The Seven Provocative Findings of the Dr. Paul H. Duray Research Fellowship Foundation, and what they mean to Medicine and Patients

by Tom Grier, Microbiologist
July 2016

1) Mother-to-child transmission of Borrelia across the womb

2) Finding *Borrelia burgdorferi* and *B. miyamotoi* associated with Amyloid Plaques in Alzheimer’s disease brains

3) Finding Borrelia in Lewy Body Dementia

4) Nematode worms found in the CSF (spinal fluid) of Multiple Sclerosis patients

5) Nematode worms found in Alzheimer’s brains

6) Borrelia found in five deadly brain tumors (Glioblastoma multiforme)

7) *Borrelia Mayonii* and *Borrelia burgdorferi* found in human testicle

Since 1975 when Lyme disease was first introduced to the medical literature, it has been surrounded by controversy and misunderstandings. Much of the problem stemmed from trying to understand this disease entirely through antibody tests (serology) based entirely on just one species – *Borrelia burgdorferi*.

We now know that there are many species of Lyme disease. Borreliosis is not just one disease, it is part of a family of diseases that can no longer be considered separate or isolated from Lyme disease. The best example of this is *Borrelia miyamotoi*.

It is found in hard-shelled ticks just like Lyme disease, but it is a Relapsing Fever borrelia.

It took over 10 years for microbiologists to place it in the Relapsing Fever category as opposed to the Lyme-genetic grouping.

Not surprising is the fact that *B. miyamotoi* is found in the human brain right alongside *B. burgdorferi*. They may look like two separate diseases on paper, but in the human brain they are pathogens and must be eradicated together.

An even more concerning part of the Lyme disease story is that virtually no funding in any country has been put into Borrelia pathology. The fact is Lyme serology simply
cannot detect this family of bacteria with any reliability, mostly because Borrelia, once inside the human brain, is behind the blood-brain barrier, and inside human brain cells. As a result, the human immune system can no longer recognize it.

Add to this the fact there are more than a dozen species of Borrelia that cause Lyme disease, many of which can penetrate any tissue, and add a couple Relapsing Fevers that tag along for the ride, and it becomes clear that the Lyme disease blood tests based on Borrelia burgdorferi detection that have been used for 30 + years have become pretty much useless.

I first met Dr. Alan MacDonald and Dr. Paul Duray in 1992, and I was excited because they were doing the kind of research that I thought was beneficial, focusing directly on the disease process. Their pathology-based research got to the heart of it. In a sea of endless tick and deer studies, pathology seemed to me the best way to understand this disease.

I met Dr. Paul Duray through two friends of mine. Barb Jones, RN had been attending Lyme conferences for years because her elderly mother and her husband had severe Lyme disease, and I was a Lyme patient barely able to walk, stand or remain awake. At Lyme Disease conferences, we often spoke with Dr. Duray’s lab assistant Amy Jones and got the inside story.

When Barb’s mother died in 1994 I arranged for Dr. Paul Duray to do a brain autopsy to look for Lyme disease.

Barb put $2,000 of the estate towards the cost of the research, but Dr. Duray was between jobs and institutions and to cut a long story short, the Fox Chase Cancer Center kept the money and lost the brain. I was devastated.

It took over a year for them to realize they had no clue as to the location of an 87 year-old’s formalin-soaked brain!

Then in 1997 we had another opportunity to do brain Lyme research.

I had worked with three sons who had all been disabled from Lyme disease. They all made noticeable recoveries while on antibiotic therapy. The boys’ father was in a nursing home with Alzheimer’s dementia.

When the family learned that their father was just a few days from dying, the oldest son wanted to do a brain autopsy to see if their father had Lyme disease just like his brothers and himself.

They all grew up on the same farm, as hunters, farmers and loggers, so their exposure to ticks was nine months a year in an area that was highly Lyme-endemic.
I urged Jim Forris to contact Dr. Alan MacDonald to do a brain autopsy. The results were stunning, confirming my suspicions and beliefs. Borrelia entered the brain and most probably caused dementia just like syphilis.

