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PART III

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BIOLOGICAL WARFARE

Previous to our visit to see Dr. Burgdorfer, Karen and I already knew that ticks and other insects had been in used for bio-warfare purposes in the past. At one point I had discussed the subject with Dr. Burgdorfer after reading a book written by Rachel Verdon. Verdon's book, "Lyme Disease and the SS Elbrus" (2002) is a very well-documented book that implicated Lyme disease as a major component of biological warfare (BW) that purportedly arrived on the shores of America via infected rats on fur-trade ships from Russia. The back cover of the book reads, in part, "Did Nazis and Soviet Communists agree to implement long range plans to overthrow world democracies via tick borne diseases of the nervous system?" That sounded bizarre to me at first glance, then, I read her book...

Verdon describes an alliance between Soviet BW factories and Nazi scientists, and she lists the reservoirs, primarily rats, and the diseases of BW to include a plethora of tick-borne as well as other vectors of dangerous disease agents.

The above are potential agents of infectious diseases that can be transmitted from their animal reservoirs to humans via ticks, fleas, and other insect vectors. This method was employed by the Japanese and Russians, and possibly others, during war years, by dropping fleas and ticks on human populations.

Verdon challenges the reader to weigh for himself from the documentation she presents in her book as to whether the US Government used ... "the 100 Person's Act to cover up international organized crime, biological terrorism and the illegal drug trade by Nazi war criminals."

Verdon mentioned a Dr. Hefezi from Iran who petitioned a visit to Dr. Burgdorfer's Montana lab. (She states that Dr. Hefezi's records were noted in the National Archives State Department correspondent files as "Missing.") Verdon stated that Dr. Burgdorfer had collected a large number of Middle East tick species from Dr. M. Baltazard of Tehran's Pasteur Institute.

Up to that point it had never entered my mind that Dr. Burgdorfer had anything to do with biological warfare.

I called him and asked him about what I had read in Rachel Verdon's book. He had not read her book but he admitted that what she said about the Middle East ticks he had collected was true. He said, "Yes," he recalled Dr. Hefezi's visit to his lab.

This was the first time he had ever talked to me about being a biological warfare expert. He further disclosed that one reason the United States government had recruited him to fill a position at Rocky Mountain Laboratories was that he was an expert on biological warfare.

Although that was the era of the European "brain drain", also known as "operation paperclip", Dr. Burgdorfer recently said that he was unaware whether he was ever considered to be part of that particular group.

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After his arrival at Rocky Mountain Laboratory (RML, NIH, NIAID) from Switzerland in 1951 he was given his own lab to conduct research on ticks and the biological agents that can be transmitted by ticks, lice and fleas. He was already a world expert on the subjects of Borrelia, plague, and Rickettsiae agents such as the different types of typhus to include the spotted fevers. (See spotted fevers under "Typhus")

He told me that he worked under a "Dr. Phillips" at Rocky Mountain Laboratory in cooperation with the bio-warfare activities ongoing at Fort Detrick, Maryland. He could not publish any of his work because he was not yet a citizen of the U.S. However, he traveled to Fort Detrick on one occasion where for a few weeks he shared his knowledge and demonstrated his expert techniques to U.S. military scientists.

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After working for three and a half decades at RML he was assigned the title of "Scientist Emeritus" at his retirement.

He was provided with a small laboratory that would remain at his disposal during his retirement. He was to serve as a part-time biological warfare consultant for the ongoing research staff at Rocky Mountain Laboratories. RML is at present a biological safety *Level-four* facility. A level four lab is considered the most dangerous because it contains incurable pathogens that are lethal to humans and animals.

Fort Detrick was the center for America's biological weapons program between the years 1943 and 1969. By 1970 the program was terminated by President Richard Nixon who ordered that all stockpiles be destroyed.

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Dr. Burgdorfer had participated in the Fort Detrick work for nearly 3 years during the years 1951-1953, working primarily with plague, a serious often fatal illness, caused by the Yersinia pestis bacillus. One form of the disease is *bubonic plague*, its microbial agent normally transmitted to animals and humans from squirrels, rats and other rodent reservoirs via flea vectors; whereas *Pneumonic plague* is a contagious respiratory tract infection.

Another agent he investigated for Fort Detrick was the louse-borne agent of typhus to see if it could be maintained in ticks. He tested thousands of ticks, yet he was unable to demonstrate that it could be maintained in the midgut of ticks. This is true with many insect and arthropod vectors. The microbial agents they harbor are very specific as to their preferred reservoirs and vector hosts necessary for their life cycles. For example, all types of mosquitoes cannot vector the agent of malaria.

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In 1972 The Biological Weapons Convention prohibited the research, development and proliferation of offensive biological weapons. The treaty allowed defensive work to continue. (See "History of Bio-warfare Incidents" Arizona Daily Star Nov. 4, 2001, A11).

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Aside from the passage that mentioned Dr. Hefezi's visit to Dr. Burgdorfer's laboratory, I recalled another disturbing connection about Borrelia and MS in Rachel Verdon's book. She alluded to Dr. Byron Waksman, the MS society, and Byron Waksman's Russian immigrant father, Nobel laureate Dr. Selman Waksman as figuring "... heavily into the history of Lyme disease, trapped in a vice between the feuding FBI and CIA".

Verdon further states that Dr. Byron Waksman, while serving as The National Multiple Sclerosis Society's vice president, had "... gone into great detail over Lyme's involvement in the diagnosis of MS." She implies a collaborative (but not proven) connection between Selman Waksman, Merck Pharmaceuticals and Russia, based on Waksman's frequent trips to Russia. ("Lyme Disease and The SS Elbrus", Rachel Verdon, p. 12, Elderberry Press, 2002).

As I mentioned earlier in this book, in 1984 Dr. Byron Waksman had sent me information about a virologist, Dr. Vincent Marshall, DVM, the researcher who had theorized that MS was caused by spirochetes. He warned me to never try to contact Dr. Marshall. After reading the letter copies of Marshal's material that Dr. Waksman had sent to me, I ignored his dire warning and called Dr. Marshall at his office in Iowa.

Dr. Marshal was the first person other than Dr. Waksman to discuss in detail with me the research regarding spirochetes as a cause of MS. I had already read the copies of letters he had written to Dr. Waksman that Dr. Waksman had forwarded to me. The letters were part of Dr. Marshsall's request for a grant application and for building a solid case for a spirochete cause of MS. The letters contained a great deal of information about Dr. Gabriel Steiner, MD, and microbiologist Rose Ichelson, PhD. Each of them had independently determined the proof of MS-cause during their separate research studies that revealed Borrelia-like spirochetes via serology and autopsy studies.

What Dr. Steiner discovered during autopsies were borreliae–type spirochetes in and around MS patients "fresh brain plaques" but not in the older "burnt-out plaques."

(See G. Steiner, *Borrelia myelopthora* and Vincent Marshall, Med. Hypoth.)

In addition, Marshall discussed the German MS research done during the Holocaust at a time when it was considered a fact that spirochetes caused MS.

Dr. Marshall went into detail about the MS/ Borrelia spirochetes' proof-of-cause evidenced independently by Dr. Gabriel Steiner, MD, and microbiologist Rose Ichelson. He gave me the name of Ichelson's physician, a prominent Philadelphian, Dr. James Guiffre, MD. I later contacted him as well. He agreed with Ichelson's MS/Borrelia studies.

I shared all of this with Dr. Burgdorfer in the early 1980's by sending the Waksman/Marshall correspondence copies and asked that he keep the information confidential.

Dr. Marshall at that time was very much interested in my experiences with MS following TBRF exposures and we had quite a long discussion comparing notes. He asked that I send him any information that I had published.

Our discussion came to an abrupt halt when his relaxed tone suddenly changed and he said furtively, "The CIA is watching me ... two of their agents just pulled up and are parked in front of my office." He became quite agitated and we quickly ended our conversation.

It would be four years later, 1988, that I met him at a Lyme disease conference hosted by Pfizer pharmaceutical company. His demeanor appeared calm, rational and sophisticated. He apparently knew who I was, and we discussed the conference and MS. I took a photo of him and, oddly enough, neither he nor I made any reference to our four years earlier conversation of 1984.

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THE EIS

The Epidemic Intelligence Service (EIS) was formed in 1951 for the purpose of creating an offensive bio-warfare capability. It is of note that Lyme arthritis was first publicly acknowledged by two CDC-trained (EIS) officers. Rheumatologist, EIS officer Dr. Allen Steere at Yale University was contacted by EIS officer Dr. David Snydman from the health department in Connecticut. At that time it was natural to think that EIS officers should be involved with investigating local Lyme, CT., health issues that had been reported to the local officials. In later years EIS officers became under suspicion within patient groups whose members noted that far too many of them were involved and they seemed to be at odds with Lyme disease patients.

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Dr. Steere, who seemingly took charge in a leadership role among his colleagues at Yale University in 1975, originally called the disease "Lyme arthritis".

