my then-MD simply because the rash was not in the shape of a bull's eye. A return visit within 10 days complaining about low-grade fever, stiff neck, blasting headache, and incredible fatigue topped off by the worst sore throat I'd ever had in my life did not raise any concern either. This was dismissed as viral, and having to run its course. In better-informed hindsight now, I'm glad I did not accept the steroids treatment offered. The headache stayed with me for two years, getting only 'less-worse,' never going away, until...

I recently discovered that I have late, disseminated Lyme disease and have been undergoing treatment for it with antibiotics. Ceftriaxone 2g per day for 6 months. some Amantadine for the viral component of a Lyme infection, followed by massive doses of ascorbic acid. This treatment is helping me maintain strength, and I have even been able to make gains there.

So, with the blessing of Jane, my homecare PT person who is grinning with me from ear to ear over my progress to date, and Andy, my neurologist-hero, I am signed on to a rehab facility about 40 miles from my home, and I am excited to be 'up to it.' A few months ago, the mere thought of it would have made me want to pull the covers up over my head! I've met the therapist there and she is not one to give in, no matter what the books say. My kind of therapist! Jane's the same way.

Recently, I connected with another PALS whose condition is more advanced than mine is. I'll call him David, until he decides whether to participate in this forum. With his diagnosis of ALS, further investigation was halted and treatment was limited to the palliative, until someone nagged him and his caregiver into ferreting out whether he had Lyme disease or not. Anyone who has tried to do this knows that it can be enormously difficult to figure it out... But he did. **When David successfully treated his Lyme infection, his ALS progression was stopped dead in its tracks.** I believe I am experiencing the same thing. But time will tell.

⁷⁰ <http://www.als.net/forum/Default.aspx?g=posts&t=48447> 4810 Haymarket Dr. Virginia Beach, VA 23462, United States

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I look forward to meeting David in person very soon to hear his story - and the reason I will do that is because I want his survey entry data to be the first in the database I'm building to produce reports that will show the frequency with which Lyme infections co-exist with other neurological conditions such as ALS, MS, Parkinson's, and Alzheimer's.

Having ALS symptoms onset a month following a tick bite is just TOO much of a coincidence for me to ignore. What do I believe?? I think we dismissed the possibility that disseminated Lyme disease can wreak exactly the havoc observed in these conditions waaaaay too soon. There are some who say that ALL ALS is end-stage Lyme. Of that, I'm not sure - but based on what I am learning from reading lots of journals, I can also say that I am sure it is possible that a Lyme infection could be a leading cause, a trigger, or, or - because Lyme infections tinker with the immune system altering genetics... Yep, the little bugs carry plasmids in their cytoplasm and have been observed exchanging them with the cells the innate immune system sends to identify and destroy them.

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Make sure proper Lyme tests are done - they never are!

Virginie Bijon's Lyme test was negative and she did not protest

Virginie - originally French - wrote this on November 4, 2009. She died eleven days later in Toronto. She never received antibiotic treatment because a Lyme test came back negative. Lyme tests are notoriously unreliable, so by trusting her doctors and accepting that negative test, she perhaps agreed to a death sentence. As Dr. Martz described, he only tested positive for Lyme after taking antibiotics - and he did not take an unreliable Lyme test either. Virginie's test undoubtedly was unreliable, as the medical mainstream has agreed upon two-tiered testing: First an ELISA with up to 95% false negatives (depending on which expert you ask), then a Western Blot with up to 60% false negatives - but only when the ELISA was positive.

At 41, I considered myself an accomplished woman. I had two healthy, smart children. I was happily married to a supportive man. I had an enviable career as a marketing executive. I was super healthy, exercised regularly with a personal trainer and was always careful to eat what was good for me. Things started to change in July, 2007, with a weird sensation in my left leg. No pain, just a kind of stiffness that made it difficult to walk and forced me to make a conscious effort to activate my muscles for an activity that should be automatic. I also noticed the muscles in my left thigh twitched uncontrollably.

When time didn't heal my leg, I decided to see a chiropractor. We had several sessions with no result. My walking was getting so awkward; I would follow someone and get into the rhythm of their steps to help pace my own movement.

The turning point was in October while walking down our street. My left ankle gave way but instead of bouncing right back, it collapsed and I fell to the ground in great pain. As it turned out, my ankle was broken. That was when I admitted I needed to see a doctor.

Six weeks later, I went to my first appointment with a neurologist. He

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confirmed there was something wrong in the way my nerves were sending signals to my muscles, but further tests needed to be done to understand why. I had three MRIs for my brain, neck and spinal cord.

My next appointment was short. The doctor had prepared himself. “I am not specialized in this area and I have only come across two cases before. I am quite sure you have ALS. It's a neuromuscular disease that affects the nerves that make muscles move. I am very sorry to have to tell you this.”

Still shell-shocked by the news, I drove back to my office and Googled ALS. I read that amyotrophic lateral sclerosis, or Lou Gehrig's disease, can be fatal within two to five years of being diagnosed. It is a disease with no cure or treatment that progressively paralyzes sufferers until they can neither breathe nor swallow. Some people with ALS refer to this moment as their death sentence, and that's how it felt to me. My husband was much more practical. We needed a second medical opinion.

I recovered well from my injury and was determined to walk every step left in me. I fell quite a bit and started using a cane. I now had twitching in every muscle.

Our first visit with a specialist was in February, 2008. The doctor explained that ALS was difficult to diagnose and was the last possible thing when all other illnesses had been ruled out. So I was booked for a day-long series of tests. I counted 17 samples of blood that would test, among other things, for Lyme disease, West Nile virus, rare forms of cancer and so on.

The whole process gave my husband and me hope. What if I “only” had cancer? Hope gave me energy, and during the winter I went for long walks with my husband and my dog, equipped with hiking poles.

Unfortunately, all the tests came back negative. “Wait and see how your condition evolves,” the doctor said. “We'll see you in August.”

I had a lot of ALS symptoms but not all of them. The stiffness in my legs and arms caused my difficulty moving, so I was prescribed a potent muscle relaxant. At work, people kept asking why my ankle wasn't getting better and

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why my walking was so bad. My medicine made me so drowsy I would literally fall asleep. Most of all, I didn't want to spend my last days of relative mobility working. My final day at work was April 21, 2008.

That summer, I had a full clinical review with a physiotherapist, occupational therapist, speech therapist, breathing specialist and nutritionist. Finally, the doctor saw me to confirm the bad news: I had ALS. I received three hours of care daily to help me with breakfast, getting bathed and dressed. I was also entitled to a nurse visit every week and a doctor available 24/7. I discovered the devotion of caring people who came forward to help our family and especially to relieve my husband, who had become my primary caregiver.

I kept track of the progress of my disease in terms of the activities I could no longer do. I dressed by myself for the last time in June. I drove for the last time soon after. I cooked my last meal July 1. I had a few falls, one of which landed me in the hospital needing 12 stitches in my jaw, and the other one at the dentist with two broken front teeth. I lost my ability to wash and feed myself and brush my teeth.

The worst, however, was my mouth. Like most people with ALS, I have swallowing problems and had to adapt my food to my ever-evolving condition. But what became more worrisome was that my speech was getting slurred. It was as if my mouth was forgetting how to form the sounds, as if my tongue and my lips were numb and too slow for the words to come out properly. The ALS clinic gave me a special computer that allows me to write with a mouse and a keyboard displayed on the screen. It takes a long time, but then again, I have time.

It is hard to believe there are still diseases with no cure. I am now 43 and two years into my journey with ALS. I have lost more than 30 pounds out of my original 125 pounds. Bound to a wheelchair and this communication device, I feel in prison in this useless body of mine, and the silence isolates me from those I love. I am scared for this last leg and I pray that it will be short. I have declined all forms of life support, including a breathing machine and feeding tube. I long for the end because my spirit will be free at last.

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Why is the Lyme - ALS link still ignored by medicine?

Since the publication of the Halperin paper, approximately one hundred thousand people have died of ALS in the United States alone. At the time of writing, 22 years have passed since the publication of that paper. As of yet, there has been scant progress in treating ALS, in spite of the fact that of a group of 24 people mentioned in the paper with ALS, 21 tested positive for Lyme. And that this could, beyond any reasonable doubt, only have been the case if Lyme disease is the cause of ALS.

Why this inaction, why this lethargy in the face of an obvious relationship between Lyme infection and Amyotrophic Lateral Sclerosis? Infectious disease specialists have no choice but to follow the pack. We have to remind ourselves that medical doctors are not scientists. Doctors are not microbiologists. Microbiologists look for the causes and cures of diseases. Especially Western doctors follow orders. They do what the negotiated contracts with the insurance companies tells them to. They do what their "experts" tell them to. They prescribe Big Pharma's expensive, patented symptom relievers. The doctor who experiments can look forward to a malpractice lawsuit. The medical industrial complex is run by multinational pharmaceutical corporations and only those medical professionals that follow the party line have a chance of a career. A doctor is not unlike a judge. A doctor applies medicine as a judge applies justice. And just as "justice" in a court of law is defined as what the law says is permitted and forbidden, medical diagnosis and treatment is defined by what its self-declared experts say is recommended and not recommended. Just as high court judges in the US are appointed by political parties, the top medical specialists, the high priests of medicine, are "appointed" by Big Pharma. It's all about money.

A doctor has very little latitude in how he treats a patient. Just as a judge has to follow jurisprudence, a doctor has to follow established medical practice - even when it flies in the face of medical facts that are perhaps disputed or

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not universally known, but proven facts nevertheless.

And apart from the with Lyme patients infamous rheumatologist Dr. Steere, the Halperin study was well-received. Dr. Steere who, with his frequent and highly publicised attacks on chronic Lyme patients has staked his scientific reputation onto the non-existence of chronic Lyme disease went on to conduct his own study. It should not surprise the informed reader that he did not find a single ALS patient positive for Lyme disease.

Steere's influence on medical opinion on Lyme disease is highly controversial. An anti-trust investigation initiated by Attorney General Blumenthal of Connecticut produced a report in 2008 that appeared highly critical of an Infectious Diseases Society of America (IDSA) guidelines panel, on which Steere had played a leading role. The judgement stated that "The IDSA blocked appointment of scientists and physicians with divergent views on chronic Lyme..."

Steere's study of course does not disprove the Halperin study. Absence of evidence is not evidence of absence. He had a high stake in its outcome. One can't prove that something doesn't exist. But one *can* prove that ALS patients are overwhelmingly Lyme-positive. The Halperin paper did just that.

Its authors admit that the logical conclusion would be that Lyme causes Motor Neuron Disease (ALS). And they say that they were "puzzled", especially because antibiotics caused in a significant number of cases either strong improvement or spectacular deterioration, which would in both cases be indicative of a neurological infection. Yet they dismiss any causal relationship between Lyme and ALS. *They do not believe it*, they say. It must be a *coincidence*, they say. Thus they remain within the constraints of medical dogma but are obviously drawing the wrong conclusion - which anyone who went to school can verify, because they report a statistical correlation of an acquired Lyme infection and ALS be 0.88, which is three orders of magnitudes higher than what it should be, namely 0.00086.

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Instead of seeing virtually no correlation whatsoever, we see a nearly perfect correlation. Therefore, statistically, the likelihood that we're dealing with a coincidence is *vanishingly small*.

What would have been the consequences of drawing the only logical conclusion?

1. Halperin et. al. would draw the negative attention of IDSA. The Infectious Disease Association of America has always maintained their position that Lyme disease is trivially easy to cure with a short course of antibiotics and that there is no such thing as chronic Lyme disease. Whereas LLMD's (Lyme Literate Medical Doctors) and microbiologists worldwide have been warning that late-stage Lyme is common and that the disease is virtually incurable at that stage, causing symptoms indistinguishable from Multiple Sclerosis, Alzheimer or ALS. The IDSA has enormous clout. It sets medical standards and when standards are violated by doctors, doctors may find themselves investigated for alleged malpractice. The IDSA would have to explain their defiant stance - that has always been dismissive of the real experts.

2. Halperin et. al. would draw the ire of the self-proclaimed Lyme "specialists" - especially Allan Steere. Steere has spared no effort in making Lyme appear innocuous. He has implied that the majority of chronic Lyme patients are hypochondriacs and malingerers. He has implied that most LLMD's are charlatans and quacks. Steere had a major financial stake in the Lymerix vaccine but that vaccine was taken off the market due to side effects. Steere holds key Lyme-related patents and has a massive financial stake in how the disease is defined, diagnosed, treated and prevented. Steere would hate to be proven wrong all along, especially considering the fact that he has gone public in the New York Times, alleging how he needed the very best bodyguards because those crazy people who were imagining to suffer from chronic Lyme were "threatening to kill him". He has never supplied any credible evidence for those claims - but they served his purpose of further

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marginalizing chronically ill people. Apart from his massive ego, a lot of money is at stake for Dr. Steere, who continues to dismiss chronic Lyme patients as hypochondriacs - even though he is a rheumatologist and neither a neurologist nor a psychiatrist.