In 1997 Alan MacDonald was the first to prove by this brain autopsy, that the Lyme bacteria not only entered the brain, but that the spirochetes penetrated neurons (nerve cells) and survived intracellularly inside human brain neurons.

This one brain autopsy proved that Borrelia entered the brain and the spirochetes penetrated brain cells!

When the attending physician at the clinic managing Jim’s father saw the autopsy report, he spewed out that it had to be syphilis. The family was outraged so everyone in the family and the tissues were tested for syphilis. Of course all were negative and the only positive tests in the family were for Borrelia burgdorferi.

The clinic immediately got a restraining order against Jim and his family.

At the time I naively thought that the clinic would be genuinely interested in the true cause of dementia, and look into other patients’ conditions. Instead they barricaded themselves away from the truth. To my way of thinking as a scientist, this was a criminal action.

The patient had been treated previously with three rounds of doxycycline for pulmonary infections, and had sulfa drugs and metronidazole for bladder infections. So he met the IDSA criteria for proper treatment of Lyme disease, yet pathology showed his brain was positive for Borrelia in almost every cross section.
This one case proved that both the tests and the treatment for Lyme Borreliosis were flawed, but not as flawed as the doctors who must diagnose Borreliosis and treat it!

--------------------------------------------------

The association of Borrelia with brain cells in an Alzheimer’s patient was too coincidental to ignore. How was Borrelia contributing to the process of Alzheimer’s pathology?

Years later Dr. Alan MacDonald and I would team up again to try and answer that question. Only instead of waiting for people to die, Alan decided to use brain banks to get instant access to possibly infected brain tissue.

Over the years several researchers working with silver-stains had noticed that, when staining the brains of dementia patients, they would find what they thought were syphilis spirochetes.

In many of the dementia brains the silver-stain would clump and form circles. Most thought this was artifact or residual stain that didn’t get washed off. Some researchers commented that they thought these clumpy circles were actually spirochetal colonies - perhaps of syphilis - and represented hundreds of spirochetes.
With today’s more accurate stains and higher resolution, we now know that these clumps were not artifacts but were “biofilms” of Borrelia, and the circles of silver-stain were probably masking the fact that the “colonies or biofilms” were covering Alzheimer plaques.

Although Alan had been doing Borrelia research for three decades, nothing propelled his research further and faster than the development of Borrelia FISH stains to allow fluorescent detection of spirochetes in human tissue.

In 2014 we had been doing our own research independently of each other and decided that we would collaborate in forming a non-profit corporation dedicated to pathology-based research. I became more administrative and Alan delved into research full force, uninhibited by bureaucracy.

We formed the Dr. Paul H. Duray Research Fellowship specifically to do the pathology-based research on Borreliosis that no one else was doing.
Armed with fluorescent FISH stains for 3 different species of Borrelia, Alan took his research in new directions.

FISH, or fluorescent in situ DNA hybridization, entails finding a perfect match to a unique sequence of DNA – technology so accurate it is used to identify criminals destined to receive capital punishment.

I would like to do an overview of the seven provocative findings of Dr. Alan MacDonald, findings that will have a positive impact on medicine for centuries.

Lyme disease is not only a fundamentally misunderstood disease: quite frankly what the public and doctors are being told by the CDC is wrong.

The longer the CDC repeat the untruths despite findings that contradict them, the more their incorrect assertions have come to be seen as out-and-out lies, and purposeful misdirection.

The Seven Discoveries

First provocative fact about Lyme disease:

Let me give a simple example. From 1987-1989 Alan MacDonald reported on eight fetal autopsies from miscarriages in New England, all of which showed the presence of Borrelia burgdorferi not only in the fetal tissues, but also in the umbilical fluid, the placenta, and the fetal organs.

Transplacental transfer of Borrelia was a published fact in 1989, yet the medical experts on Lyme disease insisted as late as 2008 that there was no evidence of transplacental transfer from infected mother to fetus. Why?

Now with fluorescent FISH stains providing incontrovertible proof, only a blind man or an idiot would ever continue to deny that Borrelia is transferred from infected mother to child in the womb.