The cases were soon linked to tick bites, but it was not until well into 1981 that the mysterious spirochete Borrelia burgdorferi, cause of the clustered arthritis cases, was discovered. (SCIENCE, Sept.1982).

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Why would it take nearly seven years before the pathogenic Borrelia cause was discovered? After all, I had discovered the source of my husband's fevers as being caused by TBRF. It took me only two hours at the library to determine that it was caused by chipmunk ticks that transmitted *Borrelia hermsii*, a specific species of Borrelia. As a housewife who lacked a college degree, I was certainly no match for Yale physicians

such as Dr. Steere and other top government scientists, many of whom were trained in epidemiology at CDC. Why would it take seven years for them to discover the Borrelia cause of Lyme disease?

Furthermore, it was two housewives in Connecticut that actually "discovered" Lyme arthritis, not the two CDC- trained EIS officers Snydman and Steere.

It soon became clear that disproportionately high numbers of EIS officers played key roles in nearly every area of LD public policy. This included EIS involvement in case definitions, diagnostics, and treatments as well as control over select peer-reviewed scientific publications. Lucrative funding grants seemed to favor this esoteric group as well.

As time went on, in almost every case the EIS officers seemed to be in disagreement with patient groups' efforts regarding diagnoses and treatments. Lyme disease soon became a volatile arena of mistrust between patients and public health officials. Patient groups asked, "If Lyme disease is so 'hard to catch and easy to cure,' Why are overwhelming numbers of CDC-trained EIS officers assigned to it?" I realized in retrospect that EIS officer Rusty Gerber played a key role in our Arizona support group's clash with our local health department when Dr. Gerber requested that Dr. Burgdorfer send copies of all of our member's serology reports to him at ADHS in 1984.

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I was convinced that the Tahoe chronic fatigue epidemic was caused by TBRF, yet it was never under consideration until Dr. Komaroff contacted me in the 1980's at Polly Murray's suggestion. When he asked me to participate in his New England/Tahoe study to compare his CFS patients with our Arizona support group, our group provided serum samples. I suggested that he contact experts such as Dr. Louis Magnarelli to perform the serology. Instead, he contacted EIS officer Dr. Steere. I had not mentioned Dr. Steere to him. I could only wonder why and how Dr. Komaroff came to collaborate with Dr. Steere.

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CONSPIRACY THEORIES AND THE EIS

It is disturbing that a widening division has escalated over the last 30 years because of controversies between scientists, physicians, and patient

groups. Conspiracy theories are rampant, especially in regard to the high numbers of CDC-trained EIS officers who seem to be overly represented in Lyme disease as well as within the members of the Infectious Disease Society of America (IDSA), the society being openly hostile toward patient groups and physicians in regard to LD issues.

The society members and their self-appointed investigative panel selected to issue directives to physicians, regarding the denial of chronic stages of LD, claim to offer scientific "proof" in a handful of their published literature that is miniscule compared to the over 300 published scientific publications regarding the persistence of Borrelia.

EIS officer Dr. Alan G. Barbour was transferred from Utah to the Rocky Mountain Laboratory facility in Hamilton, Montana a year or two before Dr. Burgdorfer discovered his now famous *Borrelia burgdorferi* in 1981. Dr. Barbour, a highly esteemed, well-qualified expert of Borrelia as well as anthrax, was the first to culture the Borrelia burgdorferi pathogen. He was skilled in regard to the development of serological stain, culture medium, and convalescent tests for it. It is amazing how quickly he was able to develop such complicated stains, culture medium and especially the tests; something that might well have taken not months but years to do.

One of Dr. Barbour's close colleagues within the Lyme disease spectrum is EIS officer Dr. Jorge Benach of New York. Dr. Benach and Dr. Ed Bosler (who served with me on the Board of Directors of the LBF in 1988) were the two epidemiologists that provided the Shelter Island ticks to Rocky Mountain Lab. Dr. Bosler collected the ticks and Dr. Benach sent them to Dr. Burgdorfer. Dr. Benach received the credit, was promoted, and became Dr. Bosler's boss. Those Ixodes hard-bodied ticks were supposed to be dissected by Dr.Burgdorfer to detect the Rocky Mountain spotted fever pathogen *Rickettsia rickettsii.* He instead found *Borrelia burgdorferi.*

During the time of Dr. Burgdorfer's Borrelia discovery, EIS officer Dr. Allen Steere claimed that since he had repeatedly contacted Burgdorfer at his lab in Montana to inquire about his expert techniques in dissection for the purpose of detecting microbial tick-borne agents, the bacterium Burgdorfer had discovered in 1981 should be named "Borrelia Steeri" after himself, instead of after its discoverer Dr. Burgdorfer. Although Drs. Steere, Benach and Barbour were all CDC, EIS-trained officers, Burgdorfer and Dr. Bosler were not. Dr. Burgdorfer was a bio-warfare expert, however.

Dr. Burgdorfer told me that Drs. Barbour and Steere claimed that they had "discovered the spirochete cause" of Lyme disease, but when

Burgdorfer asked to see it, he recognized immediately that the spirochete they (Barbour) showed to him was... "... a *Leptospira*, not Borrelia. "It was certainly not "*Borrelia burgdorferi*," the now famous cause of LD.

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I can't help but wonder if Dr. Burgdorfer, the world's top tick-borne disease-agent expert, was set up to be challenged. Was it a set-up, just as a test, to see if this microbiological agent was virtually undetectable by this highly esteemed tick-pathogens expert scientist?

Was Dr. Burgdorfer's find "serendipitous" as he puts it, or the result of the familiar Louis Pasteur quote, "Chance favors the prepared mind."?

Was this discovery not supposed to happen? Was this new Borrelia species, possibly designed or weaponized at a facility such as Plumb Island, and was it being tested with the purpose of it being *undetectable* even to the top worldwide tick pathogens expert, Dr. Burgdorfer?

If an expert like Burgdorfer had failed to find this new spirochete, it would have indeed proved to be an excellent, undetectable, bio-weapon.

Dr. Burgdorfer has been described by some as not being party to any wrongdoing, and I firmly believe that he was likely an unwitting participant if indeed there was any ongoing secret operation of a weaponized Borrelia.

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FOLLOW THE MONEY

By 2005 EIS Officer Dr. Alan Barbour was placed in charge of the multi-million dollar, newly created bio-warfare facility at UC-Irvine in California. He serves as Director, Pacific-Southwest Regional Center of Excellence for Bio-defense and Emerging Infections.

A press release from Stony Brook News (Stony Brook Univ. of N.Y., Jan 19, 2011) lauds the many outstanding, esteemed, scientific contributions credited to EIS-trained Dr. Benach. His work focused on the area of spirochetal infections and vector-borne diseases, and his establishment of the Center for Infectious Diseases (CID). The CID has received major funding from NIH and NIAID and a 2009 grant renewal is ... "part of the NIH's 'Biodefense and Emerging Infectious Disease Research Opportunity' Program that will total $23 million by 2012".

EIS officer and bio-warfare scientist, Dr. Mark Klempner, was commissioned by NIH to investigate Lyme disease as a chronic persisting syndrome in some people. He, like the IDSA, concluded that persistent infections were non-existent. I can personally and unequivocally testify to that being absolutely false. If he truly believes that, he does not know the nature of spirochetes.

Dr. Klempner, a fellow EIS colleague of Drs. Steere, Benach and Barbour, became the recipient of a three million dollar bio-warfare research grant in 2004, a year after Dr. Benach was chosen to receive a three million dollar bio-warfare grant.

Dr. Durland Fish was another EIS officer that I knew during the 1980's when he sent some of his collected ticks to me from New York. When I visited microbiologist Julie Rawlings on a stopover flight in Austin, Texas, her boyfriend, an EIS officer, went to dinner with us. Julie warned me, "Be careful what you say about Lyme disease at dinner tonight because, Tom is an EIS officer." She did not explain why.

EIS officer Dr. Jacob Pinnas had supported our TBRF efforts in Arizona. He even went to bat for our support group and early on served as a mentor as well. For instance, he told me during the mid 1990's that the triatoma cone-nosed bug commonly known as "kissing bug" or "assassin bugs" could, through contaminated mouth parts, transmit Borrelia as well as Chagas disease, something very few people knew.

A few months ago I dropped by his home in Tucson. I had tracked him down after I caught a Reduviidae bug in my Arizona desert neighborhood and I needed an expert to identify it.

Dr. Pinnas recognized me immediately during my surprise visit to his home, although I had not seen him for twenty-eight years (since 1984) when Alice Holmgaard and I consulted with him at his University of Arizona facility.

I had contacted Dr. Pinnas after 1984 by phone and sent a letter to him in 1997 when I was bitten by a Riduviidae, Triatominae bug (aka conenose bug or assassin bug) at our home in Gig Harbor, Washington. It looked similar to the kissing bugs that Dr. Pinnas was familiar with in Arizona. But this family of bugs was not supposed to exist in Washington State, according to the physicians at the University of Washington whom I consulted when I became ill after having been bitten.