3. Halperin et. al. would turn the "ALS experts" into mortal enemies. Those "experts" (of nothing, as they can offer neither an explanation of the cause, nor any significant treatment) derive their status and income from ALS remaining enigmatic. If it would become clear that it's an infectious disease of the central nervous system, their claims to "expertise" would evaporate, and all that would be left would be a cry for adequate diagnosis and antibiotic treatment.

4. Halperin et. al. would bankrupt and eliminate many ALS charities, foundations and patient advocacy groups.

5. Halperin et. al. would cause many millions of dollars of damage to those pharmaceutical companies that are invested into developing symptom relievers for ALS, or are already selling them. Big Pharma only makes money when we are sick. It is vastly more profitable to peddle symptom relievers indefinitely than to sell a cure. Besides - a cure for a bacterial infection of the CNS likely can't even be patented any more, as the patents for most antibiotics have expired.

6. Medicine has a long history of turning their sages into pariahs. Revolutionary breakthroughs are rarely embraced without a fight. Those with the audacity to disclose them are often ridiculed and professionally ostracized, such as [Ignaz Semmelweis](#), who discovered that washing one's hands before delivering a baby nearly eliminated the substantial risk of the mother dying of infection. Semmelweis died in obscurity in a mental institution, considered by his peers to be hilariously wrong about his silly concept of such a strange concept as "hygiene". Only after his death were they slowly forced to accept his theory of germs causing infections. By that

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time, innumerable women had died because doctors continued to be unwilling to wash their hands.

As similar story is that of James Lind. He found that men suffering from scurvy were cured when given oranges and lemons and he published his findings in the Treatise of the Scurvy in 1753. He was the first person to understand the importance of what would later be called vitamin C. His findings were not widely accepted by the rest of the world and scurvy continued to cause widespread death throughout the 19th century.

Medical doctors are rather average and ordinary people - resisting change when it means they have to make extra effort. People prefer to ignore facts that urge change. People resist changing the status quo ever so slightly in their disfavor unless they are forced to, even if it means risking countless deaths for others. Halperin et. al. appeared to be satisfied in showing that Lyme appears to be directly implicated in ALS - but the team obviously did not want to end up like so many before them - ignored, marginalized and criticized at best, but with a real prospect of a ruined career. This way, they still would get the credit for the discovery but they avoided the implications of All Hell Breaking Loose over their heads if they would have ventured taking their study to its logical conclusion.

Most medical doctors are not interested in making new discoveries. Or else they would be microbiologists, or at least very busy trying to discover the cause of all those thousands of afflictions with impressive sounding names, but neither cause nor cure. Doctors are not engineers, who need a lot of deep understanding of the subject matter - or bridges collapse. Patients collapsing is what doctors expect of them - nearly all serious illnesses have no known cause and no effective treatment. Doctors are not computer programmers. When a doctor makes a logical error, his boss will not reproach him. There is no schedule that will slip, a machine to go awry or a customer that will get angry. All a medical doctor does is do what he's told - by medical journals, hospital leadership, insurance companies and oversight

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bodies. Doctors know nothing really helpful about 99% of serious illnesses. And there are absolutely no consequences - because they are the self-appointed high priests of Medicine. And there we hit upon the core of what's wrong with the medical field: It is dominated by beliefs instead of facts. The absence of common sense instead of logic. Financial forces instead of best care. Your doctor doesn't know any better. He has been brainwashed.

It is easily verifiable that they are bombarded with propaganda. Take Lyme disease. Not ALS in some cases being Lyme disease - just Lyme disease by itself. Doctors are being told that it is a bad idea to test people with Lyme symptoms for Lyme disease. They are being told not to treat longer than a few weeks. They are being told that there is no such thing as chronic Lyme disease - even though there is hard proof that Lyme disease is massively underdiagnosed, that Lyme tests are so unreliable as to be virtually useless, that neuro-Lyme causes MS-, Alzheimer's- and ALS-like symptoms, that patients usually see dozens of doctors before they find one who makes the correct diagnosis and so on.

A random sample from Medicine & Health Rhode Island of July 2008:⁷¹

"When is a somatic disorder psychiatric?" by Joseph Friedman

Dr. Friedman expresses **no faith in doctors who are "self-proclaimed Lyme experts"** and, ignoring all the evidence to the contrary, says that long-term antibiotic treatment for post treatment Lyme disease *"doesn't work"*. **He suggests in the article that people who are still sick after a few weeks of antibiotics are neurotics.** Consider this however: The treatment duration for neurosyphilis, another spirochetal infection of the CNS is often longer than a year. The same with Tuberculosis. But Dr. Friedman thinks two weeks will do for Lyme disease. Dr. Friedman warns about the bad side effects associated with using antibiotics - without having made a cost/benefit analysis for chronic Lyme patients – because that would

⁷¹ <http://www.unfictional.com/pdf/msm-anti-lyme-propaganda.pdf>

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have proven him wrong.

"Introduction: Lyme disease" by Jerome Larkin and Jennifer Mitty

The authors claim that "*Over the next few years, effective antibiotic regimens were developed*". That is not supported by the available evidence. Many people go on to develop late-stage Lyme disease - wrongly considered psychosomatic or auto-immune by mainstream medicine. They call neurological symptoms "atypical" for Lyme, which is again wrong. Neurological symptoms have traditionally been dismissed by doctors, but atypical they are not. They lament on how patients have been "*threatening*" doctors, demanding antibiotics. And how "*dangerous*" antibiotics are, of course. Especially for people diagnosed to have the world's most evolved bacterium eating away at their brains.

Then they say that even the sickest patients - those with severe neurological symptoms and even heart block **should not be treated with antibiotics unless they test positive for Lyme - and even then not longer than a month.** They condemn to death anyone with neuroborreliosis with negative test results - and there is ample proof that those tests are utterly unreliable. Their advice is either corrupt or criminally insane. They condemn to death anyone who still have one living *Borrelia* bacterium left in their brain after a month of treatment - while *Borrelia* can easily encyst itself for a few months, or produce DNA granules that can lie dormant and later grow back into spirochetes. *Borrelia* reproduces very slowly so it needs a much longer exposure to antibiotics, *Borrelia* is known to survive even after years of antibiotics. But without antibiotics, the patient steadily deteriorates and with the right antibiotic regimen, the patient slowly but steadily improves.

They go on to claim that the CDC guidelines are based on "*the best available scientific evidence*" (a blatant lie) and say that ***under no circumstances should any Lyme patient get more than a month's worth of antibiotics.***

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"Musculoskeletal manifestations of Lyme disease" by Imad Bitar and Edward Lally

Why is it that the only authors published in "industry rags" such as Medicine & Health are those that tow the party line of "Chronic Lyme disease does not exist"? The authors claim that all and any ongoing neurological symptoms after "*treatment adequate for Lyme disease*" can't be due to an ongoing infection, but must be due to something else. What that would be, they say they don't know. But it certainly can't be Lyme disease any more they say, after "adequate treatment". Which lasts never more than four weeks.

Why the double standards for other spirochetal brain infections? Neurosyphilis is treated as long as there are symptoms. One year is the minimum. Three years is not uncommon. Sometimes, treatment is open-ended. Could it be because Lyme disease is more prevalent than Syphilis? A year of intravenous antibiotic treatment would be extremely costly for insurance companies. Or is it Big Pharma, who prefers to sell you a lifetime of symptom relievers rather than a cure? It is hard to say whether we are dealing with ignorance or malice at the very top. Likely a combination of both.

"Neurological complication of Lyme disease" by Syed Rizvi and Amanda Diamond

Their story has, in huge print in the center: "*Prolonged courses of antibiotics do not improve outcomes and are not recommended*" - a blatant falsehood - do they get kickbacks from the medical insurance industry or are they just regurgitating drones?

They claim that without a positive Lyme test, neurological symptoms can not be Lyme. They claim that even when a patient has Lyme antibodies in the cerebrospinal fluid, it should not be assumed to be Lyme. Absolutely disgusting - **they hand out death sentences to those unlucky enough**

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to have a false-negative Lyme test as well as those with a positive Lyme test. They encourage the medical specialist to find pretexts to deny treatment. Perhaps the patient lives in a "non-endemic" area? These folks really take the cake. They say that it's better to prescribe a little Doxycycline for a while. No need for intravenous antibiotics. Lyme disease is easy to cure! If you do administer ceftriaxone or claforan, don't bother with a month - two weeks will do!

Still sick after that? "Post-Lyme syndrome" - please don't whine or we'll send you to the asylum!

"Updates and controversies in the treatment of Lyme disease" by Jennifer Mitty and David Margolius

Again, displayed prominently in the center of the article is the text: *"At this time there are no randomized controlled studies that show a sustained benefit of long term antibiotics"*.

Wow. You know what that means? **That they can't deny anymore that there are randomized, controlled studies that show that long-term antibiotics work at least for the duration of the study and follow-up period.**

Yet they claim in the article that there is no such thing as treatment-resistant Lyme (treatment being defined by some weeks of low-dosed antibiotics) and that instead of antibiotics, people who still suffer from the debilitating effect of the infection should take *antidepressants*.

Doctors have been hammered with this disinformation, so not even Lyme patients will get proper diagnosis and treatment. And you think that someone with "ALS" will be diagnosed with Lyme and receive proper treatment? Impossible, because current medical dogma does not allow it.

How to counter disinformation about the ALS-Lyme link

It is generally taboo to suggest on ALS forums that Lyme neuroborreliosis could be a significant cause of ALS. Some ALS forums have been set up by companies with a commercial interest in ALS being its "own" disease, and on such forums, any hint of "dissent" is met with stern admonishment. ALS associations routinely disseminate disinformation about Lyme disease.⁷²

Other ALS forums are dominated by doctors who consider themselves "experts", and they will let no opportunity slip to attack the Lyme-ALS angle.

And then we have Joe sixpack the average ALS patient, who merely goes with the flow, does not think for himself and simply regurgitates whatever is considered the most uncontroversial point of view.

People are usually highly intolerant towards views that oppose those that they are heavily invested in, and ALS patients who have accepted their fate have "flicked a switch", they have come to terms with their imminent demise and resent the thought that perhaps there still is a way out - or worse: That perhaps their doctor has made a key mistake and they could have been on the track to recovery. It is the "head in the sand" attitude.

Doctors, especially neurologists and ALS specialists, are also heavily invested in ALS being "its own cause", because they are regularly condemning people to death who suffer from it - usually without even bothering to order a Lyme test. If ALS would be caused by Lyme disease in a significant number of cases, they would be guilty of medical malpractice of the worst kind.

Those forum participants that seek to make ALS patients aware of the Lyme-ALS link will have to tread on eggshells and will be the subject of constant ridicule. Depending on the forum, they will face a barrage of abuse or a plethora of baseless claims, "debunking" the Lyme-ALS connection.

This chapter identifies the most commonly used false claims, and how to correct them.

⁷² <http://www.unfictional.com/pdf/ALS-disinfo.pdf>

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"Lyme doctors use dodgy IGeneX Lyme tests that are always positive"

Nonsense. Dr. David Marz, the MD who had been diagnosed with ALS but got better with antibiotics after testing positive for Lyme, tested Lyme-negative several times on the much-maligned IGeneX tests, until he used antibiotics prior to testing. No Lyme test has no false negatives, but it is important to understand that there is no such thing as a false positive. Testing positive means that it is an absolute fact that living *Borrelia* spirochetes have once found their way into the bloodstream. And as long as it is not proven that they are fully eradicated from the CNS - something exceedingly hard to do since there is no reliable test to date that can exclude neuroborreliosis - it has to be assumed that a case of ALS could be a Lyme symptom. There are countless examples of people testing Lyme-negative on IGeneX tests.^{73 74 75 76 77 78 79 80 81 82 83 84 85 86}

IGeneX' stance is that Lyme testing according to the deliberately limited "Dearborn" criteria finds only 8% of Lyme infections. IGeneX's explanation:⁸⁷

The problem with "ordinary" Lyme tests is that they have been made so insensitive that they are worse than useless. Western Blot tests are usually specific to the B31 strain of *Borrelia* only, while there are many other strains. And they do not look for band 22, 23, 25, 31 and 34, even though those bands have been patented as the most specific bands for *Borrelia*.