All Photos property of and copyrighted by Alan MacDonald, MD
Fetal Autopsy of Brain Neuron *Borrelia burgdorferi* is transversing through the brain cell

*Burgdorferi in Placenta*
What this discovery means:

Since we now know that Borrelia can traverse both blood vessels and tissue, we know that Borreliosis and its associated diseases can cross the placenta and enter the developing fetus. So infected mothers must be treated with antibiotics.

In the 19 patient study done by the Lyme Disease Foundation, amoxicillin was given to mothers infected with Lyme disease during their pregnancy. Amoxicillin was found to be safe for mother and child and had no adverse outcomes for the fetus.

Looking at pathology data dating back to 1987, we must now consider that Borreliosis is a possible factor in miscarriages and Sudden Infant Death Syndrome (SIDS).

Second Provocative Fact:

In 1982 when the cause of Lyme disease was found to be a spirochete, immediately the CDC tried to distance this Lyme disease from a group of diseases caused by Borrelia spirochetes known as Tick Borne Relapsing Fevers.

We can no longer do this since the Relapsing Fever Borrelia miyamotoi was discovered to be in the same hard-shelled Ixodes ticks as the Lyme bacteria.

_B. miyamotoi causes a Lyme-like disease in humans._
*B. miyamotoi* likes warmer temperatures than *B. burgdorferi*.

And *B. miyamotoi* enters the brain and can form biofilms in amyloid plaques just like *B. burgdorferi*.

What we discovered in 2015 was even more disturbing.

*Borrelia miyamotoi*, a Relapsing Fever borrelium, was routinely being seen inside human brains. As Alan continued to do his brain autopsy research on Alzheimer’s brains from Harvard Brain Bank, he found, using fluorescent FISH stains, that *B. miyamotoi* was not only in many of the brain tissue samples, but that it was usually associated with amyloid plaques.

It may turn out that *B. miyamotoi* is as prevalent as, or even predominant over *B. burgdorferi* in human brains and dementia patients.

This was bad news because *B. miyamotoi* is not detected with the Lyme serology tests that are used, and doctors rarely think to test for any Relapsing Fever Borrelia. So this kind of brain infection probably goes undiagnosed for decades. Despite the severe neurologic symptoms the patient exhibits, the patients most often are left untreated leading to dementia.
The correlation with amyloid plaques was intriguing.

Looking at research done by others, it was obvious that Borrelia had a profound effect on the brain cells.

Jorge Benach showed that when Borrelia was added to rat-brain culture, the brain cells would immediately swell and die.

Jill Livengood and Robert Gilmore of CDC showed the *Borrelia burgdorferi* has a tendency to seek out blood vessel lining cells, brain glial cells (cells which function in support of the nerve cells), and brain neurons.

When Judith Miklossy isolated Borrelia bacteria from a human brain with dementia, she cultured the cells along with rat-brain cells that were also grown in culture. The brain cells enhanced with added microglia, started to overproduce *amyloid-precursor-protein* (APP).

This APP quickly converted to beta-sheet amyloid, the hallmark of Alzheimer’s.

Then other markers of Alzheimer’s also occurred:

- hyperphosphorylation of microtubule protein tau
neurofibrillary tangles
vacuolization

In short Borrelia induced amyloid formation and became toxic to brain cells.

What we showed was that every amyloid plaque we stained for Borrelia had associated with it a biofilm of either B. burgdorferi or B. miyamotoi. What for centuries had been seen with silver-stain as amorphous globs, now shone brightly as Borrelia spirochetes.

Many people in the Lyme community suspected that Lyme disease was somehow associated with dementia, but the Lyme tests kept coming back negative. We now had the answer: *Borrelia miyamotoi* was a common pathogen of the brain hidden away from the immune system behind the blood-brain barrier and sequestered inside biofilms and brain cells.

Of course the Lyme tests were negative!

Now the question was: What about other neurologic diseases like Parkinson’s, Multiple Sclerosis and Lewy body dementia?

**What this means to patients:**

The presence of Borrelia in Alzheimer’s plaques means some dementia patients if caught early, may improve on antibiotics or stop the progression of the disease.