I sent the bug to Dr. Pinnas to identify since the experts at UW were unable to identify it. He got back to me with, "It looks like, but isn't a kissing bug." I sent bugs to Julie Rawlings in Texas and also to Dr. Paul

Duray at NCI who passed it on to someone named "Armando." None of them identified the specific species. Amando (or Armando) at NCI never sent my wax block specimens back to me as I requested. Of course by now I was quite used to researchers keeping my specimens for their own use and not sharing with me whatever findings they observed.

Finally, I sent one of the several bugs that I caught resting on our patio furniture on our upstairs balcony that overlooked the golf course, and two bugs found in our master bedroom to Dr. Burgdorfer. He said it was indeed a Riduviidae bug and he sent it on to a world expert, a "Dr. Nadelman" to identify the specific species. When I then told Dr. Burgdorfer that I developed a skin lesion within a month and my eye was swollen shut, (manifestations that occur with Chagas disease) after having been bitten by one of the bugs, he exclaimed with obvious concern, "Oh, no! You got BIT by that bug!?"

I never heard from Dr. Nadelman. Dr. Burgdorfer was puzzled by that because he believed that Dr. Nadelman would identify the species of this bug and contact me.

The next time I called Dr. Pinnas, he sounded flat and distant, as though he couldn't talk to me. This was so very unlike his former exuberant demeanor that was more than complimentary in a letter he had recently sent. It reminded me of the way Dr. Gay and others such as Dr. James Webb sounded when they were obviously under some sort of pressure from their superiors. And now, unlike all my previous telephone calls, it seemed whenever I called my former friends in the scientific community, I was consistently put on "speaker phone".

I wondered if I may have opened another can of worms with the discovery that assassin bugs were on the loose in the Pacific Northwest, a supposed non-endemic area. This bug had the potential to transmit disease agents such as Chagas, borreliae etc.; disease agents that could later cause serious neurological consequences among others. Had someone sent a directive to Dr. Pinnas to cease contact with me? I could only wonder, lacking any evidential proof of it.

Now, after all these years, I came face to face with Dr. Pinnas in 2011. He treated me, and my friend Paula who accompanied me, cordially although he seemed ill at ease with my questions. I told him of my continuing distrust and frustration regarding CDC. Then he said, "Perhaps if you got to know CDC better, you would not think it was so bad." Is it possible I have held this grudge unfairly for all these years?

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Dr. Pinnas said that he could not identify the Arizona bug I brought with us. It was not completely intact with my having squashed it a bit when I captured it. It contained blood. In any case, after visiting Dr. Pinnas, I came to the conclusion that, without the ability to learn anything new from our infectious disease experts, especially the Infectious Disease Society of America, and, many of whom are acting in lock step with chain of command public health directives to no longer communicate openly with the lay public, we patients will be left to seek answers on our own.

We are left to deal with the consequences of being exposed to non-identifiable toxins and pathogens. We must cope with the later disease manifestations without much help from the very people employed by our government agencies who are mandated by law to address communicable disease issues.

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LOOMING QUESTIONS REMAIN

Certainly, no proof of anything clandestine is evidenced by these coincidences. It is possible that all EIS bio-warfare scientists, or at least most of them, abide by the rules and their studies are designed for defensive purposes only. However, if there is any truth to the rumors and speculations about toxins and pathogens being purposely, or accidentally, dispersed amid animal and human populations from Plumb Island and elsewhere, it would explain some of the bizarre and downright hostile stances between agencies such as CDC, NIH, physicians, and the public.

Looming questions remain about what is rumored to be BW activity within our shores. Is it necessary for our defenses against foreign terrorist attackers who are threatening or have already planted these agents? Or, on rare occasion, is there careless incompetence, causing accidental escape from laboratories? Or, are there more sinister reasons?

As outrageous as it sounds, our government is not innocent of conducting appalling experiments, already having been accused of experimenting on animals and citizens within our own borders as well as on military personnel.

Historical examples of this are experiments that were carried out on blacks in the state of Alabama during the Tuskegee syphilis study, a Public Health Service study; and sheep deaths tied to the army's Dugway Proving Ground in Utah, a military facility that tests chemical and

biological weapons such as nerve gas and anthrax; and the required Memphis minority school children's hepatitis vaccines; and the CDC/Kaiser Permanente, Los Angeles minority children experimental vaccines for measles given without proper parental consent; and the Seventh Day Adventists' voluntary Fort Detrick studies, to say nothing less of radiation and vaccine experiments on military and civilian populations; and more. (See web sites and, "The Cold War's Dirty Secrets", Radiation experiments ignored ethics guidelines, Scientific American, May 1995).

The above-mentioned are not secret, and information about all of these can be accessed in libraries and internet searches as well as by pursuing the "Freedom of Information" archives.

Was the Lyme disease bacterium, whether designed or not to become a stealthy new pathogen, dispersed among our citizens on purpose via a foreign enemy or, heaven forbid, by our own scientists? Or, did it "accidentally" vent its way out of the not-so-air-tight, Plum Island, bio-safety level-three laboratories? The accidental release scenario has been well documented in the book "Lab 257" by Michael C. Carrol in regard to Plum Island. It appears to be so.

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One particular argument can be opined in favor of our scientists having not created or weaponized this new Borrelia pathogen. That argument being that *Borrelia burgdorferi* is not "new".

Historically speaking, once Borrelia was retrospectively studied at Montauk (Long Island) and elsewhere it was found to have been present for many years before Dr. Burgdorfer's discovery in 1981.

As repeatedly cited in this book, Dr. Gabriel Steiner discovered what he called *Borrelia myelophthora* in MS plaques during the 1950's. Borrelia and its hallmark erythematous rash had been diagnosed in Sweden in 1909 by Afzelius and/or others before him in Europe. "Montauk knee" had been an arthritis described since the middle and late 1960's, before it was "discovered" to be a hallmark of early LD. The Greek physician and Father of Medicine, Hippocrates described the clinical manifestations of Borrelia during his lifetime of c460—c377 naming it "ardent fever".

In November of 2010, an autopsy performed at the South Tyrol Museum in Bolzano, Italy, on the mummified body of the famous Neolithic "Iceman" ... "researchers found the genetic footprint of bacteria known as *Borrelia burgdorferi* in his DNA -- making the five

thousand-year-old Iceman the earliest known human infected by the bug that causes Lyme disease." (Ntl. Geographic, Nov.,2011, pp 126-132).

So, how new is Lyme disease really?

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The questions remain as to why physicians had never diagnosed their Montauk area patients as having Borrelia spirochete infections over the span of so many years when we now know it was there. Why were Dr. Steiner's and Rose Ichleson's MS research studies ignored? Is it possible that someone else within the scientific community had already discovered Bb and, because this *species* of Borrelia was almost undetectable in humans, it became a perfect candidate for bio-weaponry?

Is that why the excellent scientific works by researchers such as Steiner and Ichleson were scorned, lest attention was drawn to this new Borrelia species in a new tick vector?

Dr. Burgdorfer's discovery of *Borrelia burgdorferi* is of such magnitude as to rival or best all of the greatest medical discoveries since the beginning of science. It is because of his discovery that this elusive spirochete is now known to be capable of imitating or causing almost every chronic disease known to mankind. It has ignited research throughout the world, providing lucrative incomes to scientists, universities, pharmaceutical houses and more. In light of this, why is the real truth behind its controversies so difficult to discern?

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In retrospect, during the middle 1980's Lyme arthritis had been associated with multi-system health issues, likened to syphilis as the great new imitator of all disease. The discovery of *B.burgdorferi* had finally made it possible to treat patients with specific Borrelia-targeted antibiotics for their mysterious infection-caused complications that had gone unrecognized for nearly seven years.

This "new" Borrelia species was vectored by what was thought by some to be a new species of hard-bodied Ixodes ticks. The ticks were identified at Yale and named *"Ixodes dammini"* by Harvard epidemiologist (EIS officer) Dr. Andrew Speilman.

Years later it became controversial between Dr. Speilman and other tick experts that the older, long-recognized (since 1805) *Ixodes scapularis* ticks were one and the same as *Ixodes dammini.* Dr. Speilman disagreed,

citing that *I. dammini* is a northeast area variation of the southern states' *I.scapularis* species. The editors of the Journal of Medical Entomology ruled that these ticks should be ... "synonymized under the *Ixodes scapularis* name."

Conspiracy theorists speculated that this hard-bodied tick was altered or weaponized by scientists located at the nearby Plum Island facility or maybe Fort Detrick, and that this tick could vector multiple disease agents.

A tiny larval tick the size of a typewritten period could deliver not only Borrelia, but a whole variety of co-infectious human pathogens such as Babesia, Erlichia, over two hundred arboviruses, and more.

These pathogens could collectively cripple or even kill an enemy. Historically, that has been the purpose of bio-warfare, to cripple and weaken the enemy physically and financially.