Therefore, the very best Lyme tests that are commonly used suffer from 69%

73 <http://www.alsforums.com/forum/archive/index.php/t-1868.html> (search for "Igenix")

74 <http://www.healingwell.com/community/default.aspx?f=30&m=2156991>

75 <http://www.medhelp.org/posts/Lyme-Disease/Desperate--Please-help-with-diagnosis--cognitive-dysfunction--igenex-results/show/1319098>

76 <http://www.roadback.org/forum/viewtopic.php?f=1&t=5439>

77 <http://index.healthboards.com/lymedisease/866014/negative-igenex-lyme-test/>

78 <http://www.boarddigger.com/t-pnGfjlvwjl/Lyme-Disease-Breathing-muscle-twitches-nausea>

79 <http://www.prohealth.com/lyme/blog/boardDetail.cfm?id=4061>

80 <http://www.latitudes.org/forums/index.php?showtopic=9610>

81 <http://phulicohanmd.com/?cat=26>

82 <http://www.medhelp.org/posts/Lyme-Disease/Question--Anyone-ever-heard-of-any-negative-results-from-IGeneX-labs/show/1449258>

83 <http://www.roadback.org/forum/viewtopic.php?f=1&t=3959>

84 <http://www.boarddigger.com/t-u9jiLzL5hm/Lyme-Disease-My-IGeneX-test-results>

85 <http://eu-es.facebook.com/topic.php?uid=2239977820&topic=9299>

86 <http://www.arthritisinsight.com/archives/test12378.htm> (search for "I tested negative by Igenix")

87 <http://www.unfictional.com/pdf/igenex-lyme-testing.pdf>

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false negatives, and that is best-case. With ALS patients, that figure may well be much higher, because of documented OSP variation in the brain - an immune-privileged site. Meaning, there are no IgM antibodies and very little IgG antibodies circulating any more, months or years after infection, but an active infection is firmly established in the CNS. A PCR of cerebrospinal fluid usually turns out negative as well, because spirochetes avoid this fluid like the plague, as Dr. Øystein Brorson has shown.

Tom Grier is a Microbiologist at the University of Minnesota Medical School, Duluth, Minnesota, USA. He explained why Lyme tests are notoriously unreliable - even less reliable than tossing a coin.⁸⁸ Dr. Grier wrote in even much deeper detail⁸⁹ about the deep inherent flaws of Lyme testing.

⁸⁸ <http://www.unfictional.com/pdf/unreliable-lyme-tests.pdf>

⁸⁹ <http://www.unfictional.com/pdf/when-accurate-lyme-tests.pdf>

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"Antibiotic trials showed that antibiotics make ALS worse, not better"

False. There have been many theories postulated about the etiology of sporadic ALS - viral, autoimmune, cyanobacteria, glutamate transport, free radicals - but in spite of the fact that neuroborreliosis is known to be able to cause ALS, a bacterial etiology has, paradoxically, never been seriously considered.

We think the reason for this glaring omission, this elephant in the room is the "Lyme controversy". In spite of overwhelming evidence to the contrary, government guidelines, insurance companies and self-proclaimed "experts" with financial stakes in the status quo claim that chronic, treatment-resistant Lyme neuroborreliosis doesn't exist and that current diagnostic procedures and treatment protocols are fully adequate. For decades there has been epic trench warfare going on between Lyme patients and their Lyme-literate doctors on the one hand, and financial stakeholders in not diagnosing and treating properly on the other hand. Just as in the battle of Verdun, progress is measured in individual trenches and the number of victims in this battle can't be counted any more.

Therefore, were anyone to propose a bacterial etiology of ALS - say the spirochete *Borrelia burgdorferi sensu lato* - he would summon the wrath of the entire medical-industrial machine. For it would greatly harm Big Pharma, if it turned out that ALS, MS, ME, CFS, Parkinson's, Alzheimers and a host of other neurological syndromes are neurospirochetoses and effectively curable with the right antibiotic approach. Perhaps some new antibiotics would have to be developed - a totally neglected area nowadays - but then a cure would be in reach, and how much money would that ultimately cost the industry in unsold symptom relievers?

Similarly, the health insurance industry is not going to want to pay for long-term antibiotic treatment because advanced neuroborreliosis needs years of expensive treatment and the results are often still meagre.

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Back to trials with antibiotics for ALS. There have been trials with Ceftriaxone and Minocycline. Such trials were always based on the premise that ALS is *not* caused by a bacterium. Instead, the antibiotics were administered to verify some assumed beneficial effect of the antibiotics, such as an anti-inflammatory effect or an effect on the elimination of glutamates.

Therefore, those trials were "sabotaged" from the start. The dosages used were too low to attain a spirocidal level in the central nervous system, often not even sufficient to attain minimum inhibitory concentration in the CNS. Treatment failure for neuroborreliosis has been amply documented at the dosages used in the Ceftriaxone trial for example. 2 grams/day is too little and 4 grams/day should have been used, as has been used previously to treat Lyme-ALS.⁹⁰ It may be essential to address coinfections as well.

Late stage Neuroborreliosis, like its close cousin Neurosyphilis, often needs years of antibiotic treatment. Dr. David Marz did not improve noticeably until after ten weeks of taking 4 grams of Ceftriaxone per day. 2 grams a day was ineffectual, and 4 grams/day did not result in noticeable improvement until ten weeks of treatment. Dr. Marz was eventually cured of his ALS, but it took more antibiotic treatment at dosages much higher than have ever been used in any antibiotic trials for ALS, and a treatment duration much longer than any treatment duration ever used in ALS trials.

The interesting thing is that antibiotic trials for ALS have in fact shown that in a statistically very significant number of cases (around 25%), the patient deteriorated significantly after taking antibiotics, compared to patients taking a placebo. This is an extremely important finding, as in previous trials, many different substances have been tried against ALS and never has any substance been identified that made ALS worse⁹¹. Except two substances - totally different substances, but both antibiotics: Minocycline and Ceftriaxone. Why would that be? The best explanation - and so far the only one - is the Jarisch-Herxheimer effect, observed in both neurosyphilis and neuroborreliosis. Patients get worse, sometimes much worse, before they get better. With ALS

⁹⁰ <http://www.unfictional.com/pdf/4g-day-ceftriaxone-als-bb.pdf>

⁹¹ <http://www.unfictional.com/pdf/als-report-2005.pdf>

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patients, getting worse may very well result in death or the need for artificial ventilation.

Hence the myth that "antibiotics have been tried and they failed".

We wrote more about the flawed antibiotic trials in the final chapter of this book, where we examine a trial with Minocycline.

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"It is unlikely that an infection such as Lyme disease would cause ALS"

"It would be unusual for the bacterium that is responsible for Lyme disease to lead to both upper and lower motor neuron signs and symptoms as well as the progressive motor weakness and paralysis that characterize ALS."

Sure it is unusual - that is why for each case of ALS, there are many more cases of Lyme! It is of course totally irrelevant how great the statistical likelihood is that neuroborreliosis causes ALS symptoms. There are roughly 30,000 yearly cases of Lyme reported to the CDC in the US, with an additional 60,000 cases reported as "probably Lyme". Experts estimate that the actual numbers may be three to ten times higher. **Sticking to the CDC data, it would mean that if Lyme only in about 5% of cases ultimately causes ALS symptoms, that all ALS cases would be attributable to Lyme disease.** And if Lyme disease only in 1% of cases results in ALS, then still about a quarter of all ALS cases would be caused by neuroborreliosis. 1% would be a good guesstimate, as the Halperin study saw 12.5% of ALS patients strongly improving on antibiotics (Ceftriaxone), and 12.5% rapidly deteriorating - both indications of a bacterial infection in the CNS.

Similarly, 25% of ALS patients receiving the antibiotic Minocycline deteriorated markedly. The oral antibiotic Minocycline, especially in the dosages used in the trial, is a less effective antibiotic than IV Ceftriaxone and therefore the spirochetes may merely have been "stirred up", trying to migrate to lesser tissue concentrations. This understandably wreaks havoc in the brain stems of ALS patients. Some deteriorations may be attributable to Jarisch-Herxheimer reactions. The study made it impossible to identify improvement in individuals and only presented a general trend.

Doctors often use statistics as an excuse not to test for troublesome-to-treat conditions. Specifically, they abuse a general lack of understanding of statistics in the average patient. How many percent exactly is this "unlikely", and can the good doctor back up his opinion with facts? As shown, 5% would account for all ALS cases. 1% would account for a quarter of all ALS cases. Is

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1% unlikely enough? Opinions are only as good as the hard data to back them up. Using the same insane argumentation of "Lyme rarely causes ALS", doctors routinely refuse to test the ALS-diagnosed for Lyme disease. In their pathological circular logic, they perpetuate their misconceptions.

Because if you don't test for Lyme, how will you find out whether a case of ALS is caused by it? How can you maintain accurate statistics of Lyme-ALS when you refuse to test? It's like claiming that criminal psychopaths don't commit any more murders than ordinary people, while refusing to test convicted criminals for psychopathy. Again it's just an opinion, and doctors with such unfounded opinions simply say: "I don't want to get into the can of worms also known as Lyme disease - let's just keep things nice and easy and not even go there".

Statistical likelihoods are irrelevant when the life of a person is at stake. The cost of a Lyme test is trivial compared to its potential to save the life of the patient, unlikely or not. And we say it again: All commonly used Lyme tests are so unreliable as to be worse than useless. If you do get tested, make sure you do an antibiotics-provoked test done by IGeneX or a similar lab that employs reliable tests - not the rubbish used by mainstream labs.

"A positive Lyme test means nothing much - only past exposure"

If a doctor has the audacity to dismiss a positive Lyme test in an ALS patient as "carrying little diagnostic weight" then it is time to fire that doctor for gross medical incompetence. Imagine pus coming out of your penis while testing positive for Gonorrhea. How would you react to a dismissal of the findings as having "little diagnostic weight"? This is comparable to ignoring a positive Lyme test in the ALS patient, because Lyme is known to be able to cause ALS as its sole symptom. The fact that the current dogma is that this is "rare" is irrelevant, because the next logical step would simply be aggressively treating the Lyme and observing whether the ALS is affected by that. Instead, indolence prevails and positive test outcomes are ignored.

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Studies finding a Lyme - ALS connection dismiss their own findings

Sceptics may claim that the Halperin paper is an anomaly. That somehow those positive Lyme tests were falsely positive. And that somehow those of the control group were not. Or that due to an inexplicable twist of fate, the patients enrolled in the study happened to defy mathematical laws and "coincidentally" had been infected with Lyme disease. Well, for those sceptics there are more studies, such as:

“Lyme disease serology in amyotrophic lateral sclerosis” by Qureshi, et al., Muscle & Nerve, October 2009, Volume 40, Issue 4, pages 626-628.

As we have estimated before, if Lyme infection and ALS would have been unrelated, we would expect to see 0.086 of ALS patients testing positive for Lyme disease, even when we assume Lyme disease to be five times more prevalent as statistics say. **However, this study found 5.8% of ALS patients positive for Lyme, which is 67 times higher.** But, without giving much other reason but a "belief", the notion that Lyme disease could be responsible for at least some cases of ALS under examination is summarily dismissed.

Whatever those who conducted the study may themselves conclude, the fact remains that their study can be interpreted to document that people with Lyme disease have a much higher chance to develop ALS than the average person. To dismiss this vital piece of medical detective work seems strange. "This gun is not smoking", they say - holding their noses to the acrid smell of burnt gunpowder.

The Lyme - ALS connection remains ignored. *Then how can it be explained that studies show that people with ALS have been Lyme-infected two to three orders of magnitude more than the general population?*

Delaying the answer to this vital question can mean the difference between life and death for countless ALS patients now and in the future. Ignoring it is a willfull act of sabotage against the most basic human right of the ALS-afflicted: Their right to live.

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Most ALS patient are Lyme-positive when tested properly

We had a detailed look at the Halperin paper, a limited study of just two dozen patients, using only a limited spectrum of Lyme tests to determine whether they've even been infected with *Borrelia* - but what happens when you start looking more thoroughly for Lyme disease in ALS patients?

Dr. Martin Atkinson-Barr, CPhys PhD studied at Cambridge and did microbiological experiments at Rhone-Poulenc.

Dr. Atkinson-Barr asked thirty random people with ALS to get tested for Lyme disease. **Every single one of those ALS patients tested Lyme positive.**

We reproduce Dr. Atkinson-Barr's own postings on the USENET newsgroup sci.med.diseases.lyme (emphasis ours):

Posting #1 on sci.med.diseases.lyme:

From: Martin Atkinson-Barr (mcmab@peoplepc.com)
Subject: Successful treatment of late-stage ALS

I am pleased to announce the following:

Since April 1999, 150 ALS patients have been tested for Lyme disease with a panoply of tests - incl Western Blot, LUAT, PCR. Not one patient has been found to be negative across all tests. Many have been shown to be PCR positive.

The prognosis and disease development of these patients is entirely consistent with ALS.

Treatment with oral antibiotic therapy has shown mixed results. ***In particular the use of conventional antibiotics (esp. doxycycline) has been associated with deterioration of ALS patients.*** In one case the

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patient rapidly succumbed. In earlier stage ALS patients there is some evidence for improvement, with restoration of speech in two patients and some reported easier swallowing, when treated with oral metronidazole or tinidazole.