But Lyme tests will not detect B. miyamotoi so treatment must be done by clinical diagnosis and response to therapy.

It is likely that lengthy therapy is needed, perhaps life-long treatment. *(Later we will see why adding an antiparasite medicine like albendazole might be the first step.)*

----------------------------------------

**Third Provocative Fact (the case of Jack Gordon from Iowa):**

Lewy body dementia is the second most common dementia in humans. It is recognized at autopsy only by the presence of round bodies mostly composed of a viscous protein called alpha-synuclein.
There are both intraneuronal Lewy bodies found inside brain neurons, and extracellular Lewy bodies that float around the brain between brain cells.

They are toxic to the brain. This is what plagued Robin Williams before he committed suicide. He was quite sick and was probably headed for assisted living within a year or two.

A Lyme patient from Iowa - Betty Gordon contacted us in 2014 about her husband who was dying. Betty had a diagnosis of Lyme disease. And she was convinced her husband had it too. Was his dementia also due to Borrelia bacteria?

Betty donated a large sum of money, and this helped fund many aspects of our research and of course Jack’s brain autopsy. Not only did we find the Lyme bacteria in Jack’s brain, but also he clearly had Lewy body dementia.
Shortly after Jack Gordon’s brain autopsy and the discovery of a link between Lyme and Lewy body Dementia, Alan decided to look at another chronic neurological disorder often associated with Lyme disease: **Multiple Sclerosis.**

**What this means to patients?**

Lewy body Dementia is the second most common form of dementia in America. If these patients can be found in the earliest stages it is possible that antibiotics could halt the formation of alpha-synuclein.

Treatment would have to be aggressive and prolonged.

If we better understand how alpha-synuclein is formed, we would have additional treatment options.

*(Later we will see why adding an antiparasite medicine like albendazole might be the first step.)*

The Fourth Provocative Finding: Across America in every Lyme Disease Support Group, there is at least one recovering patient who was once diagnosed with Multiple Sclerosis.

Yet the medical community refuses to take a serious look at this connection and has failed to dedicate a single taxpayer dollar to investigate a possible link. This is even
harder to understand when you do some digging and find that prior to WWII over fifty published studies linked MS to the presence of spirochetes in the brain. This pathology-based research started in 1911 in both England and Germany but stopped abruptly due to the war.

Dr. Gabriel Steiner had clearly shown that in about one in every ten brain autopsies he could find spirochetes using his own silver-stain. We still use Steiner’s stain today to detect spirochetes, but for some reason we don’t acknowledge his discovery of spirochetes being associated with Multiple Sclerosis.
I, myself, had been diagnosed with Multiple Sclerosis in 1989. When it turned out to be Lyme disease I remained curious about other MS patients.

So in 1994 I designed and implemented the Lyme-Endemic Area Multiple Sclerosis treatment study in Pine County, Minnesota.

I screened several dozen MS patients for Lyme disease. Anyone who was positive got treated, but our study only took the MS patients that were seronegative (antibody negative) for Lyme disease.

The idea was that Lyme was being missed because the tests were poor, so we wanted only seronegative patients.

We then treated all 26 patients for three months with antibiotics. The results were not spectacular.

Eight patients had some positive response to antibiotics, and three of them seroconverted showing us that they did in fact have Lyme disease.

But the fact is that the patient recovery was only fair was disheartening and disappointing.
However one patient continued on amoxicillin for 15 months after our study ended and she had a nearly full recovery and was able to go back to work. Perhaps our treatment period was too short?

Still I felt we were missing something.

While we had a 30% positive response rate it was disappointing to me.

Later in 1998 I attended a Lyme conference where Patricia Coyle of SUNY had nearly identical results in a group of 47 MS patients from New York area (a highly endemic area for Lyme disease). She had 15 of 47 respond and all showed Borrelia proteins in their spinal fluid. Honestly I expected more and better responses. The response by the medical community was completely nil. No one cared!