Ticks have the potential to transmit a virtual soup of pathogenic and other toxic agents. Local area residents feared that Borrelia and other infectious agents could have escaped from the Plumb Island bio-safety level-three facility via birds that may have picked up ticks from the infected test animals caged outside in open labs.

The worn-out seals of air vents on the roof above the Plumb Island laboratories reportedly also gave access to escape routes for aerosol agents. Were these pathogens accidentally released through unthinkable incompetence? That seems to be the most likely case, or in a more sinister scenario, released intentionally? Either way, the consequences are frightening. (See Michael Carrol's book, "Lab 257".)

Some diseases as well as toxins such as anthrax are contracted by inhalation, ingested in food, some transmitted by insect or other arthropod-vectored bites, or via blood and body fluids, just to name a few possibilities. For instance, Hanta virus can be contracted by merely breathing in the dust clouds encountered while sweeping the floor clean of mouse droppings that contain the lethal pathogen. Likewise, inhaled airborne bird feces particles can cause histoplasmosis and many other illnesses. Countless illnesses can be spread via contaminated feces.

A major difference between the new Lyme disease Borrelia infection and the older louse and tick-borne relapsing fever Borrelia infections is that the older LBRF and TBRF versions have multiple times more borreliae spirochetes circulating in the blood at the time of fevers than does *B.burgdorferi.*

Relapsing fever is extremely difficult to detect unless it is suspected and blood smears are done during initial fevers that reveal spirochetes, so it is rarely diagnosed. But, the even lower yield of

Lyme disease spirochetes circulating in peripheral blood at the time of initial fevers makes it *ten times* more difficult to diagnose because it is estimated that ten times fewer spirochetes are present in the peripheral blood. That adds another plus to the elusive pathogen for use as a BW weapon.

It was widely known and admitted openly by Plum Island scientists that tick and other insect colonies were kept at their facility for bio-warfare studies. The *Borrelia Lonestari* hard-tick vector *Amblyomma americanum* was the only Borrelia species that the officials admitted to studying. Its habitat was supposedly located in the southern U.S. not Connecticut. Could southern ticks have been selected and genetically altered and bred specifically to be new unsuspected delivery-system vectors of diseases?

Had I stumbled, unwittingly, upon things that were of a top secret nature in Tennessee? If so, was that the reason that Dr. Burgdorfer was concerned over Daniel's strange but highly positive TBRF serology reaction? The southern Lone Star hard tick, *Amblyomma americanum* can vector several agents including rocky mountain spotted fever, Human Ehrlichiosis, Tularemia and American Q fever. Unlike the Ornithodoros soft ticks, this Ixodes species of hard ticks was not thought to be capable of harboring Borrelia until recently.

Could Daniel have had a mix of heretofore unknown pathogens and borreliae species to include the highly controversial lonestari infection that Dr. Ed Masters discovered in his Missouri patients? Daniel's test was positive for relapsing fever, *B.hermsii* not *B.burgdorferi. B.hermsii* cross-reactions are not documented to do so, but Master's disease *B.lonestari* and B. burgdorferi are recently known to cross-react on tests. The *B.burgdorferi* reactions are described as "weak."

After my tick study in 1989, *something* must certainly have caught the attention of the head of the Department of Health and Human Services director, Dr. Sullivan, who said, "No more tests for Bonnie Bennett..." The CDC conducted an investigation by questioning neighbors on our street in Germantown after we moved to Washington State, yet CDC never contacted me. After repeated calls to CDC I could never get an answer as to what their investigators learned in Germantown, Tennessee; strange to say the least.

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MOVING ON

In December of 2010 I wrote to the director of NIH, NIAID, Dr. Anthony Fauci. It was my last effort to request that someone qualified be selected to look into the complexity of chronic TBRF and our family's and friends' decades-old struggle with the sequelae of this tick-borne disease. It has never been done before even though the documented history of TBRF Borrelia predates Lyme disease by one and a half centuries, since 1854, when Dr. David Livingstone first associated the bite of the African tampan ticks with relapsing fever. I sent the following letter to Dr. Fauci: (See Fauci letter in Addenda)

I do not know if Dr. Fauci ever actually received or even read my letter because it was eventually answered by someone else who did not address what I had specifically requested. In the two and a half years since, I have not heard if anyone qualified is doing that research or not. It appears that my efforts since 1982 have all been in vain and no help is available to victims of chronic TBRF.

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After our recent visit with Dr. Burgdorfer in September of 2010, Karen and I have realized that we may never know all the answers to great numbers of puzzling questions regarding tick-borne disease, especially if the conspiracy theories of its involvement in biological warfare are true. Patients like my son Doug will continue to struggle with devastating consequences, unless things change within our public health community that no longer serves our citizens' concerns honorably.

The current battle between Lyme disease patients and the Infectious Disease Society of America over the question of chronic LD versus the hard to catch easy to cure opinion of the IDSA continues. The list of 301 scientific publications, in support of the "PERSISTENCE OF LYME DISEASE SPIROCHETE, *BORRELLA burgdorferi,* compiled by John D. Scott, Research Division Lyme Disease Association of Ontario, exemplifies the dire need to further prove beyond doubt the chronicity of Borreliae with new laboratory science and technology. Information about latest techniques and news can be accessed on line. See "Canlyme" as well as Alan B. MacDonald and Tom Grier sites.

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Dr. Burgdorfer warned me during the writing of this manuscript, "Did it ever dawn on you that all those guys who used to be so supportive of your ideas are now dead?" It was true; Charles Thornton, Dr. Paul Lavoie, Dr. James Webb, Dr. John Doll, Dr. John La Montagne, Dr. Byron Waksman and Dr. Paul Duray and others are now dead. I heard that Julie Rawlings is no longer doing Lyme disease work in Austin, Texas, but she worked for Homeland Security, and is now retired. I recently tried to locate Dr. Robert Quackenbush, Dr. Russ Johnson and Dr. Andrea D'Lesk without success. I was able to locate Dr. Ron Johns, PhD., who is now an M.D., Internal Medicine.

Karen and Tom have finally evolved to new careers, and closed their Lyme Disease Foundation after twenty-four years of service. They, and their daughter Christy, are moving on. When we talked in April of 2013 Karen said, "Someone else will have to pick up the torch and run with it".

I am finally moving on too, after watching this insidious TBRF disease complex progress, unchecked, as it continues to destroy the health of our family and others who have not received much help from our public health officials for the past thirty-plus years. Our family, friends and others are still coping with serious, chronic, TBRF health issues, and doing the best we can on our own. I hope someone reading this book will take up this cause and run with it. It is time for me to say goodbye.

I didn't tell you everything.

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AFTERWORD

By Tom Grier

We've known for a long time that patients with Lyme disease (LD) are often misdiagnosed as having multiple sclerosis (MS). Part of that is when we look at magnetic resonance images (MRI's) of the brains of Lyme patients, we see lesions in the brain that look quite similar to those seen in MS patients. But, we have not looked closely enough in the past. Now that we have more data to sort through we have found some patterns that are interesting and important.

It is considered by MS researchers at Mayo Clinic (*most notably work by Claudia Lucchinetti, M.D. at Mayo Clinic)* that since December of 2011, all MS starts with inflammation of the micro-vessels in the gray matter of the brain. We rarely find this inflammation in the white matter of the brain.

Coincidentally, in Lyme patients we only find the "classical spiral-shaped form" of spirochetes in the gray matter of the brain. Also associated with the gray-matter inflammation is inflammation in the subarachnoid space. (*A fluid filled space that cushions our brain from injury)* It is only much later that we see the resulting white-matter lesions near the central regions of the brain.

In the central region, that is where we have the white matter and heavily myelinated area of the brain and, for whatever reasons the spirochete chooses to manifest itself in a different form in the matter, it will take on a cyst-like or granular form which was described as far back as 1922 by researcher, Gabriel Steiner, MD in Germany and, many researchers along the way including LD researchers.

When we look at multiple MRI's of brains of LD and MS patients we start to see a pattern emerge. Those that have had MS for a very long time tend to have enlarged central ventricles in the brain, bases in the brain that are filled with cerebral spinal fluid. In the lining of those bases is a membrane, the *ependyma membrane.*

The difference between LD patients and true MS patients is that we often see enlarged central ventricles and inflammation of the ependyma lining and, white matter flares around the ventricles. We know that this is Lyme disease because, often with antibiotic treatment these white matter flares will get better and we will see a reduction in the pressure because, most of these patients report feelings of increased pressure in the head

and, quite often upon the withdrawing of spinal fluid during a spinal tap, patients report feelings of reduced cranial pressure.

Although many MS patients complain of feeling pressure in the head, this is not really an MS symptom. It is, however, a marker for LD. So the markers that we are looking for are; enlarged central ventricles, white matter flares around the ventricles, inflammation of the ependyma lining of the ventricles, increased intracranial pressure, and then we look for Borrelia antigens in the spinal fluid which may or may not be there.