The reactivity of ALS patients to Lyme tests has been previously reported.

In the course of the past 9 weeks a patient (body weight 125lbs, 66 years of age) with advanced ALS symptoms has been treated aggressively with IV metronidazole + conventional antibiotics (Biaxin initially) at doses of 500mg tid metronidazole IV and 500mg Biaxin bid orally. The diagnosis of ALS was made at the Mayo clinic. The patient was admitted in respiratory failure with tongue fasciculations, weakness in the right arm. The immediate prognosis was poor and the attending physician expected the patient to expire within 24 hours. The patient was ventilated. In the course of 7 weeks of the above therapy the patient has improved and is now ambulatory and off of ventilation using only occasional nasal oxygen. On the IV therapy the tongue fasciculations disappeared.

After 7 weeks the patient was taken off of the IV meds and treated with only oral tetracycline (500mg qid). On this treatment the tongue fasciculations returned. The IV therapy was reinstated with IV Rocephin replacing the oral Biaxin and the tongue fasciculations ceased. ***The patient continues to improve on a daily basis.***

Tentative conclusions:

The etiologic agent of ALS is *Borrelia burgdorferi*.

Effective treatment of late stage ALS is possible with aggressive antibiotic therapy that must include metronidazole.

Other researchers have recently reported success in treating early stage ALS with antibiotic therapy.

ALS patients should not be treated with simple "textbook" antibiotic therapy which does not include a nitroimidazole.

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This therapy should be considered experimental at this stage.

Regards

Martin Atkinson-Barr PhD

Posting #2 on sci.med.diseases.lyme (May 21, 2001):

Let us be clear how I come up with the figure of 150.

When I published my work on metronidazole in Lyme around 20 patients contacted me and said they had Lyme AND ALS.

Once I had decided there may be a connection between the diseases *I encouraged every ALS patient I came across, and a few physicians to test for Lyme. All told that is about 30. This is the most important group for they were unselected. There were NO negatives in this group and for the most part they were either IgG or IgM Western Blot positive*, mostly to CDC criteria (which is over strict and ignores the 39kDa line)

Dr Nick Harris has been sending on to me all of the ALS patients that have contacted him who were positive. These patients I questioned closely to determine if they had a clinical picture of ALS. All did. There have been around 20.

When my website was up (thanks to the ISP it was lost) around another 80 ALS patients contacted me with their results, all positive. No ALS patient has ever been in contact with me who is negative. There must be some.

If we were to take 150 Lyme patients we would be VERY surprised to have 150 positives, more like 100. However many of the above were pre-selected (why would you contact me if you were diagnosed ALS but Lyme negative, I would be snake oil.).

BUT, if the diseases were independent, we would expect a MAXIMUM of 100 cases in the whole of the US, so 150 becomes a significant

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number.

Now, those patients who were diagnosed ALS and tested Lyme positive carried on to develop the sequelae of ALS. Last Thursday we lost Dean Chioles, note that he was diagnosed with Lyme in 1998, before I published anything on Lyme.

We should also remember: Brian Pierson and Katherine Crowe who we also lost, both Lyme positive. Brian was 43 years old or so.

Now there have been 5 papers that describe clinically diagnosed ALS patients with Lyme reactive serologies (including the Halperin paper which finds a statistically significant number of Bb positive patients with ALS) and one letter by Mandell, Steere et. al. NEJM 1989;320:255-6 which found NO Bb antibodies in any ALS patients.

The responsibility is now with Mandell, Steere et. al. to come forward and explain how they can find NO Lyme positive ALS patients when I can find them so easily and even ALS patients themselves can see the connection.

How about it Dr Steere, this is an open venue? Perhaps one of the Yale workers will read this and we can enter into a debate that will explain why this connection has been stalled for 12 years.

With best regards to all.

Dr Martin Atkinson-Barr

If Dr. Martin Atkinson-Barr is telling the truth and if there have not been made major mistakes that led to the false-positive testing (for Lyme) of the majority of those 150 people with ALS, then there can be only one conclusion:

Lyme and ALS are causally related.

Are Lyme-infected ticks thousands of times more likely to bite people with

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ALS, or has neuroborreliosis a hand in ALS?

Of course Lyme bacteria would be the cause of ALS, instead of ALS the cause of getting infected with those bacteria.

The doctors that make a comfortable living being "ALS experts" claim that even when ALS sufferers have been infected with Lyme before they developed ALS, that this is by no means an indication that Lyme has anything to do with ALS.

"It could be just a coincidence", they say. When confronted with the infinitesimally small likelihood that such a high percentage of ALS patients would be Lyme positive, they'll dig in their heels and proclaim that there must be some "coincidental, inconsequential factor" at work, something that makes people infected with *Borrelia* bacteria more susceptible to ALS - but that "something" is *not* the actual bacteria eating away at people's central nervous system. Neither is it an inflammatory immune response to those same bacteria living in the CNS. Because they do not *believe* that. That would namely do away with their entire medical specialism in one fell swoop - they would have to get educated on *real* medical science instead of receiving yet another grant to spend a year or so, speculating on a genetic risk factor against some environmental toxin or other.

Instead, they say that instead of the *Borrelia* spirochetes causing ALS, it could perhaps be some mistake in the immune system, which for some reason decides to destroy the brain after a patient did get infected with Lyme disease bacteria. They say it's not *Borrelia* bacteria eating away at the neurons. It's not an inflammatory immune response to those bacteria. No, it is some elusive error in the genes of the person with ALS that wreaks all the havoc. In spite of all those positive Lyme tests. "Auto-immunity" of some sort. Perhaps. Say the "experts".

What people with ALS should require of their doctors

Get tested for Lyme disease

It is currently very rare for doctors to be willing to test ALS patients for Lyme disease. They will usually not bother trying to exclude neuroborreliosis as a diagnosis, because they are ignorant of the fact that Lyme can manifest itself mainly by ALS. Even two-thirds of liberal-minded clinician-scientists, willing to participate in investigating a Lyme-ALS link, make an ALS diagnosis without ever testing testing for Lyme, except when the patient explicitly asks for it or when the clinical picture shows more symptoms than "just ALS".⁹²

Note how the authors of the article entitled "Investigating a bug (Lyme disease) and a drug (Iplex) on behalf of people with ALS" dismiss all available published medical case studies such as the Halperin paper as useless garbage when they say: "The evidence for their proposed link between Lyme disease and ALS comes from poorly documented case reports or small series in which there is either **insufficient evidence provided to establish a diagnosis of ALS in the first place**, or **inadequate follow-up to establish a true improvement in the ALS** coincident with Lyme treatment". They wrap that argument in strong allegations of malfeasance on the part of any doctor proposing to Lyme-test ALS patients or even treat them with antibiotics when they turn out positive, because they imply there are nefarious financial objectives at play.

However, the anonymous author of these unsubstantiated incriminations - identifying himself only as "The ALS untangled group" is being self-contradictory. He claims that *either*: A sizeable percentage of ALS diagnoses are bogus diagnoses, that those people did not have ALS at all, but they have something else such as Lyme disease, and that even if their patients really had ALS, that he does not *believe* that those ALS patients really improved permanently. This in spite of a plethora of people who are still with us, because they insisted on getting tested for Lyme and underwent successful treatment. Dr. Marz is an excellent example, but this book contains more.

⁹² <http://www.unfictional.com/pdf/ALS-experts-not-testing-for-lyme.pdf>

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Who is the "ALS untangled group"? It is not easy to find out. The owners of alsuntangled.com pay extra money to their registrar for anonymous registration. They don't have an "about" page or an email address. It is clear that they are not interested in the health of the ALS-diagnosed, otherwise they would not discourage testing for a treatable disease that has been proven to sometimes cause motor neuron death as its sole symptom - yielding an ALS diagnosis. They use manipulative language such as "The Curious Case of Dr. Martz", implying that something must be wrong with his version of events. That his case is an enigma, a deep mystery, as if misdiagnoses of Lyme for ALS aren't perfectly common, both anecdotally as well as in medical literature.

ALS Untangled appears to have a simple agenda: Convincing ALS patients and their families that misdiagnosis of ALS is extremely rare. That an alternate diagnosis should not be sought, especially not one of Lyme neuroborreliosis. The site is rumored to be run by ALS experts. It is of course much easier for a doctor to give a patient a death sentence than to take responsibility for the complex and lengthy task of treating neuro-Lyme - not to mention the fact that according to the CDC, chronic Lyme doesn't exist - which makes anyone treating it long-term a quack in their eyes and eligible for disciplinary action.

Not testing for Lyme makes ALS specialists guilty of serious medical malpractice, because there are sufficient published cases where a patient had no "typical Lyme" symptoms and exclusively suffered from motor neuron disease - classic ALS - but it was in fact Lyme disease causing that ALS and they recovered on antibiotics.

So if a doctor does not routinely tests for Lyme before making the ALS diagnosis, the doctor is clearly incompetent to make the diagnosis in the first place and guilty of criminal negligence as well of medical malpractice when the patient dies and *Borrelia* bacteria are found to be coiled up in the neurons of the deceased. Taking this risk means that the doctor is either reckless and lazy or fundamentally ignorant of Lyme being able to be clinically indistinguishable from ALS, and in both cases that doctor is

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unsuitable to make the diagnosis.

A patient should better not ask for Lyme testing but rather wait and see whether the doctor thinks of that himself, because a doctor who's medical knowledge is outdated has no excuse making ALS diagnoses in the first place. **The best way to uncover incompetent neurologists is by allowing them to make a diagnosis, but as soon as they omit a Lyme test at an early stage, to point out their obvious unsuitability to arrive at a reliable diagnosis. And then fire the doctor.**

Insist on reliable Lyme tests

If your doctor does order tests and they turn out negative, you should first ensure that those tests are reliable (Igenex is a recommended lab), and that sufficient tests of different types have been done., such as a spinal tap PCR, a Western Blot, a Lyme Urine Antigen Test and an immunofluorescence test.

If sufficiently many, reliable tests have been performed and were all negative, they have to be re-done, after having taken antibiotics for a sufficiently long time (at least a week). It would be preferable to do this in the first place, but in case of severe bulbar ALS this may result in severe deterioration if not done properly. Those antibiotics have to be of the kind that penetrate the blood-brain barrier. Suitable antibiotics are Doxycycline and Minocycline plus Metronidazole or Tinidazole. Their dosage should be relatively low when the ALS has bulbar aspects, because the Jarisch-Herxheimer reaction in those patients has been shown to be possibly fatal in the Halperin paper.

If the repeated Lyme tests are then still negative, but if the patient experienced either a significant improvement or a significant deterioration (permanent or temporary) in symptoms, it has to be assumed that this is due to the antibiotics, and that the ALS is of bacterial origin. If the doctor does not propose immediate continued antibiotic treatment in that case, the doctor is clearly not competent to properly diagnose or treat the patient and should be replaced. Ask for copies of the patient's dossier and waste no time finding a doctor or even private clinic - domestic or abroad - willing to

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immediately initiate antibiotic treatment. If no antibiotic treatment can be obtained through the medical system, suitable antibiotics should be sourced through alternative means, as inaction will certainly lead to rapid deterioration and the inevitable death of the patient. The more severe the neurological damage, the more entrenched the bacteria are and the greater the likelihood of treatment failure or even an accelerated demise due to the Jarisch-Herxheimer reaction.

In case of Lyme, insist on proper antibiotic treatment

It is of the utmost importance to receive proper antibiotic treatment. Dr. Atkinson-Barr says that without an imidazole added to the antibiotic, treatment failure or even exacerbated symptoms are to be expected. Imidazoles such as Fasigyn (Tinidazole) paralyze bacterial flagellae (rotating hairs they use for propulsion and tissue/cell penetration) and without it, the spirochetes will attempt to move to tissue areas with a lower antibiotic concentration, causing a lot of extra damage as a result when they "drill" through neurons and Myelin. Imidazoles, when combined with antibiotics, greatly increase the likelihood for opportunistic fungal infection of the mucous membranes, so care should be taken to ensure meticulous oral hygiene and it is greatly recommended to add an antifungal medicine, as ALS patients have lowered defenses against such infections.

Treatment for neuroborreliosis should always be open-ended. Treatment only stops when all symptoms are gone, and when they return, treatment should resume immediately.

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Antibiotic treatment of Lyme-induced ALS

Atkinson-Barr urges an imidazole to be added

First a stern word of warning. We know from medical literature as well as anecdotal reports that **ALS patients with bulbar ALS may quickly die as a result of the wrong antibiotic treatment.** This is what Dr. Atkinson-Barr has to say about the topic:

Treatment with oral antibiotic therapy has shown mixed results. In particular the use of conventional antibiotics (esp. doxycycline) has been associated with deterioration of ALS patients. In one case the patient rapidly succumbed. In earlier stage ALS patients there is some evidence for improvement, with restoration of speech in two patients and some reported easier swallowing, when treated with oral metronidazole or tinidazole.