Then Alan found another missing piece of the puzzle. After ordering ten CSF (spinal fluid) samples from the Boulder, Colorado bank, he was focusing his microscope at low power and saw something at fourfold magnification.

When he stained the slide and went to 10x he found something that had been invisible to labs for decades. All the patients had a parasite in their spinal fluid. Alan found nematode worms!
Upon further examination, the nematodes he found also contained *Borrelia burgdorferi* inside their gut.

This discovery helped make sense of what I had found in my Multiple Sclerosis study. If the MS patients had Nematodes and *Borrelia* in the central nervous system, then the proper treatment wasn’t just antibiotics.

Rather, it would have to be high dose antiparasitic drugs followed by antibiotics, and this combination would have to continue until all parasites in all their growth stages including eggs, had been cleared from the brain.

Why had so many missed this discovery?

In today’s fast-paced medicine, anytime MS is considered the CSF specimens are placed directly in an automated analyzer that looks for MS markers, but not for parasites.

On a slide without stain, the CSF looks normal.
Then if the lab looks at the slide at 40x magnification or higher, they will miss the worms.

It’s like looking at an elephant’s leg an inch away and trying to figure out what the whole animal looks like.

**What this means to MS patients?**

Clearly this suggests that despite any other treatment, the MS patient should first be placed on an antiparasite medicine for nematodes, followed by antibiotics for Borrelia.

Treatment would have to be aggressive to get past the blood-brain barrier, and prolonged to eradicate the Borrelia.

-----------------------------------------------------------

**Fifth Provocative Discovery:**

Alan then decided to take another look at his Alzheimer’s brain samples. Had he missed nematode worms in the brain?

When Alan looked at both Alzheimer’s brains and Lewy body brains he did indeed find nematodes, and inside the nematodes were *Borrelia* bacteria!
This changes everything, we ever thought about dementia! Since this is the single most provocative discovery in all of Alan’s work, he decided to confirm his findings. He used a red “pan Cytokeratin” stain to recheck the samples.

This nematode found in an Alzheimer’s patient’s brain had a second staining for Borrelia. The DNA beacon probes lit up indicating Borrelia burgdorferi is inside the nematodes that are inside an Alzheimer’s brain.
Cytokeratin should never be in the human brain.
If it is in the brain it came from somewhere else. In this case Alan found clearly defined spirochetes and something that chilled me to the bone: **worm eggs! Hundreds and hundreds of nematode eggs.** This explains why dementia is progressive. It explains why dementia accelerates with time.

Since nematodes have both a mouth and an anus, it dictates that if the nematodes are eating, that they are also pooping. Alan found nematodes in all life cycles including eggs, and found their excrement throughout the brains of Alzheimer’s patients.

Once again everything we knew about the treatment of dementia had changed. These patients need both an **antiparasitic** drug, and antibiotics.

And yes the nematodes contain Borrelia bacteria; so killing them probably releases more Borrelia into the brain.

In the case of Lewy body dementia, no one had ever found an adequate hypothesis to explain what caused the formation of Lewy-Bodies.

No one knew why Lewy bodies that were outside neurons were bigger and contained “granules and spaces” surrounding the alpha-synuclein spherical bodies.
The fact that Borrelia bacteria can penetrate neurons and the fact that neurons produce the alpha-synuclein that forms the Lewy bodies, once again seemed like a coincidence that had to be explored.

It turns out that there may be several possible explanations in the formation of toxic alpha synuclein which have not been considered up till now. (See Alan MacDonald’s paper on nematodes and Lewy-bodies at the F1000 website)

One possibility is that Borrelia or more likely Nematode worms causes the brain-neuron to produce the Lewy body inside the nerve cell by inducing alpha-synuclein production in the nucleus.

Then with time the *Borrelia* and nematode secretions and alpha-synuclein kill the neuron. The neuron wraps itself around the alpha-synuclein sphere and becomes an extracellular Lewy body (outside of the nerve cell).

The nematodes too could theoretically be responsible for the Lewy Body formation.

The worms produce a type of protein called LEA.

When Alan stained the worms using an antibody-based stain to detect human alpha-synuclein, they lit up.