The patients will often respond to antibiotic therapy even though we can't find positive serology or antigens in the spinal fluid. But, usually in these cases if we treat the patient with a bactericidal antibiotic like Rocephin or amoxicillin at a very high dosage for a week or two and we collect their urine for a week and, then we test their urine for bacterial proteins we often are able to use antigen capture tests and find that the patient has active infection that is now being sloughed off through the urine but, their serology tests are negative.

That means that this infection that is literally trapped in the brain is not eliciting antibody response in the patient and, that our serology tests are no longer capable of detecting an infection like LD that has sequestered itself in the brain of a patient for many, many years. This demonstrates one of the dangers we have with current testing when the Centers for Disease Control (CDC) call their two-tiered tests the "gold standard" of Lyme tests. But, it is not a very good standard if it cannot detect sequestered infections in the brain. These brain infections are the most important of all.

It is important that we open up discussions and start designing research that will give us better answers. One of the things we have learned during brain autopsies is that we, in fact, find spirochetes in the brain of some MS patients and Lyme patients and, there are changes. The bacteria seem to take on different forms in different tissues of the human body. We have to be cognizant of that change, the ability of these bacteria to change. Unfortunately, the medical community seems unable to change so we have a very difficult task ahead of us in trying to get this research done.

The big question then in Lyme patients that mimic MS is how and why do the bacteria enter the brain. We have known since the late 1980's that the bacteria, when they attach to blood vessels very quickly, cause the endothelial cells to become leaky and the bacteria will travel right through blood vessels including the blood brain barrier. Once it enters the brain, one of the first tissues it encounters is the cortical regions of

the brain, or the gray matter. This may be why medical institutions like the Mayo Clinic recognize that MS in its very earliest stages starts in the gray matter with inflammation. In our model we actually know what the inflammation is from. It is from the introduction of the bacteria into the brain.

Once it is in the gray matter of the brain it is in close proximity to the cerebral spinal fluid channels including the arachnoid space. And, once the bacteria, accidentally, enters the arachnoid space or the cerebral spinal fluid channel it is in a medium that the bacteria does not favor; it does not like it. So, it is going to try to find its way out of those areas.

As it enters the central ventricles of the brain, the first tissue it encounters is the ependyma lining, the membrane. So, now we see the bacteria trying to escape the spinal fluid and the first tissue it encounters is the ependyma and this introduces the bacteria to the central regions of the brain where the white matter is located. And, of course, over time the "marker" for MS is white matter lesions. We see this also in Lyme disease.

What we think is, the bacteria have originally entered through the gray matter and then resurfaced in the central region's white matter of the brain. So, by the time you see these lesions you are really looking at a late, late stage of Lyme disease. By this time, the blood brain barrier has long-since sealed itself back up, the bacteria is sequestered and, there is no immune system defense available in the brain to fight this infection. Furthermore, the peripheral immune system is no longer going to make antibodies. While we can treat the peripheral system, the blood stream, with drugs like doxycycline to eradicate the infection there, the infection that is trapped in the brain will remain and will constantly change.

One study that was done in 1989 by neurologist, Andrew Pachner, MD, showed that the bacteria changed so quickly in the brains of mice that the antibodies from the mouse's blood no longer recognized the infection in the brain. This is very significant because, what it tells us is that when you close off the infection, the immune system is only going to keep up with that part of the infection that it is in contact with. And, since the brain is shut off from the immune system that infection continues to progress and the immune system, has no way of producing antibodies that recognize the infection. This becomes a real problem for the patient as well as for tests to detect persistent infections in the brain. How do we detect this infection in the brain that has changed so significantly and, does it rather quickly? In mice, it only takes about four to six weeks. In humans we just don't have the data to know what we are

really dealing with. We have to start doing more brain autopsies to get to the truth of this disease.

Lyme disease, *Borrelia burgdorferi*, is really a part of the extended family of relapsing fevers of which we've known about since the late 1800's. One thing we knew about the old world relapsing fevers was that they were tick-borne in nature. Ornithodoros (soft shelled) ticks transmitted the agents of Borrelia, the cause of relapsing fevers. Several kinds of relapsing fevers were very neurogenic, they entered the brain very quickly, could cause encephalitis and kill patients. Some of these relapsing fevers could linger in the brain. One study done in 1945 by Dr. V.T. Schuhardt, (SCIENCE, 1945) showed that once the relapsing fever (TBRF) agent of infection *Borrelia turacata* entered the brain of rats, it was incurable even by injecting penicillin directly into the brains of rats. They did not have good tools for detection so it required a lot of work to show that the bacteria were still there and still active.

Two years later, they tried to cure the rats again with the infection in the brain. What they found was that you had to treat the body of the rat with injections of penicillin and injections directly into the brain at the same time with huge, heroic, doses. Two out of every seven of the rats died from this process and they had to give the injections every three hours. In using the tools they had at the time they determined that they had eradicated the infection.

Using the more modern tools that we have now, it might show that if the rats lived another two years and then were tested with the tools of immunofluorescence and antibody tests, it could be shown that the rats were not cured. We simply do not know because, we are not doing that kind of research anymore, but, we certainly should be.

The idea that TBRF and other relapsing fevers as well as LD can mimic multiple sclerosis or induce changes in the brain is really a possibility because their species and strains of Borrelia are so closely related to one another, much closer than the medical community would have us believe.

Instead of isolating differences of *B burgdorferi* and and other Lyme species from relapsing fevers, government-paid scientists used restrictive gene groupings that looked at just one genetic parameter: the 16s ribosome protein. These new guidelines showed that the Lyme grouping was slightly different from all relapsing fevers on paper. This new "Lyme" group was further divided again by introducing the confusing and unnecessary nomenclature of "Senso strict and Senso lato" Lyme species which now have become archaic expressions of trying to contain

nature in proper little boxes. Yet nature keeps finding ways of avoiding containment, and one relapsing fever species *Borrelia miyomotai* (which has many characteristics of Lyme disease and is transmitted by the same hard shelled Ixodes species) is uncomfortably out of place on the genetic phylogenetic tree for either relapsing fevers or the Lyme group.

What is disturbing is that no matter where a Borrelia species falls in a table or genomic ranking, what is not being addressed is how to test for all of these different species of neurogenic Borrelia that collectively are now a pandemic throughout the Northern Hemisphere.

Regardless of a name or grouping, or scientific semantics, it is the detection and treatments of all types of Borrelia that determines if a patient will overcome the illness, or if the illness will overcome the patient.

"*Borrelia miyamotoi* sensu lato, a relapsing fever *Borrelia* species is transmitted by the same ticks that transmit *B burgdorferi*, the Lyme disease pathogen," and it occurs in all Lyme disease-endemic area of the United States, but a new study suggests that *B burgdorferi* antibody testing is not an effective surrogate for detecting *B miyamoto* senso lato infection. (See *Borrelia miyamotoi senso lato seroreactivity and seroprevalence in the northeastern United States.* Krause PJ, Narasimhan S, Wormser GP, Barbour AG, Platonov AE, Brancato J. et al. *Emerging Infectious Diseases.* 2014 July 6 (located on the internet, May I, 2014).

This is not the first and probably not the last time we will see relapsing fever and Lyme disease species overlap. Unless current testing changes to pick up all pathogenic Borrelia species, we are likely to see many more sero-negative cases of illness caused by Borrelia other than *B. burgdorferi,* just as many patients who test negative for *B miyomotoi* go undetected and untreated for lack of an effective surrogate Borrelia test.

"Despite being transmitted by a tick-borne vector, despite relapses being common after antibiotic treatment, despite similar microbiology of blood vessel penetration, and despite similar pathologies of having a tropism for brain and nerves; microbiologists would again distinguish Lyme disease from all relapsing fevers. Before the term "Lyme disease" was ever uttered, the medical community knew certain things about Borrelia bacteria that should have been a harbinger of problems associated with any new disease caused by the bacterium."

Tom Grier

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Bonnie Bennett
37841 South Skyline Drive
SaddleBrooke, AZ 85739

August 8, 2009

R. Lanny Hunter, MD and Karen Apodaca, MD
Arizona Dermatology 450 S. Willard St. Suite 115
830 Ainsworth Cottonwood, AZ86322
Prescott, AZ 86302

Attention: Drs. Hunter and Apodaca,
Re: Walter 'Alan' Cressey

Enclosed, you will find the supportive literature information we discussed yesterday on the telephone regarding my antibiotic treatment views for my father-in-law's Cutaneous T-cell lymphoma. I told Alan that Dr.Apodaca has ordered doxycycline to get started and that I will send him copies of this letter so he can make up his mind if this treatment is worth a try. As the two of you and I agreed, Alan is mentally intact except when he is under heavy narcotic type medications but, when alert, he is very capable of making all of his treatment choices wisely. We also agree, as does Alan, that bone marrow studies and other exhausting tests are not advisable for him in his present, frail, condition. My husband and I were shocked to see how bad he looked last week during our visit in Camp Verde. I believe 4 months of antibiotic treatment is worth trying.