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In the course of the past 9 weeks a patient (body weight 125lbs, 66 years of age) with advanced ALS symptoms has been treated aggressively with IV metronidazole + conventional antibiotics (Biaxin initially) at doses of 500mg tid metronidazole IV and 500mg Biaxin bid orally. The diagnosis of ALS was made at the Mayo clinic.

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ALS patients should not be treated with simple "textbook" antibiotic therapy which does not include a nitroimidazole.

This therapy should be considered experimental at this stage.

We think that the reason for the rapid deterioration in ALS patients with significant lower motor neuron damage when treated with conventional antibiotic treatment (without the use of an imidazole such as Flagyl) is very likely not a Jarish-Herxheimer reaction or the result of increased local inflammation due to increased antibody production. Because Metronidazole impairs the bacterial flagellum (its brand name is "Flagyl" for that reason), preventing them from moving. This strongly suggests that the physical movement of the spirochetes is responsible for the deterioration, as the extra damage sustained to the remaining lower motor neurons proves fatal. Spirochetes are known to migrate or encyst themselves into protein envelopes when they sense cell wall damage to themselves. Both involves movements. Since *Borrelia* spirochetes are relatively large bacteria, a great deal of damage to neuronal tissue is the inevitable result.

There have also been trials done where ALS patients deteriorated on certain antibiotics (Minocycline):

Paul H Gordon MD, et. al.

Efficacy of minocycline in patients with Amyotrophic Lateral Sclerosis: A phase

III randomised trial

The Lancet, Nov. 2007

This trial found a significant, dose-unrelated deterioration in ALS patients on dosages of up to 400 mg/day of Minocycline. **This is a very exciting outcome, because it supplies strong evidence for the hypothesis that ALS has a bacterial etiology (underlying cause).** Minocycline has a neuroprotective effect as well as an antibiotic effect, so any deterioration should most likely be attributed to a Jarish-Herxheimer effect (neurotoxins pset free during a dieoff) or an inflammatory immune reaction to lysed spirochete fragments or, more likely, physical actions of the highly motile bacteria trying to move to lower tissue levels of antibiotics, causing new

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damage. This damage could be very substantial, because *Borrelia* has been shown in animal models to be directly neurotoxic - for some reason it kills the neurons it touches. **If ALS's cause would not have been bacterial in origin, we would expect to see neither a positive nor a negative effect of Minocycline.** Combination therapy with an imidazole is mandatory says Dr. Martin Atkinson-Barr - or deterioration of the patient and treatment failure is a result. Again, it is unfortunate that the trial did not take this into account.

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Why Allan Steere disputes the Lyme - ALS connection

Alan Steere is the discoverer of Lyme disease, but unfortunately he is badly qualified to be "the expert" on neuroborreliosis because he is neither an infectious disease specialist nor a neurologist. Rheumatologist Dr. Steere initially warned for Lyme's insidious nature but when he saw it becoming a "fashion diagnosis", he dedicated most of the remainder of his professional life to a personal crusade against the very concept of chronic Lyme neuroborreliosis, and he has been known to do "counter-studies" that always discredit studies that find links between Lyme and neurological syndromes of unknown etiology such as ALS. Of course one can't prove a negative, and his studies are therefore of no value with respect to either proving or disproving a link between Lyme and ALS, but he leaves no opportunity unused to pull rank and call chronic Lyme patients hypochondriacs and malingerers and Lyme-literate medical doctors quacks.

Dr. Steere has been a driving force to get LLMD's (Lyme-literate medical doctors) out of business, as in their license to practice revoked and they themselves sued for malpractice - as happened to Dr. Burrascano. Dr. Burrascano had this to say:

I am approaching a critical time in my battle with the New York State Office of Professional Medical Conduct (OPMC). They have notified me that they intend to fully prosecute me in a formal administrative hearing. Statistically, based on past experiences, there is a 98% chance that I will have my license to practice medicine revoked, suspended, or restricted (i.e.: cannot treat Lyme patients), and face a possible fine. This will be scheduled within the next few weeks. Obviously, the goal is to halt this process NOW, before a formal hearing commences. Not only is such a hearing potentially damaging, the defense can cost upward of \$100,000.00, and is not covered by insurance!

In addition, I just received a copy of a letter sent from the OPMC to Senator Moynihan stating that, in so many words, the feelings of my supporters and their letters are being ignored by OPMC, because OPMC follows the Lyme guidelines of the ALDF (Steere et al) and the recommendations of the Medical Letter (Lyme recommendations written by Dr. Steere). In other words, the reality and truth of Lyme, supported by valid, peer reviewed

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publications and our experience with thousands of patients counts for nothing. OPMC is blindly listening to the ALDF physicians and NOT critically reviewing their recommendations, which DO NOT stand up to scientific scrutiny.

What can we do now? Now we have to fight even harder! We need to have our message VERY STRONGLY conveyed directly to New York Governor Pataki. He must immediately halt all actions against all Lyme MDs and initiate an independent investigation of OPMC and whom they are relying upon for their misinformation about Lyme.

Another very important thing all of us must do, patients and doctors alike, is to join the organization known as FAIM, the Foundation for the Advancement of Innovative Medicine. They are a powerful and successful advocacy and lobbying group, and because they support my views, and know that there is power in numbers, they welcome the Lyme community to their organization. I am formally requesting that all my supporters join FAIM. Look them up or call them toll free at 1-877-634-3246.

We also must get as much media attention as possible, not only for us to tell our story, but also directed toward Governor Pataki so he will feel additional pressure to act.

If my ability to treat Lyme Patients is restricted or if I am fined for what I do, this will have a very large negative effect on the current and future health of not only my own patients, but by ripple effect, it will affect all Lyme patients worldwide.

Call and e-mail all your friends, family and other contacts as soon as possible and spread this message. We need a gigantic call to action now!

Thanks to all my friends!

J.J. Burrascano Jr. M.D. January 8, 2000.

On May 11, 2008 Dr. Steere attacked Lyme patients and the doctors taking them seriously in a speech.⁹³

T. L. Kittle's opinion on Steere's crusade against diagnosis and treatment for neurological Lyme disease:

⁹³ <http://www.unfictional.com/pdf/steere-attacks-chronic-lyme-speech.pdf>

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Lyme Lies: Allen Steere's horrifying comments regarding Lyme disease in last week's commencement address.

LOS ANGELES, May 24 /PRNewswire/ -- As some people are already aware, there's a debate going on regarding Lyme disease (it's actually more like a war) regarding who should be treated for Lyme disease and how long that treatment should be. (Lyme disease is a bacterial infection caused by tick bites that can lead to debilitating symptoms, including severe neurological problems.)

Allen Steere, as a member of the IDSA (Infectious Diseases Society of America) and having spent decades studying the disease, is well regarded by many as an "expert" on Lyme disease. Therefore it is of utmost importance that he presents the facts on Lyme disease as they are currently known truthfully to the unknowing public -- especially given his stature in the medical community.

The main problem with this speech and his argument about Lyme Disease, is that he's basing the entire argument on whether or not someone who is ill with Lyme symptoms should be treated with antibiotic therapy and for how long on diagnostic testing -- presenting diagnostic testing as if these tests were 100% reliable, which is false information -- horrifying by someone who is supposed to be honoring the science.

"Diagnostic tests based on scientific studies fail to show evidence of Lyme disease in most of these patients," stated Allen Steere.

It's only true that these patients did indeed 'for sure' not have Lyme disease if it's true that diagnostic tests are 100% accurate-when in fact, Lyme tests have been scientifically proven to produce false negatives.

Therefore, Allen Steere misrepresented the facts about Lyme disease to the Ohio Wesleyan University 2008 graduates. Since diagnostic testing for Lyme disease is not 100% reliable, then this means some of these patients with negative results might have actually had Lyme disease, making antibiotic therapy appropriate.

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Since he states he holds scientific facts so valuably, then why is he basing his argument on whether or not someone should be treated with antibiotic therapy for Lyme disease on a falsehood?

It is irresponsible and an insult to scientific evidence to misrepresent the evidence by leaving out the false negative rate for Lyme tests -- especially by someone who claims to be standing by the science without informing the audience that the science itself has proven not to be 100% reliable -- that it is a scientific proven fact that Lyme tests are not always accurate, making it absolutely necessary to treat some people with Lyme disease who have negative diagnostic testing-people with no laboratory evidence of the disease -- people on paper who 'scientifically' don't have it.

It seems he's throwing his weight around, using the power of the IDSA and the medical institutions he's connected to, to misrepresent the decisions behind when to treat people with antibiotic therapy for Lyme disease and for how long by omitting a discussion of the false negatives in this speech.

He's right that antibiotics can cause harm, and he's right that there's always going to be patients who get better based on the placebo effect alone -- this is **not** specific to Lyme disease --

However, there's another side to "do no harm": the harm caused when something is not given to someone who needs it (ie, not feeding one's children). Therefore, it should be the patient's decision in collaboration with their physician on how they want to proceed medically when their diagnostic tests have come back negative.

It's a shame that Steere didn't use this speech as an opportunity to talk in depth regarding how many unknowns there are in medicine--therefore, in order to care for people adequately, it is essential to listen to patient's clinical information until science has all the answers--since it is impossible to tell the 'faker' from someone who is genuinely recovering, then the medical professional has to make a choice regarding how they want to practice medicine--based on test results that have been scientifically confirmed to be faulty, or to be a physician who practices medicine based on clinical

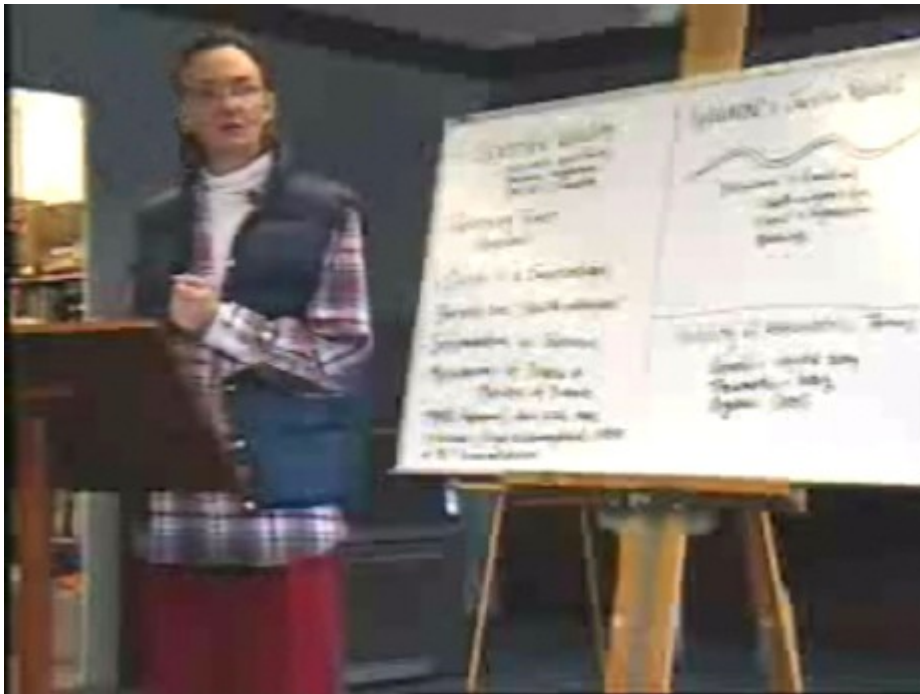
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information and human compassion?

Since the tests are not 100% accurate, then test results are obviously not the only way someone should be diagnosed and determined 'scientifically' to have Lyme disease -- As Steere states in his speech, "If you ignore scientific reality, if you twist it, if you wish for a particular answer, you will miss Mars and drift in space." The message behind this statement is absolutely correct-it is absolutely vital to honor the science-which includes when the science is saying it doesn't have a definitive answer, and therefore it is impossible to make definitive conclusions regarding all aspects of the disease.

To hold diagnostic testing up as the only marker for determining whether or not someone has Lyme disease is misrepresentation of the disease.

It's amazing to think of how many people are suffering unnecessarily based on the omission of this one key scientific fact-especially since he has a ethical (and legal?) responsibility to share the whole truth.