But no nematodes have ever been known to produce alpha-synuclein.

It may well be that the LEA proteins which are similar in shape to the alpha-synuclein, were cross-reacting.

And this might mean that nematode worm proteins are mimicking human alpha synuclein in the brains of people with dementia.

The neurons would be duped into taking up the foreign protein. It would then destroy them, with the dead neurons coiling up into Lewy bodies.

Staining these extracellular Lewy bodies for neuron proteins reveals the truth. Extracellular Lewy-Bodies were once healthy neurons. What killed the neurons? Was it Nematodes? Was it Borrelia?

Much is to be learned but both nematodes and *Borrelia* seem to be playing a major role in Lewy body Dementia.

What does this mean to Alzheimer’s and other dementia patients?
It means antibiotics isn’t enough. Many doctors have tried antibiotics in Alzheimer’s disease with limited success and mostly disappointment.

Treatment should begin with a systemic antiparasite medication for nematodes and treatment must be prolonged to kill all life stages of the parasites.

Since the nematodes carry live *Borrelia* in their gut, therapy must include several rounds of antibiotics.

Since the tests for parasites and Borrelia are poor for a central nervous system infection, diagnosis and therapy are made on the basis of clinical symptoms and response to treatment.

---

**Alan’s Sixth Provocative Discovery:**

Almost by sheer luck Alan was able to obtain simultaneously five samples of a rare and fatal brain cancer called *Glioblastoma Multiforme*. Remember that the CDC through Livengood and Gilmore’s work had shown that *Borrelia burgdorferi* seeks out human glial cells in the brain.
So once again using his species-specific FISH stains, Alan tested cancers for the presence of *Borrelia* and it was clear that *Borrelia* was associated with the tumors.

What role *Borrelia* played in the formation remains unclear, but one has to ponder the possibility that antibiotics might have delayed or even stopped the cancers from forming?

Subsequently nematode worms were found in Glioblastoma multiforme brain tumors.
Back in the early 1990s Dr. Paul H. Duray had speculated that some cancer-like conditions seen in Lyme patients were probably due to Borrelia bacteria, but proving that connection eluded him.

Paul often collected and froze tissues sent to him especially granulomas (sarcoidosis), lymphomas, and others.

These samples and Paul’s research were all lost when he died unexpectedly.

What does this mean to Glioblastoma patients?

If a patient has Borreliosis we know they are at risk. So aggressive antibiotic treatment must be given.

If the patient develops a Glioblastoma then antibiotics and an antiparasitic agent should be considered.

If we find the mechanism by which Borrelia induces this cancer, we may have better answers to both diagnosis and treatment.

The Seventh Provocative Discovery by Alan MacDonald:

In 1992 Amy Jones, Dr. Paul Duray’s pathology assistant, found Borrelia burgdorferi in the breast milk of infected mothers, and she found B. burgdorferi in the semen of infected men.

While sexual transmission of Lyme disease has been speculated, it is a difficult thing to prove cause and effect.

Borreliosis is not like syphilis where shortly after sex a noticeable uniquely identifiable rash (chancre) forms.

In Lyme it is suspected that it may take months or years to manifest symptoms, but no one has proven sexual transmission in humans.

Work by the legendary veterinarian Dr. Elizabeth Burgess, DVM, PhD showed in 1990 that dogs infected with Lyme disease were transmitting and infecting female dogs through sexual transmission.
Still the proof in humans is lacking. The presence of the bacteria is not proof, but indicates a strong likelihood.

What Alan found was the presence of both *B. burgdorferi* and *B. mayonii* in the testicle of a logger from Twig, Minnesota. What is provocative is that this man had been treated almost continuously on antibiotics for the last seven years of his life. Yet he still had live *Borrelia* in both his brain and testicle. He also had *B. miyamotii* in his brain.

*The case for sexual transmission is stronger than ever. Borrelia survives in the testicle despite aggressive and prolonged antibiotics.*

FISH study
Positive Tissue Control
[ Testicle patient SS ]
----
DNA probe bbo 0740
*Burgdorferi* Borrelia family
----
Bright Green/white Signals
are sites of
*Borrelia* binding the
DNA probe 740
(inner Cell membrane
-Gene Equivalent ORF)

What this means to the Medical Community?