My familiarity with lymphomas began in 1977 when I was diagnosed as having some type of "cutaneous lymphoma." This diagnosis was further described in a second opinion from dermatologist, A. B. Ackerman, MD as being, "Probable mycosis fungoides." I communicated with Dr. Ackerman intermittently over a period of 25 years and, after locking horns with him initially (see letter enclosed) he later became quite friendly and even invited my husband and me to join him for a friendly dinner 'discussion' if ever we were in his area.

In 1977 I was unaware that the vacation townhouse we had purchased at Lake Tahoe in 1976 was highly endemic for Borrelia hermsii. After becoming aware of this fact by 1982 and researching borreliae and related spirochetal infections, I retrospectively connected the dots to Borrelia being a possible incitant of lymphomas and/or pseudo-lymphomas. Thereafter, I believe periodic courses over the years of tetracycline and doxycyline have held my, lymphoma/pseudolymphoma at bay.

One cannot rid the body of spirochetes once infection has occurred, because their nature is to relapse and remit over a lifetime and it only takes "one" surviving spirochete to start an entire new colony.

Most people don't suffer from noticeable or debilitating sequelae. But those who do get later complications, desperately, need help. Protective immunity is dependent on a wide variety of factors including; innate immunity, childhood immunity, genetic chromosomal variations and markers to name a few, plus, co-infections, as is especially true with initial arthropod bites that can transmit agents of potential human disease that include over two hundred arboviruses as well as bacteria, Rickettsia, Babesia, Eherlichia, and more.

The important factor with Borrelia parasites is that they have not changed much in the past 3.4 billion years, yet they have evolved and adapted, and for the most part and symbiotically branched out to ever changing environments through clever antigenic alterations and slight differences in morphology. In human borrelioses, once threatened by adequate immune responses or normal courses of tetracycline and other appropriate antibiotics, borreliae quickly break up and sequester into protective areas of the body, and only emerge as new colonies when conditions are favorable. This demonstrates how ludicrous it is to treat Borrelia infections after the relapse is over during quiescent phases.

It could very well be that other co-infecting tick pathogens should be addressed once relapses of borreliae remit. However, borreliosis should be treated *during* every relapse or exacerbation of symptoms if patients are suffering from chronic autoimmune type conditions where adequate protective immunity is compromised. I think, initially three weeks to one month of doxycycline once or twice daily is enough, thereafter, ten days to two weeks for each relapse which, in my experience, usually amounts to about three times per year.

It is my hope that we can come up with a plan that will work well for my father-in-law. I believe that his culture-positive infections are acting collectively to shoot this strong man down, and may well be among the factors that have precipitated his lymphoma, or possibly are opportunistically taking advantage of his weakened condition. In either case, they are part of his clinical picture and if treated, maybe he can regain some strength and enjoy life once again for many months or years to come.

The Epstein-Barr virus is often thought to be causal to many diseases such as multiple sclerosis, adenoid cystic carcinoma, and of special interest, Burkitt lymphoma. However, EBV is a universal disease agent so it does not make sense as the single cause and effect when it occurs in clusters. But, if other disease agents, namely malaria parasites, can impair specific immune responses that allow for an increase in the numbers of EBV carrying B cells in the circulation, as is precisely the case with sub-Saharan clusters of Burkitt lymphoma, a variety of other disease agents causing human infections could be factored into the equation and investigated as possibly affecting T cells in like manner.

Hypothetically, perhaps the tick-borne protozoan, Babesia, can dysregulate the immune response as a co-infectious agent with Borrelia etc. and incite other types of lymphomas.

Malaria, a protozoan infection, paired up with EBV, has already been demonstrated as one of the partners in crime for Burkitt lymphomas. The lymphomas only occurred in cases where both malaria and EBV were present. Many more infectious or parasitic agents need to be investigated in other types of malignant lymphomas and cancers. Of interest, during the early days of testing for Yale, Univ., Dr. Louis Magnarelli had a very difficult time distinguishing between positive Borrelia and EBV serology because they cross-react.

It is my guess that by treating Alan with doxycycline for about three or four weeks, he could benefit from addressing not only the broad spectrum bacteriostatic infection-fighting property of this low-dose course, but from some immune modulating and anti-inflammatory benefits as well.

My next best *guess* would be to try treating for his culture-positive Staph-aureus with vancomycin. I have a feeling that his longstanding staph infection may be drug-resistant unlike borreliae that are rarely drug-resistant. "S. aureus has been consistently identified as the most common pathogenic organism recovered in cutaneous infections and bacteremias in CTCL patients." (See enclosure, "Infectious Complications of Cutaneous T-Cell Lymphoma: Discussion") This discussion also includes pseudomonas. We can provide acidophilus supplements as no doubt his treatments will kill off his natural flora. And if nystatin is given at some point this can hopefully be kept in check.

Alan has been rapidly going downhill within the last few weeks so, I thank you both for your interest, care, and kindness in honoring my wish for you to offer Alan this viable anti-microbial treatment approach choice, rather than the oncology-based treatment modalities that have not proven to extend life (see enclosed literature) and that would only serve to make him feel worse and likely kill him.

Sincerely yours,

Bonnie and Dick Bennett
Enclosures

Bonnie Bennett
37841 S Skyline Dr.
Tucson, AZ 85739

December 30, 2010

Anthony S. Fauci, M.D., Director NIAID, NIH
31 Center Drive #7A03
Bethesda, Maryland 2089-0001

Dear Dr. Fauci,

My family, friends and others have been suffering the later effects of *tick-borne relapsing fever* (TBRF) for nearly 30 years. Later effects following TBRF have never been studied, documented or published in medical literature. In years past I cooperated with several knowledgeable researchers such as Paul Duray, MD by sending sera and other specimens that he could examine at NCI. Now that he and others have retired, I am asking if you could take a look at this and possibly find a highly qualified, unbiased, researcher who would be interested in receiving such specimens.

My husband's atheromatous mass and tissue blocks have been preserved at my request following his left carotid endarterectomy six weeks ago at Barrow Neurological Institute in Phoenix. BNI has an excellent Human Specimen Procurement Service and can provide specimens of my husband's "snap frozen" excised tissue, wax block, brain scans etc. to qualified researchers. My nephew's brain tumor from 1994 may also be available. He had TBRF in 1984 after a ski vacation in Tahoe, two years after my husband's TBRF.

The propensity for spirochete bacteria such as borreliae (like Chlamydia pneumonia) to *nest* in the walls of blood vessels as well as vessels of the brain offers a guilt by association causality of aneurysms,atherosclerosis, tumors, and many other later effects similar to those of late syphilis. I hope my husband's specimen can be put to good use for TBRF research as his case was well documented in 1982 with demonstration of B hermsii on blood smears and my nephew sero-positive for B hermsii.

In 1983 when I first learned of B. Burgdorferi, I initially assumed after reading Oscar Felsenfeld's monograph, regarding all borreliae as a single entity, that *Lyme disease* was nothing more than a newly recognized tick vector capable of transmitting a newly described species of TBRF. I was told early on by Dr. W. Burgdorfer and others that TBRF and LD are two 'different' diseases.

Two years after my husband's TBRF illness, journalist Charles Thornton published an account of our medical ordeal on the front page of the Arizona Republic newspaper, May 20, 1984. The paper received more calls from that article than it had ever received for any story. It resulted in an organized local support group whose members totaled over 200 cases. During those years it was evident that people suffered from chronic health issues from what appeared to be various syndromes developing within weeks, months, and years following arthropod bites and exposure/s to body fluids from animal reservoirs or other modes of infection including sexual and transplacental transmissions.

Our group, not knowing what to leave out, documented over 100 symptoms and signs believed to be linked to Borrelia. Among them were certain cancers including lymphomas, brain tumors, breast, prostate and much more. We documented six brain tumors (see enclosure) within our support group of about 200 patients. We had three cases of diagnosed multiple sclerosis in our Tahoe townhouse, four-unit building. That became my primary cause for concern.

There were three cases of cutaneous, T-cell lymphomas or pseudolymphomas in our immediate family members, my husbands's father, step-father and me. (See step-father-in-law letter enclosed). CTCL was rare statistically; about one or two per million. Likewise, the three MS cases from our townhouse are over the top, especially when I predicted in 1982 that TBRF could later cause MS. Published; (Arizona Republic, Family Circle magazine, Sacramento Bee, scientific newsletters, and more). When my son was diagnosed with MS, over a decade later in 1995, Dr. Paul Duray (NCI) called to interview him.

A record epidemic-level of three MS cases within 100 feet of our four-story, four-unit townhouse building calculated out to be "one in ten" residents developing MS within ten years following TBRF exposures. Our only other son has evidence of brain damage on SPECT scans similar to late syphilis and neuro-LD.

In 1984, Byron Waksman, MD, National MS Soc., helped me do a serological study with his hand-picked neurologitst's selected "true" MS patients at the Univ. of AZ. Two of eight patients (25%) were positive for Borrelia in a non-endemic area. Dr.Waksman turned over my Borrelia/MS study reports, as well as our other correspondence to the neurology department at Yale, University.