Kathleen Dickson

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The feud with Lyme patient advocates

Why is Dr. Steere so intolerant of those doctors who point out the wealth of medical evidence for late-stage neurological Lyme disease? **Most likely, Steere is so vindictive of Lyme patients because Lyme-activists and a Lyme support group leader prevented him from becoming a very wealthy man.** He holds crucial OSP-A related patents to the Lymerix vaccine, taken off the market due to serious side effects. Instrumental in that was a main patient advocate for chronic Lyme disease and critic of CDC-recommended Lyme tests, Kathleen Dickson. Mrs. Dickson is an analytical chemist who worked for Pfizer. She described herself as a high-functioning autistic and her Lyme website actionlyme.org testifies to that, featuring a treasure trove of her Lyme-related detective work, sadly presented in the most inaccessible way imaginable and interspersed with unrelated, but highly inflammatory material. It would be wrong to call Mrs. Dickson a "loon" or "wacko conspiracy theorist", as she has been by her anonymous detractors. Evidence of the fact that Mrs. Dickson thinks very analytically and has studied the intricate details of the Lyme controversy is in her testimony, containing a detailed explanation why Dr. Steere had a strong financial interest in enforcing Lyme-tests with a very large percentage of false negatives: It would greatly minimize the quantity and severity of the vaccine's reported side effects, thus guaranteeing its FDA approval, which would have made Dr. Steere a multimillionaire, through Lyme-related patents he owned. Mr. Dicksons testimony can be read here:⁹⁴

After Steere's humiliating defeat on the vaccine front by Lyme activists, he demonstrably bears a major grudge against them because he won't let an opportunity pass to discredit them personally or their claims by any means possible. Once he had a NYT reporter write that Lyme patients threatened to kill him. Without offering the slightest substantiation for this claim. Dr. Steere is still alive, whilst many Lyme patients died as a direct consequence of him sabotaging both diagnosis and treatment for this disease.

⁹⁴ <http://www.unfictional.com/pdf/dickson-lymerix-testimony.pdf>

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Mrs. Dickson's take on Dr. Steere:

What Allen Steere did in Europe to falsify the diagnostic standard for Dearborn

Allen Steere is the author of both CDC standards, the 1990 standard and the 1994 Dearborn standard. For the fake standard - the current CDC Dearborn standard - Allen Steere went to Europe alone in 1992 with bogus high-passage strains and recombinant OspA-B with no lipid attached and therefore little immunogenicity (means, "produces antibodies") to leave OspA and B out of the standard. Osp A in the diagnostic test, when the antigen is also the vaccine, invalidates the test. One never tests for vaccine efficacy with the same antigen as the vaccine. **Steere also falsely claimed that his pool of Dressler/Steere neurologic Lyme patients must have proteins or cells in the CSF, when it was known that at least half of neurologic Lyme victims do not have antibodies in their CSF or is an "aseptic meningitis."** So, **all of Dressler/Steere was deliberate scientific fraud.** Steere had previously proposed that we perform sequential Western Blots to diagnose Lyme.

The new but false term "Lyme Disease," is actually the diagnostic test re-invented by CDC "officer" Allen Steere in Europe in 1992-1993. **Steere's deliberate falsification of the blood test for "Lyme Disease" was meant to exclude all neurologic Lyme cases and 85% of all cases.** The former formal name of "Lyme Disease" was "Lyme Borreliosis."

At the present time, therefore, there are two types of diseases, 1) "Lyme Disease," or Steere's alleged autoimmune arthritis in a knee - which some individuals claim needs no treatment - and then there's 2) **"Seronegative Lyme", which are all the neurologic cases, and which the same individuals also claim needs no treatment.** The new, 1994, "Lyme Disease" has been linked by Allen Steere to a genetic predisposition to having a reactive (allergic) arthritis to OspA (a palmitoyl 3-cys triacyl peptide like the main HIV antigens, the kind found in mycoplasma and mycobacteria, and E. coli).

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Why the immune system can't kill the *Borrelia* in the brain

When a *Borrelia* bacterium enters the blood, it is able to out-swim the immune-system's T-cells by a factor of hundred and quickly disappear inside tissues.⁹⁵

Therefore, not many antibodies against the bacteria will be produced. *Borrelia* is able to penetrate blood vessels and thereby pass the blood-brain barrier. As soon as it has reached the brain, it changes the molecular makeup of its cell wall, the so-called Outer Surface Proteins (OSP's). Antibodies are specific for bacterial OSP's, so the antibodies that the immune system produced when the bacterium was still not in the brain won't work any more.

Dr. Andrew Pachner infected mice with *Borrelia burgdorferi* and later extracted the bacteria from the blood and from the brains of the infected mice.⁹⁶ What he found was basically that the bacteria in the brain changed: They now expressed a new set of genes. The result was bacteria so different from what he started with, that the antibodies from the peripheral blood could no longer detect the bacteria isolated from the brain.

This is bad news as the CNS is isolated from the rest of the body. If the Lyme spirochete can adapt to the human brain and circumvent the immune system, it is less likely to be inhibited by our natural immune defenses. Further studies by Pachner in primates using PCR suggested persistent infection post-antibiotic treatment. **This is more bad news as it suggests that the CNS of primates is an isolated and protected incubator for *Borrelia* bacteria.**

These sources are written by Tom Grier, M.S. and are very accessible for the layperson but these are undisputed facts amongst microbiologists and can be verified in many publications.

Antibodies are small enough to pass the blood-brain barrier, but as we said - those antibodies are now useless, because the bacteria have "changed coats".

⁹⁵ <http://www.unfictional.com/pdf/infection-mechanics-tests-unreliable.pdf>

⁹⁶ <http://www.unfictional.com/pdf/bb-osp-variation-in-the-cns.pdf>

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"No problem", you may think. "The immune system will detect them in the brain and produce new antibodies". That would be nice - except that there is no full-fledged immune system in the brain. The brain is an immune-privileged site. T-cells do not circulate in the brain - they are not able to pass the blood-brain barrier. So that's it - the bacteria are in the brain and they are there to stay.

And it gets worse. The spirochetes have evolved even more cunning disguises: OSP cloaking. They coat their cell walls with proteins that are near-identical to proteins found in the central nervous system, such as myelin, the insulating material around nerves and neurons. When the occasional brain-based spirochete wanders back into a non-immune-privileged site, the immune system will at best do nothing to protect the CNS, and at worst produce antibodies against the bacterium myelin-like cell wall proteins - attacking the body's myelin. This gives rise to the erroneous suspicion of the ALS being caused by a malfunctioning immune system, "auto-immunity", while the direct cause is a response to living bacteria. Especially untreated Lyme patients commonly have a damaged parenchyma (blood-brain barrier), so it is also possible that immune cells circulate in the CNS and trigger antibody production that will attack both bacteria and the brain's own myelin.

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Why neuroborreliosis does not show up on any Lyme tests

Mrs. Dickson is correct in saying that the great majority of neurological Lyme cases test seronegative. The reasons for the false-negativity of not just antibody tests but also PCR tests of the spinal fluid are the following:

Antibody tests

Antibody tests can only work when there are antibodies to detect. It may come as a big surprise for people without much medical knowledge but it is a fact nevertheless: There is no immune system in the brain. The brain is a so-called "immune-privileged site". This is what Wikipedia says about those sites (abridged, emphasis ours):

Immune privilege is a term used to describe certain sites in the body which are able to tolerate the introduction of an antigen without eliciting an inflammatory immune response.

Known immunologically privileged sites include the brain.

That means that if someone would inject *Borrelia* bacteria into your brain, you would never be positive on a Lyme test because the bacteria did not first enter the body through the skin and bloodstream, eliciting an immune response, causing IgG antibodies to linger for years in many cases.

But what happens when you get infected with Lyme disease is that the bacteria spread throughout the body, but get eliminated by the immune system in all tissues with normal immunity. Immune privileged sites are not able to rid themselves of spirochetes however, because they lack an immune response and circulating antibodies are usually insufficient to fully eradicate every single spirochete in brain, eye, spinal chord or joint. **Especially not because the bacteria change their outer surface proteins as soon as they arrive in the brain - making the antibodies that the immune system produced when it detected the infection ineffective.**

So, after the bacteria are gone from most of the body, there is no more production of new antibodies against them, while the infection rages on in

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the brain and spinal chord. And after some time has passed, there are no more antibodies left to detect because without a steady new production of them, they'll eventually degrade and disappear.

PCR tests

But what about "more advanced" tests you may ask. What about PCR? PCR stands for Polymerase Chain Reaction. A PCR test can detect a single piece of bacterial DNA. It's like testing for the fingerprint of a known burglar. Burglars can wear gloves though. How would a neurological bacterial infection be able to defeat a sophisticated and ultra-sensitive test as the PCR?

For *Borrelia* bacteria, nothing is easier than that. Hiding their tracks goes all by itself - it comes naturally to them. *Borrelia* is what we call a microaerophilic organism. It needs oxygen to survive, but it is harmed by too much oxygen - such as found in the bloodstream. From Wikipedia (abridged, emphasis ours):

A microaerophile is a microorganism that requires oxygen to survive, but requires environments containing lower levels of oxygen than are present in the atmosphere (~20% concentration).

Examples include *Borrelia burgdorferi*, a species of spirochaete bacteria that causes Lyme disease in humans.

It is known from laboratory experiments that *Borrelia* very rapidly leaves the bloodstream after infection.

"At least in the dog, blood is an unreliable tissue to demonstrate *B. burgdorferi* infection." ⁹⁷

The Norwegian microbiologist Øystein Brorson (who uses a wheelchair due to Lyme disease) demonstrated that *Borrelia* spirochetes remain detectable less than a minute in cerebrospinal fluid - after that they encyst themselves into a thick protein "bubble" rendering null and void any attempts to detect their DNA with a PCR test.

⁹⁷ <http://www.unfictional.com/pdf/persistent-seronegative-lyme.pdf>

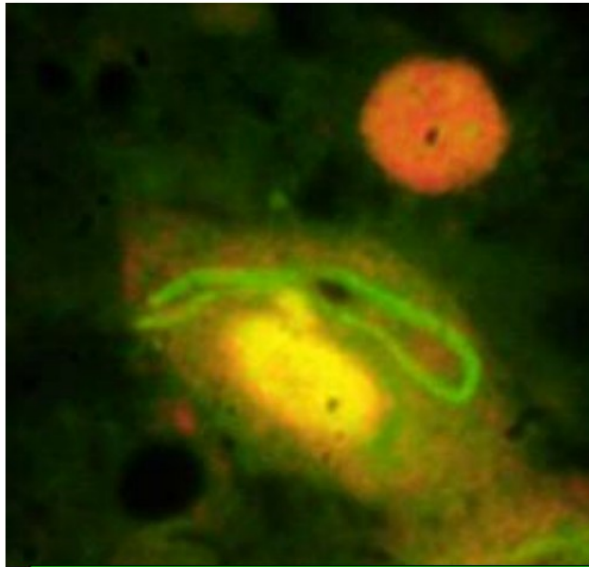
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Dr. Øystein Brorson

Moreover, spirochetes have no business swimming in spinal fluid in the first place. They lodge themselves where there is food - and myelin is *Borrelia's* favorite dish. On top of that, *Borrelia* makes itself unreachable for circulating antibodies beyond the blood-brain barrier as well as undetectable with any kind of PCR test using cerebrospinal fluid by ***coiling itself up inside neurons***, as can be seen on images supplied by Dr. Alan B. MacDonald:

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Infectious Agent Inside of the Nerve cell

Inside of the cell Infection



Borrelia spirochetes inside Hippocampal neurons in Alzheimer's disease

Note how these images suggest that Alzheimer's can be caused by Lyme. There is in fact overwhelming evidence of Alzheimer's and MS being caused by Lyme disease - 100% of twelve deceased MS patients had Borrelia in their brains, as found by Dr. Øystein Brorson, who did not shy away from using an electron microscope to find Borrelia bacteria in the all brains of those deceased MS patients. **It is not healthy to have spirochetes eating away at your brain.**

Microbiologist Judith Miklossy did a study on deceased Alzheimer patients' brains and she found 25% infected with Borrelia bacteria and the remaining 75% was infected with oral Treponema's, also spirochetes. She concludes that Alzheimer is a neurospirochetosis. She still believes this. We verified her conclusion by contacting her. We will write two more books: One about the cause for MS being Lyme disease, and another one on the cause of Alzheimer's being a neurospirochetosis (Lyme or oral spirochetes).

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Dr. Alan B. MacDonald.

Does that make Lyme tests useless to test for neuroborreliosis? Yes! We know from published studies that the overwhelming majority of tests on people with neuroborreliosis are false-negative - explaining why 12.5% of ALS patients improved markedly, and 12.5% of ALS patients deteriorated rapidly on antibiotics in the Halperin study. Both are indicative of a bacterial brain infection causing the ALS. The latter is a sign of a Jarisch-Herxheimer reaction - the setting free of toxins as bacteria die. Additionally, the highly motile spirochetes would attempt to evade areas with high tissue levels of antibiotics and hence do a lot of extra damage to the brain stem.

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How can you have Lyme if you've never been bitten by a tick?