Lyme disease and some Relapsing Fevers are probably sexually transmitted.

Aggressive antibiotics may not be enough to eradicate Borrelia from the testicle.

So short of abstinence the best prevention of sexual transmission is **the use of a condom between couples.**

Also treat all Borrelia infections early and aggressively to prevent the establishment of Borrelia in immune privileged sites like the testicles and sequestered sites where antibiotics have less effect.
Testicle positive control - DNA probe 740

Borrelia biofilm communities (2)
The larger showing internal Spirochetes

LARGE WHITE ARROW:
POINTS TO A NEMATODE EGG- OVAL
STRUCTURE - SPIROCHETES DWELL INSIDE OF THE EGG (GREEN – THREAD-LIKE )
1000 x magnification- Oil immersion
DNA probe for Inner cell membrane ORF of
*Borrelia burgdorferi* ( sl) FAMILY of borrelia sub-species
Bbo 0740 Molecular Beacon Dna Probe- FISH method study
Of Autopsy Testicle from a Minnesota Man.
MAYONII BORRELIA Species – visualized with DNA probe tagged with Nile Blue fluorochromes – 400x magnification – White arrow

**Cautionary note:**
Nematode infestations of the CNS require very careful treatment and monitoring by an experienced physician. Patients should not attempt to self-treat. Some anthelminthics can cause severe inflammatory reactions which might require concomitant steroid treatment in order to avoid a fatal encephalitis.

**FISH probes developed by Dr. MacDonald embody hope for the future**

To date Dr. Alan MacDonald has developed specific probes for three Borrelia species: *Borrelia burgdorferi*, *B. miyamotoi*, and *B. mayonii*. These have been capable of detecting a range of strains including European borrelia.

The probes are “Molecular Beacon” DNA probes which have an excellent rate of accuracy. They will not give off their fluorescent signal unless every single nucleotide (component unit) of the target DNA sequence matches perfectly. So when we use these probes we can be very sure that we are detecting Borrelia and not something else.

These DNA probes could open the door to accurate diagnosis in the future. As we have seen, they are already unlocking the secrets of Borrelial involvement in major neurodegenerative diseases.
But more research needs to be done before these findings will be accepted by the wider medical community. And to do this, we need your help.

All of the work here at the Foundation is done on a non-profit basis.

*We do not receive any government or industry funding, but rely on donations from patients and other interested individuals.*

Please support our work by giving generously, and by encouraging *organisations* with which you are affiliated to do the same.

Together we can bring to light the full spectrum of *Borrelia* and *nematode* worm involvement in neurological disease, and help bring about cure.

Please click here to donate to the Dr. Paul Duray Research Fellowship Foundation. [https://durayresearch.wordpress.com/donate/](https://durayresearch.wordpress.com/donate/)

**Further Reading:**

**Borrelia and Nematode Worms in Neurodegenerative Disease:**

**Alzheimer’s Disease and Lewy Body Dementia**


**Multiple Sclerosis**


**Brain Tumors (Glioblastoma multiforme)**

DNA Probes prove failure of antibody tests to detect Borrelia infections


Videos

Dr Alan MacDonald explains his findings of Borrelia and Nematodes in MS, Lewy Body Disease, and Brain Tumor, London AONM conference May 2016

https://vimeo.com/166685590

Presentation by Dr. MacDonald in which he traces the history of his Alzheimer's research up till 2014. Also discusses the evidence of mother-to-child transmission, and the failure of current antibody tests in diagnosis. June 2014

https://www.youtube.com/watch?v=TjfWFalivlc

Youtube and Vimeo channels of Dr. MacDonald, with many educational videos:

https://www.youtube.com/channel/UCQAFF2MF1YHuPHCF7oF2JHQ

https://vimeo.com/user27613099/videos

End of Article

Please note:
None of the information in this article is intended to substitute for medical advice by a qualified physician. Always consult a doctor in matters of your own health.