In 1984 when I was told by Dr. John Doll and others that a group of six local Phoenix health-department men contracted TBRF (B. turacatae) after spending a night in a Tucson bat cave, I suggested to one of the raconteurs telling me the story that one of the victims would likely have MS in the years to come. By 1990, one of them Mike Wright, was bedridden with MS.

Dr. Anthony Komaroff the Director of Internal Med. At Harvard's, Brigham and Women's Hosp., wrote to me in 1987 to inquire about TBRF in comparing it with his much publicized, chronic Epstein Barr virus (CEBV) studies in New England and what was thought to be an epidemic of it at Lake Tahoe. I was convinced that the Tahoe epidemic was classic for TBRF but, after reading Hillary Johnson's documentary book, "Osler's Web" I could also see that TBRF was never recognized during that intensive study.

Following our mutual correspondence, Dr. Komaroff compared his patients with our Phoenix group clinically and serologically. He concluded that his group had not had TBRF as they had a different complex of symptoms. I did not agree. I thought his comparative study in collaboration with Dr. Alan Steere was lacking in more than one respect and it did not prove or disprove anything. For one thing, they did not have antigen from Tahoe strains of borreliae. He said that in the future if they found high rates of "positives" they might well come back for more information from our group. Two years later in 1989, on page 357 of the book "Osler's Web" Dr.

Komaroff is quoted as saying, "I always believed this disease was connected in some way to lupus and multiple sclerosis." In October of 1996 I wrote to tell Dr. Komaroff that my earlier predictions of MS following TBRF had come true because my son had now been diagnosed as having MS about ten years after his tick feves of 1985. Dr. Komaroff wrote back to tell me that he had turned over all my information to Dr. Eric Logigian.

In the fall of 1989 while living in Germantown, TN, a locale presumed to be non-endemic for LD and TBRF, I instigated a serology study in our immediate neighborhood. The study revealed that a large percentage (5 of 30) of our neighbors had indeed been exposed to antigenic components of Borrelia. The study was prompted after a neighbor's baby nearly died with what was thought to be "hemaphagocytic-lymphohistiocytosis." Physicians at Le Bonheur's Hospital in Memphis wanted to administer toxic, experimental drugs when they professed that the baby "was going to die anyway." The parents refused. Dr. W. Burgdorfer's positive TBRF serology report miraculously served to save the baby's life at the eleventh hour. With appropriate antibiotics, the baby dramatically revived over night. After we moved to Seattle, our former neighbors were interviewed by CDC.

In addition to my husband's specimen at BNI, if needed, I can supply more detailed information regarding the many cases of TBRF that I have followed over the past 28 years. I hope that scrutiny of wartime (war fever) borreliosis case records and excised surgical specimens stored in such places as Walter Reed Hospital can be retrieved under your guidance to build research data for the implicated long term sequelae of human cases that have required consequential treatment/s at VA hospitals.

With my own eyes, I have seen convincing evidence of financial hardship, chronic suffering and early death among friends and family who have had histories of TBRF. There is no medical literature to support diagnoses or treatments of chronic TBRF. So, physicians are at a loss to help. It is tragic. My objective is to encourage research that will directly help the victims within our family to obtain treatments and, to protect future innocent citizens

from these devastatingly dangerous pathogens. I hope you will make this possible.

Sincerely yours,
Bonnie Bennett

(Author's note) The above letter was originally scanned to include within this text but the print, when reduced to fit this book's page dimensions, was too small to read. The above letter was re-typed from its original 3 pages for easier readability.

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GLOSSARY: pg 210 is blank

acidophilus: beneficial bacteria
ADHS: Arizona Department of Health Services
aerobic bacteria: grow in the presence of oxygen.
ALS: *See amyotrophic lateral sclerosis.*
anergy: alteration of immunity causing diminished responses to antigens.
amyotrophic lateral sclerosis: (also known as Lou Gehrig's disease) a neuron disease of the brain-stem and spinal cord.
anaerobic bacteria: grow in the absence of oxygen.
antibody: an immunoglobulin molecule that responds defensively to antigens foreign to the body.
antigen: a foreign protein or toxin that can induce a specific immune system reaction.
Argasidae: (family of soft-bodied ticks) transmit agents of tick-borne relapsing fevers, pathogenic to humans and certain animals.
arsenical: a drug containing arsenic.
arthropod: invertebrates such as ticks that have no backbone. (no vertebrae)
autoantibody: antibody that reacts against body tissue (self) as though it were a foreign threat.
autoimmune disease: A classification for diseases such as arthritis, lupus, MS and more, where the body seemingly launches an immune attack on its own tissue (self).
Avonex: A pharmaceutical drug designed to treat MS.
Afzelius, Arvid, MD.: Swedish physician, was first to describe the classic, circular rash of tick-borne Borrelia in 1909.
bacterial disease: pertaining to bacteria that are pathogenic to humans and animals.
bactericidal: something that kills bacteria.
bacteriostatic: something that prevents bacteria from growing.
bacterium: unicellular (single cell) microorganism, some bacteria are pathogenic to humans and animals.
Betaseron: a pharmaceutical drug, interferon beta-1b, designed to treat MS.
blastomycosis: a fungus infection of the lungs and organs.
blastomyces: genus of fungi(yeasts) pathogenic to humans.

Borrelia: genus name for several species of spirochetes that cause diseases in humans and animals, *relapsing fevers, Lyme borreliosis.*
Borrelia burgdorferi: A vector-borne spirochete microbial agent, the cause of Lyme borreliosis, the new species of Borrelia discovered in 1982 by W. Burgdorfer, PhD.
borreliae: plural form of Borrelia species and strains.
borreliosis: infections caused by several species of borreliae such as tick-borne relapsing fever, louse-borne relapsing fever and, Lyme borreliosis.
bubonic plague: one of several types of serious infections caused by the bites of fleas that vector the agent, Yersinia pestis, from its rodent reservoirs to humans.
CDC: *See Centers for Disease Control and Prevention.*
Centers for Disease Control and Prevention: A branch of the federal government's public health service charged with disease control and prevention. It is based in Atlanta, Georgia, USA.
Chlamydophila" (formerly Chlamydia) C Pneumoniae, C psittaci, and C Chlamydia trachomatis species can all cause pneumonia in humans.
CEBV: *See chronic Epstein-Barr virus.*
chronic Epstein-Barr virus: a herpes-like virus that causes infectious mononucleosis and can become chronic.
It is associated with Burkitt's lymphoma and nasopharyngeal carcinoma.
Ceftriaxone (Rocephin): a broad spectrum cephalosporin antibiotic.
cervicalspine: neck area of the spine.
C-myc gene: a protein involved in immune system cellular responses.
CTCL: *See cutaneous T-cell lymphoma.*
cutaneous T-cell lymphoma: a rare type of lymphoma identified when cancerous lymphocytes affect the skin.
degenerative vertebral disc disease: degeneration of the fibrocartilage and pulp-like center of the ring-like structure between vertebrae in the spinal column.
demyelination: destructive loss of the myelin nerve sheath.
dermatopathologist: An MD who performs microscopic diagnostic pathology of skin disorders.
Doxycycline: a broad spectrum antibacterial drug of the tetracycline group. *See minocycline.*

ehrlichiosis: potentially life-threatening arthropod-borne infections caused by Rickettsial species, infects white blood cells. Two main types of human disease are human granulocytic anaplasmosis (HGA)(formerly named human granulocytic ehrlichosis) (HGE) and, human monocyticehrlichiosis (HME).
EIS: *See Epidemic Intelligence Service.*
Epidemic Intelligence Service: An esoteric group of officers trained by the Centers of Disease Control and Prevention to investigate diseases regarding environmental health and to respond to epidemics domestically and internationally.
encephalitis: general term for several types of brain inflammation.
endogenous retrovirus: Human endogenous retroviruses are inherited RNA protein particles that are thought to play a role in certain cancers and autoimmune diseases.
etiology: the source or origin of diseases.
epididymitis: infection or inflammation of the cordlike posterior part of the male testis.
Glatiramer acetate (Copolymer 1, Cop-1, Copaxone): a pharmaceutical drug for treatment of MS.
glioblastoma multiforme: a highly malignant brain tumor.
Grave's disease: a thyroid disorder.
Hashimoto's disease: a progressive thyroiditis.
hemolymph: blood-like fluid in invertebrates such as arthropods.
hemophagocyticlymphohistiocytosis: a rare, potentially fatal, multi-systemic febrile disease that features overactive histiocytes and lymphocytes.
HGE: See human granulocytic Ehrlichiosis.
histoplasmosis: a yeast infection of the lungs that is caused by inhalation of fungi spores.
interferon: a class of small soluble proteins produced in the body as part of a defensive immune response to certain viral or bacterial infections. Pharmaceutical designed medicines such as injectable interferon beta (Avonex, Betaseron, Extavia and Rebif) resemble the natural interferon produced by the body.
Ixodidae: (family of hard-bodied ticks) the genus Ixodes as well as the genus Amblyomma transmits agents of Lyme disease. Other hard-bodied ticks such as Rhipicephalus and Dermacentor have been occasionally associated with Lyme disease.