Lyme disease is generally thought to come from infected ticks and from infected ticks only. However, Borrelia bacteria commonly occur and were found by Bowen Labs and other researchers in saliva, tears, semen⁹⁸, fecal matter and urine⁹⁹ and breast milk.¹⁰⁰ They have even been found in African dust¹⁰¹. And the Lyme spirochetes are not just confined to Ixodes ricinus ticks. They have been found in mosquitoes^{102 103 104}, sand flies, horse flies, mites and fleas¹⁰⁵ as well. Congenital Lyme is an increasing problem.^{106 107} Dr. MD Charles Ray Jones says that of the more than 5,000 children he treated, 240 have been born with Lyme disease.¹⁰⁸ Dr. Jones is arguably the world's leading pediatric specialist on Lyme Disease. He also stated that twelve children who've been breast-fed have subsequently developed Lyme. Many people ultimately diagnosed with Lyme disease don't recall ever having been bitten by a tick or ever having seen the typical bulls-eye rash. Dr. Jones has been prosecuted for his professional opinion on the treatment of Lyme disease.¹⁰⁹



Dr. Charles Ray Jones

98 <http://www.anapsid.org/lyme/bach.html>

99 <http://www.unfictional.com/pdf/bb-in-urine.pdf>

100 <http://www.sciencedirect.com/science/article/pii/S0732889395000278>

101 <http://www.rideforlife.com/archives/000273.html>

102 <http://www.unfictional.com/pdf/bb-in-mosquitoes.pdf>

103 <http://www.ncbi.nlm.nih.gov/pubmed/9513946>

104 <http://www.ncbi.nlm.nih.gov/pubmed/10885843>

105 <http://www.muni.cz/research/publications/796098>

106 <http://www.unfictional.com/pdf/congenital-lyme.pdf>

107 <http://www.canlyme.com/congenital.html>

108 http://www.unfictional.com/pdf/Lyme_Disease_the_unknown_epidemic.pdf

109 <http://www.unfictional.com/pdf/dr-charles-ray-jones-lawsuit.pdf>

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If ALS is often Lyme, why don't antibiotics improve most ALS?

It is true that in medical trials and many anecdotal reports, people diagnosed with ALS deteriorated faster, instead of improved. There are also plenty of forum posters who said that months of intravenous antibiotics did little to nothing to slow down their deterioration.

We are very well aware of this. We purchased and thoroughly analyzed this study that showed that Minocycline worsened ALS symptoms:

Gordon PH. Efficacy of minocycline in patients with amyotrophic lateral sclerosis: a phase III randomised trial. *Lancet Neurol* 6:1045 (2007)

In our well-researched opinion and personal experience, neuroborreliosis is incurable when it is entrenched enough to do so much brain damage as to warrant a diagnosis of ALS. It is treatable - but the higher the bacterial load, the more months you have to wait before you see solid results. And very high doses of antibiotics are required, and significant deterioration is inevitable meanwhile, due to the Jarish-Herxheimer reaction and the physical migration of the spirochetes, damaging previously unharmed neural tissue.

ALS experts say that ALS symptoms never spontaneously resolve. ALS patients only get worse - never better. **Yet Tony Hofer's severely slurred speech resolved three times in a row with three different antibiotics⁷⁶, and three times returned when the antibiotic treatment was stopped. This makes it overwhelmingly likely that Tony's symptoms were caused by a bacterial infection.** Yet again, Tony mentions intravenous antibiotics failing to help him. It is clear that antibiotics influence the symptoms of ALS. Various published medical trials and anecdotal reports mention either a temporary deterioration, or a temporary improvement. But unless people switched from an ALS diagnosis to a Lyme diagnosis, ALS hardly ever cures, with the use of antibiotics.

Why is that? Most likely, by the time any doctor is willing to treat aggressively, with parenteral antibiotics, it's already too late. Dr. Marz "cured" his ALS-that-was-really-Lyme by taking double the usual dose of Ceftriaxone - four

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grams per day instead of two. And then it still took months before there was "unmistakable improvement". And Dr. Marz did not have any serious bulbar symptoms, otherwise he may well have succumbed to the "herx".

The Halperin paper mentioned three ALS patients who significantly improved on antibiotics - something that would not be expected, if ALS was not caused by a bacterium. Three ALS patients died very soon after the start of antibiotic treatment - also unexpected - but not if they were suffering from a Jarisch-Herxheimer reaction. That makes six out of 21 patients, or roughly one in three, unexpectedly responding to antibiotics - something totally unexpected and likely only explainable if the antibiotics were killing bacteria in the CNS, because there were both improvements and deteriorations - exactly what is clinically observed in the treatment of Neuroborreliosis. The initial deteriorations in ALS patients may well prove fatal in advanced bulbar cases.

Antibiotic trials for ALS are set up to fail

Because there have been many anecdotal reports of antibiotics helping ALS patients, there has been considerable interest in trying antibiotics for this condition. Clinical trials have been conducted, testing Ceftriaxone and Minocycline. It is however important to realize that these trials have never been based on the assumption that ALS is caused by a bacterium and that therefore, an antibiotic would be a suitable medicine. Instead, opponents of the bacterial etiology hypothesis of ALS have set up antibiotic trials to fail by insisting that any improvements of ALS symptoms would be due to anti-inflammatory properties of the antibiotic, or because of some assumed beneficial role in the metabolism of glutamates. The position that there exist chronic bacterial infections that can cause serious neurological syndromes is a highly dangerous one for one's career, and everything is done to avoid drawing such conclusions in research papers or starting a clinical trial under such assumption.

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One such trial was "Efficacy of Minocycline in patients with Amyotrophic Lateral Sclerosis: A Phase III randomized trial" by Paul H. Gordon et. al., Lancet Neurol. 2007;6:1045-1053. It was done under the assumption that Minocycline may have a neuroprotective effect, but the conclusion was that it seriously accelerated the demise of ALS patients, advising against its use in both ALS patients and other groups with neurological syndromes, such as MS and Alzheimer's.

The trial's fatal flaws were its short treatment duration in regard to how long severe neuroborreliosis needs to be treated, as well as the insufficiently low dosages to treat neuroborreliosis and the murky way the dosages were lowered as soon as the patient experienced discomfort or deterioration. Because the dosing of the antibiotic was done in such a way, that as soon as a significant bacterial dieoff was achieved, the antibiotic was either withdrawn or its dosage reduced. From the study:

After the randomisation visit, participants were given capsules of placebo or 50 mg minocycline for 9 months (the capsules had an identical appearance; for more details on the medication, see webappendix). Doses of minocycline started at 100 mg twice per day and increased every week by 50 mg twice per day to the highest tolerated dose or 400 mg daily, whichever was reached first (tolerance was defined by the patient's acceptance of any side-effects at that dose). If an adverse event occurred, the investigators could reduce or discontinue the study medication temporarily for one period of up to 3 weeks. If the adverse event was judged by the site investigator to be serious, the study medication was stopped permanently (for criteria for serious events, see webappendix).

In our opinion, this makes the study useless to determine the efficacy of Minocycline against ALS caused by Neuroborreliosis, because: "The highest tolerated dose" is not a useable designation of quantity, and virtually guarantees that none of the ALS patients received a bactericidal or

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bacteriostatic dose, because before such a dose is attained in the CNS, extremely unpleasant dieoff effects will manifest themselves. **As soon as these effects were deemed "adverse" by either patient or physician, the dose was lowered or the antibiotic was withdrawn for weeks at a time, inevitably resulting in complete treatment failure.** The only effect that can reasonably be expected of such an experiment is to "stirr up" the spirochetes - to cause some serious herxing but not to significantly lower the bacterial load. On the contrary, such an approach would likely result in more, not less cerebral inflammation, as a few spirochetal fragments would trigger an inflammatory immune response. Spirochetes would also tend to migrate to tissue with lower antibiotic serum concentration, causing damage to neurons, as they are often intracellular (embedded inside neurons).

Note how the study mentions that as soon as a serious Jarisch-herxheimer reaction would occur, the Minocycline would be stopped permanently. This approach guarantees that none of the ALS patients would ever improve, if the cause of their symptoms would be Lyme disease. On the contrary, it guarantees that some would deteriorate, as a result of the extra inflammation caused by the "waking up" of the immune system and the "shaking up" of the bacteria.

Oral Minocycline is even at the maximum dosages administered in the trial not to be expected to cure or even significantly alleviate the severe neurological damage of Neuroborreliosis. Chronic (antibiotic-resistant¹¹⁰) Lyme neuroborreliosis is amply documented in medical literature.¹¹¹

We made a screen capture of the graph that showed the decline of the ALS patients treated with Minocycline versus the decline of an untreated control group:

110 <http://www.unfictional.com/pdf/antibiotic-resistant-lyme-neuroborreliosis.pdf>

111 <http://www.unfictional.com/pdf/chronic-persistent-neuroborreliosis.pdf>

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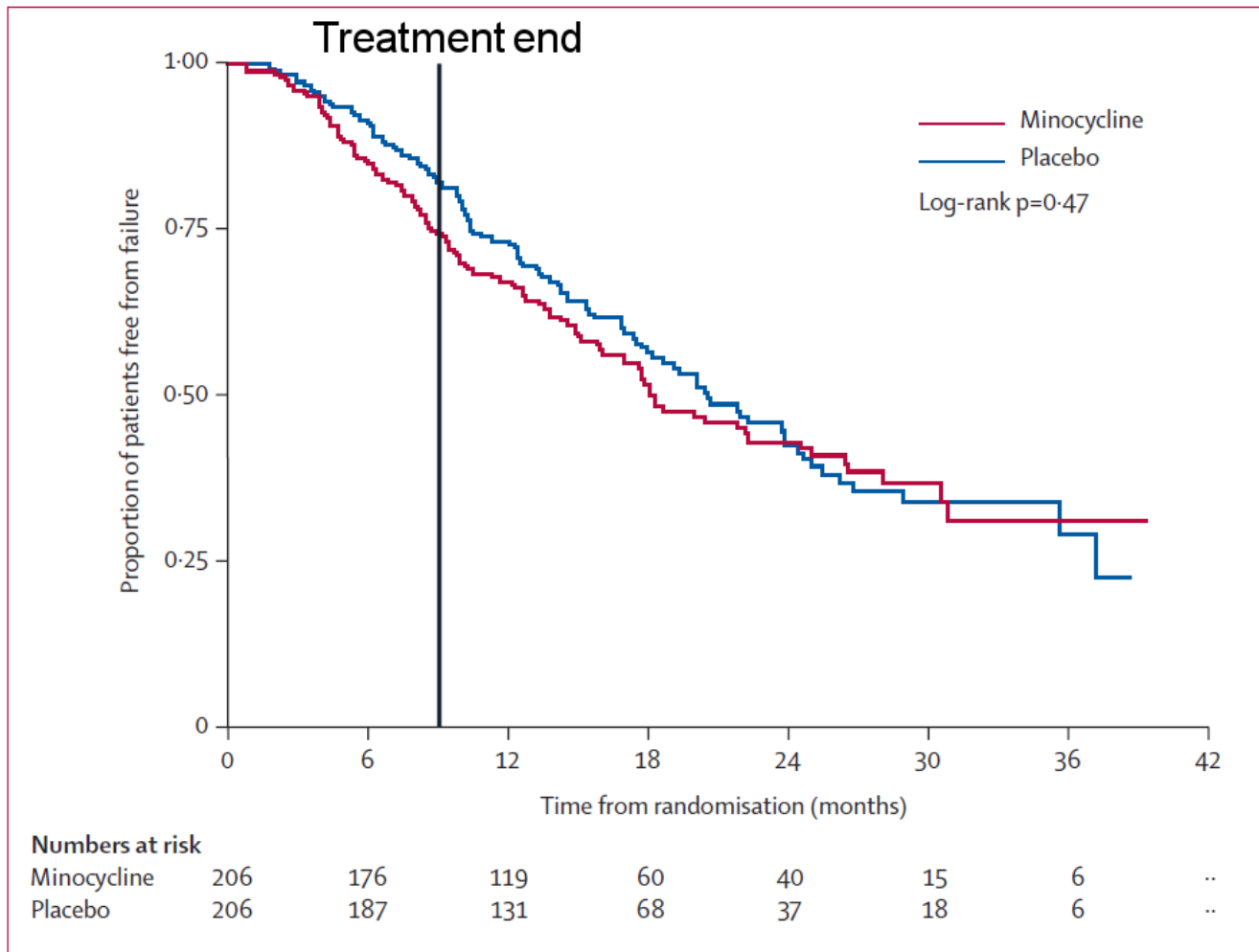


Figure 3: Failure-free survival

Failure was defined as death, tracheostomy, or non-invasive ventilation for >23 h/day.

This graph is used to justify the conclusion that Minocycline is harmful to ALS patients, because some ALS patients deteriorated much faster on Minocycline than without. As we already stated, the design of the study made it impossible for any ALS patients to be properly treated for Lyme neuroborreliosis, because as soon as a Jarisch-Herxheimer ("dieoff") reaction set in, treatment was either stopped permanently or temporarily, or dosing was reduced. What remains is the Jarisch-Herxheimer reaction, causing an accelerated demise of especially bulbar ALS patients, because their symptoms worsen due to the dieoff of *Borrelia spirochetes* in their brains.

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Ceftriaxone does not enter the central nervous system

Trials with Ceftriaxone are doomed to fail, because Ceftriaxone does not pass the blood-brain barrier.

We can adopt a conspiratorial approach and say this was deliberate, but a simpler explanation exists: The problem with most medical specialists is that they are not scientists like engineers or mathematicians. They are more comparable with bureaucrats or lawyers. They learn some facts but they do not understand the reasons behind them. Doctors think that because Ceftriaxone cures brain infections such as Meningitis, that it passes the blood-brain barrier. However, they do not understand that Ceftriaxone only passes the bbb because the capillary vessels are inflamed. Without such inflammation, there is no bbb penetration, since Ceftriaxone's molecule is not lipid-soluble and < 500 Daltons.

Lyme neuroborreliosis often does not result in Meningitis. Meningitis is the inflammation of the capillary bloodvessels that form the blood-brain barrier. Meningitis degrades the structural integrity of these vessels, and an increased penetrability for larger molecules is the result. There often only occurs Lyme Meningitis in the acute untreated stage and as soon as antibiotics reach that area, the Meningitis subsides and Cef will not penetrate anymore, leaving the remaining bacteria unexposed. Bb s.l. is microaerophilic so it moves out of the Oxygen-rich parenchyma and nestles deeper in the brain, such as inside neurons. It has a great tropism for Myelin as well.

The below study shows that Ceftriaxone does not penetrate the bbb, except in the presence of Meningitis:

Zhonghua Nei Ke Za Zhi. 1989 Jun;28(6):340-2, 381.

[The penetration of cephalosporins across the blood-brain barrier and its clinical significance].

[Article in Chinese]

Zhang YY, Wu PJ, Zhang Q.

Abstract

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"The penetration of Cefuroxime (CXM), Ceftazidime (CTZ), Cefotaxime (CTX), Ceftizoxime (CZX), and Ceftriaxone (CTRX) across the blood-brain barrier was studied in 119 patients with or without meningitis after an intravenous injection of 2 grams. Cephalosporins were undetectable or their concentrations very low in the cerebrospinal fluid (CSF), when there was no inflammation in the meninges. On the contrary, the mean CSF concentrations of cephalosporins were 2.21-5.36 micrograms/ml and the CSF/serum ratios 3.73-31.80% in acute stage of purulent meningitis."

PMID:2582913

The situation worldwide

Norway's war on Lyme patients

Perhaps the country where ALS sufferers have the smallest chance of being properly diagnosed with Lyme neuroborreliosis is Norway. Ten Norwegian doctors on the 6th of July, 2012 went on a national offensive, recruiting the little nation's #1 newspaper, Dagbladet, to go on the record saying that all the "media hoopla" about Lyme disease was scaremongering nonsense spread by "self-appointed experts"¹¹². And that Norwegian Lyme tests were trustworthy, and that alternative tests done abroad were not.

The people that mounted this attack on the basic human right of ALS/Lyme patients, the right to life (and, arguably, the right not to be tortured) are Knut Eirik Eliassen, Morten Lindbæk, Sølvi Noraas, Reidar Hjetland, Nils Grude, Jon Sundal, Per Bjark, Dag Berild, Unn Ljøstad, Åse Mygland and Randi Eikeland. We think that by their shameless abuse of the media in this manner, they have shown themselves unfit for the medical profession.

Mere days after they published their self-serving vitriolic dismissal of sound science, the same newspaper published a rebuttal: The story of the Norwegian Reiel Folven¹¹³, who had been diagnosed with ALS and who tested Lyme-negative on the Norwegian Lyme tests. He eventually collapsed, ended up in the IC ward and his family was told he would die soon. They ordered Lyme tests abroad, he tested positive and after a long antibiotic treatment he recovered and is now enjoying life again - cured of his "ALS".

¹¹² <http://www.dagbladet.no/2012/07/06/kultur/debatt/kronikk/flatt/borreliose/22441715/>

¹¹³ <http://www.dagbladet.no/2012/07/11/kultur/debatt/kommentar/flatt/sykdom/22504695/>

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More recently, Lars Monsen, a Norwegian celebrity, came forward with his Lyme story. He was unable to get a proper diagnosis in Norway but he managed to obtain positive tests in Germany. According to him, the subsequent four months of intravenous antibiotic treatment saved his life. Immediately after the story was published, Preben Aavitsland, former director of Norway's National Health Institute immediately made public that he did not believe Monsen had Lyme at all. He refused to elaborate.



Lars Monsen is famous for his three-year trek through Canada



Preben Aavitsland

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Lars Monsen in hospital.

It took a couple of weeks, but the Norwegian media finally had a "doctor" coming forward with exactly why chronic Lyme disease "didn't exist": Anders Danielsen Lie, a young actor and musician with a medical degree.



Anders Danielsen Lie

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Mr. Lie still fits his role as an "*angry, immature youth*", as he was cast in the Norwegian comedy "Reprise". In a piece entitled "*the political tick*", he pokes fun at Lars Monsen, whom he says "*sneaked out from under a shrub*" to come forward with his story.

According to Mr. Lie, the entire Lyme debate is based on **fiction** and clinics treating chronic Neuroborreliosis with antibiotics should be **shut down**. He thinks that the whole Lyme controversy is based on **narcissism** and a desire to gain votes¹¹⁴. Lyme activists are "*trying to deflect attention from evidence-based medicine*" and Mitt Romney hoped to win the elections by promising better diagnosis and treatment for Lyme disease, but Romney's cunning plan failed and he lost the elections.

Mr. Lie, six years after "Reprise", still plays the immature youth, and Mr. Aavitsland, when presented with evidence of living spirochetes in Lars Monsen's red blood cells (in the form of a video made by two Norwegian biologists where the bacteria can be seen moving around inside the blood cells), stated that he still doesn't believe Monsen has Lyme.¹¹⁵

Lars Monsen has been leading various demonstrations in front of the Norwegian government building to call attention to his plight and the plight of tens of thousands of fellow Lyme patients, so far to no avail, thanks to the relentless efforts of people like Lie and Aavitsland to tarnish them as "malingerers" and "hypochondriacs" in the media.

114 <http://www.aftenposten.no/meninger/kronikker/Den-politiske-flatten-7050611.html>

115 <http://www.dagbladet.no/2013/05/10/nyheter/innenriks/flatt/27063930/>

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Update of September 2, 2013:

Lars Monsen has been a prominent speaker to demand proper diagnosis and treatment for Lyme patients in several demonstrations in front of Stortinget, the Norwegian parliament:



The Norwegian government responded by permanently revoking Dr. Rolf Bjarne Martin Luneng's medical licence. Dr. Luneng is Monsen's doctor and the only doctor in Norway prepared to treat Lyme patients properly, instead of according to state guidelines.

By revoking Dr. Luneng's medical license, the Norwegian government punishes Monsen's participation in the Lyme-awareness demonstrations with a death sentence.

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The Norwegian government decided to permanently revoke Dr. Luneng's license to practice medicine after a "MS doctor" complained that Luneng prescribed antibiotics for his patient. It is vital for "MS doctors" that MS remains "its own illness" and in order for the status quo to be maintained, evidence that antibiotics helps against Multiple Sclerosis needs to be suppressed. The same is done in the ALS realm. Most doctors are not interested in healing their patients. They are a powerful lobby, only interested in money. Uneducated people such as Mr. Andresen bow to their demands.



Jan Fredrik Andresen (photo above) made the decision to revoke Dr. Luneng's license. Dr. Luneng's position is that ALS is often caused by Neuroborreliosis. Dr. Luneng treated Mr. Lars-Jacob Krogh who later died of ALS. Mr. Krogh had previously tested positive for Lyme disease.

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Jan Fredrik Andresen received the order to revoke Dr. Luneng's medical license from Norwegian Health minister Jonas Gahr Støre, who a few months previous, in June, warned all parties that a decision would have to be made in the case of whether there was such a thing as chronic Lyme, and whether long-term antibiotic treatment was permitted or not. Mr. Støre presumably takes his orders from US officials. It would be embarrassing for a lot of US bigwigs if there would be a progressive Western country where chronic Lyme patients would benefit from long-term treatment. That would cause upheaval and if there's anything doctors hate it's informed patients.

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Professor Emeritus in Molecular biology Morten Laane is an expert in microscopic techniques and he developed a simple method to show living spirochetes in blood by diluting it with a saline solution and incubating it for two days.



After publishing with Ivar Mysterud a scientific study on the method, Prof. Laane was fired from his tenured position in the Institute for Molecular bioscience of Blindern University in Oslo and the rest of his team was told to abandon the research. Laane's results were widely ridiculed worldwide. His method was used to prove active infection in Norwegian Lyme patients and ruffled the wrong feathers: Preben Aavitsland sent the researchers a warning that their research violated Norwegian Law¹¹⁶. The Norwegian government ordered the research to stop and all data and biological samples to be destroyed on penalty of imprisonment¹¹⁷.

116 <http://www.vg.no/nyheter/innenriks/helsetilsynet-slakter-borreliose-forskningsprosjekt/a/10146068/>

117 <http://pahoyden.no/2014/03/uio-ma-stansa-borreliaforsking>

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The Netherlands

In the Netherlands, insurers do not permit MD's to treat chronic Lyme, the condition is considered psychosomatic and therefore the country refuses to pay any type of financial support to chronic Lyme patients. An example is Joshua Verhoeven, who lost 30 kg (64 lbs) and has spent the past years in a wheel chair. He still tests positive for Lyme.

Eindhovens Dagblad 02/11/2013



■ Joshua Verhoeven (31) uit Mierlo is geveld door de chronische vorm van Lyme. Hij heeft een stichting opgericht voor (h)erkenning van de 'zeer onderschatte en ernstige' ziekte.

'Lyme sluipmoordenaar'

door onze correspondent
Vivianne van Bijsterveld

MIERLO - „Mijn leven heeft al jaren geen enkele kwaliteit meer. Het is een uitputtingsslag, ik heb doorlopend pijn”, begint Joshua Verhoeven (31) uit Mierlo zijn verhaal. De ziekte van Lyme heeft hem nu al zo'n tien jaar in zijn greep. Verhoeven: „Het begon met klachten als duizeligheid, spier- en hoofdpijnen, gewrichtspijnen, vermoeidheid. Door de tijd heen kwam er een scala aan klachten bij. Onder meer extreme zenuw-, bot- en ontstekingspijnen, hoge bloeddruk, osteoporose, geheugenstoornissen en krachtsverlies. Ook viel ik ruim dertig kilo af.”

Na een jarenlange zoektocht volgde de diagnose. „Ik had de chronische vorm van Lyme. Het is moeilijk te zeggen hoe, waar en wanneer ik het opgelopen heb. Het meest waarschijnlijke is een onopgemerkte tekenbeet.” Sinds vier jaar is Joshua volledig arbeidsongeschikt en afhankelijk van zorg. „Sinds twee jaar ben ik niet meer mobiel en heb ik een rolstoel nodig. Voor een uitkering kom ik niet in aanmerking, om-

dat mijn ziekte niet door mijn verzekering erkend wordt. Mijn familie zorgt voor me.”

Samen met zijn broer Ruben Verhoeven (38) richtte hij de Stichting Hope for Lyme op. „We willen geld ophalen voor de oprichting van een gespecialiseerd onderzoeks- en behandelcentrum voor Lyme. Ook willen we positieve aandacht vragen voor herken-

ning en erkenning van deze zeer onderschatte en ernstige chronische ziekte. Een ziekte waar duizenden mensen in Nederland dagelijks aan lijden.”

Verhoeven heeft inmiddels geaccepteerd dat hij ernstig ziek is. „Het is van levensbelang dat ik een goede behandeling krijg. Bij voorkeur in het buitenland, want in Nederland staat men nog in de kinderschoenen wat betreft Lyme.” Zijn familie vraagt middels een noodkreet in diverse bladen om een financiële bijdrage voor deze behandeling. Moeilijker te accepteren vindt Verhoeven het feit dat hij niet meer beter wordt. „De ziekte is een echte sluipmoordenaar. Ik wil de grootste rijkdom die je in het leven kunt hebben terug: mijn gezondheid.”

Het geloof in zichzelf en de onvoorwaardelijke steun van zijn broer, zijn naaste familie en zijn vriendin Danielle (29) houden hem op de been. „Helden zijn het. Zij dragen mij en de stichting een warm hart toe. Samen zullen we er voor zorgen dat de stichting Hope for Lyme haar doelen bereikt.”

www.hopeforlyme.nl



“Ik wil de grootste rijkdom die je in het leven kunt hebben terug: mijn gezondheid

Joshua Verhoeven

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