Ixodes: genus name for several species of hard-bodied ticks that are vectors of Lyme borreliosis microbial agents ticks such as I. scapularis, I. pacificus, I ricinus.
***Ixodes dammini*:** (thought to be a new species in the mid-1980's but later revealed to be *Ixodes scapularus*). Other similar species include *Ixodes ricinus* and *Ixodes pacificus.*
Jarisch-Herxheimer reaction: a dangerous, shock-like reaction and temperature elevation that can occur within two hours following antibiotic treatment of patients during febrile periods of certain infections such as syphilis, relapsing fever and Lyme disease.
LBRF: *See louse-borne relapsing fever.*
leptosporosis: infected with *Leptospira.* The agents of the infections are transmitted to humans by dogs, swine, rodents, contaminated water, especially from stagnant water. Leptosporosis can cause lymphocytic meningitis, nephritis and hepatitis.
louse-borne relapsing fever: a febrile illness caused by the crushing of lice (not bites)that contain the infectious agents, *Borreliae recurrentis,* to enter the skin and blood through the pores and scratched lesions of human hosts.
lupus: primarily a skin disease but can be multi-systemic.
Lyme arthritis: Named for the arthritis that was observed in local residents, especially juveniles, around the town of Lyme Connecticut in 1975.
Lyme disease: Once patients were observed to have multi-systemic disease manifestations by the later 1970's, Lyme arthritis was changed to Lyme disease. In 1982, an infectious primary cause (A Borrelia spirochete) was discovered by the Swiss-born scientist, W. Burgdorfer.
lymphoma: Malignancies of lymphoid tissues based on cell types, various categories are subdivided into nodular and diffuse types.
magnetic resonance imaging: an imaging test using strong magnets and radio waves to produce images of the body.
malaria: a disease of the red blood cells infected with protozoa, parasitic, microbes. The disease agent is transmitted by the bite of anopheles mosquitoes.
memory cells: T-cells and B-cells that play a major role in immunity.T-cells attack pathogens, B-cells produce antibodies that can inhibit or kill pathogens.

microbes: Tiny living organisms to include fungi, bacteria and protozoa.
microfilariae: the prelarval stage of threadlike pathogenic worms from the superfamily of nematodes, *Filarioidea.*
minocycline: a broad spectrum antibiotic belonging to the tetracycline group, used often as a drug of choice to treat Lyme disease and relapsing fever.
mononucleosis: Infection caused by the Epstein-Barr virus. It is also called "mono" and "kissing disease".
MRI: *See magnetic resonance imaging.*
MS: *See multiple sclerosis.*
multiple sclerosis: a disease that causes patches (plaques) of demyelination, primarily in the white matter and sometimes the grey matter of the central nervous system. The cause and cure are unknown.
mycosis fungoides: a type of cutaneous T-cell lymphoma.
myelopthera: a borreliae species (Borrelia myelopthera) named by MS researcher Gabriel Steiner, MD, who discovered during biopsies the spirochetes "in and around" newer MS plaques, but not in or around the... "old burnt out plaques."
National Institutes of Allergy and Infectious Disease: a National Institute of Health (NIH) U.S. government-funded research agency.
National Institutes of Health: A U.S. government- funded research agency that includes twenty-seven institutes under the umbrella of the U.S. Department of Health and Human Services.
nematoid: threadlike parasitic worm.
neuroborreliosis: a disease of the brain and spinal cord within the central nervous system, caused by various species and strains of Borrelia.
NIAID: *See National Institutes of Allergy and Infectious Disease.*
NIH: *See National Institutes of Health.*
Ornithodoros hermsi: soft, nocturnal ticks, the vectors of *Borrelia hermsii,*
Ornithodoros turacata: soft ticks, the vectors of Borrelia turacatae.
Ornithodoros coriaceus: the "pajaroello" soft-bodied tick of Texas, New Mexico, Arizona, California and Nevada. It transmits a venomous toxin, one of several causes of tick paralysis in humans and some animals. It is capable of transmitting Borrelia.
parasite: something that lives off or within another living organism such as certain viruses, bacteria and fungi.

parasite fantasy disease: a label suggestive of a psychiatric condition that is sometimes attached to patients who insist they are infested or infected with parasites when no parasites are in evidence.
pathogen: an organism/s that can cause disease such as bacteria, viruses and fungi.
psittacosis: a respiratory and multi-systemic disease caused by a species of Chlamydia that is transmitted to humans from birds such as parrots, pigeons, ducks and other fowl.
rickettsiae: microorganisms that resemble both viruses and bacteria. These widely divergent microbes are usually transmitted from birds to humans by inhalation of contaminated airborne particles, and/or by arthropods such as ticks and fleas. The three main groups of rickettsiae are *the spotted fevers (15 rickettsioses)*, the typhus group, and the scrub typhus group (different from the spotted fever and typhus groups).
RMSF: *See Rocky Mountain spotted fever.*
Rocky Mountain spotted fever: a serious infection, the agent, *Rickettsia rickettsii* can be transmitted by several different species of ticks to humans.
serology: Studies of sera (clear portion of blood) to determine antibodies that are usually, but not always, detectable after exposure/s to pathogens. Due to variability, such studies can support but do not accurately determine positive or negative, definitive, diagnoses without the further support, or lack of support, of a clinical diagnosis. Serology alone cannot rule in or rule out a negative or positive diagnosis.
Society for Vector Ecology (SOVE): Founded in 1968 by scientific and medical professionals in California who were working in the fields of vector biology, the group has an international membership. The society publishes a journal, newsletter, and sponsors annual conference meetings.
spirochete: a spiral, corkscrew-shaped bacterium.
syphilis: a disease caused by spirochetes (Treponemapallidum). It is sexually, intimately, and congenitally transmitted.Its primary, secondary and late stages include an initial chancre skin lesion, and progressively involves the lymphatics, multi-systemic complications of the organs, tissues, skin, bones, joints, cardiovascular and central nervous system.

tabes: wasting away of the body, there are various types such as cerebral dementia paralytica, cervical, and tabesdorsalis that involve degeneration of the upper extremities, the cervical, and later the dorsal spinal columns among a group of many complications caused by the spirochetes of syphilis (Treponemapallidum). Symptoms and signs include the lack of sensation and coordination, incontinence or retention of urine, loss of reflexes, bone and joint disturbances as well as progressive damage to organs and the brain.

TBRF: *See tick-borne relapsing fever.*

tick-borne relapsing fever: a febrile Borrelia disease that manifests after a 7 to 10 day incubation period following the bite of O.hermsi ticks that vector the agent B.hermsii from chipmunks and other rodent reservoirs to human hosts. In humans this extremely serious infection is marked by bouts of high fevers that relapse and remit multiple times.

tetracycline: Any of a broad spectrum group of antibiotics to include, doxycycline and minocycline that are effective against many organisms , including gram-positive and gram-negative bacteria, rickettsias, mycoplasmas, chlamydias, and certain viruses, protozoa and actinomycetes.

titer: numbers assigned during tests for pathogens in sera that relate to the amount of one substance that is needed to produce a measured reaction to another substance. In agglutination titer, the highest numbered dilution to cause clumping together of microbes or other antigens indicates previous exposure to antigenic components of a given disease.

typhus: a large group of related infections caused by many different species of *Rickettsia.* The classic form of epidemic typhus *Rickettsia prowazekii,* is transmitted by lice. Other forms of the infections are caused by several species of Rickettsia, and are transmitted by ticks, fleas, mites, lice and more.

vector: something (usually an arthrodpod) that carries an infectious agent from one host to another.

virus: an extremely small infectious agent that can only replicate and mutate within living host cells.

Yersinia pestis: gram negative bacteria transmitted to humans and some animals.

COLOPHON - This book is a revised version of the original text that was written and printed in 2013 at the author's expense in order to document information for those whose lives have been compromised by tick-borne disease. It was distributed at no cost to family and friends as well as a few professionals and to people who contacted the author for information. Because of many requests for more books this newer version was revised and edited in 2014 for the purpose of making it available at low cost to those who wish to purchase it. Any profits will be donated to researchers who seek to prove with advanced laboratory techniques the Borrelia cause of MS, as well as other chronic manifestations due to tick-borne pathogens, so that appropriate antibiotic treatments can be available to those who need them. The anecdotal information within is not meant to be taken as medical advice or proof of anything. Citations have been provided within the text rather than in a bibliography. No index has been provided because with Internet access, specific information can easily be retrieved.

Bonnie Bennett and her husband Dick divide their time between their homes in Arizona and Oregon. Her decision to write this anecdotal synopsis of the last three and a half decades is because no one else has ever observed or chronicled the tick-borne relapsing fever long-term, chronic complications, especially MS. In the words of her family physician, Dr. Harold Deal in 1982, "If you don't let your observations be known, it may take another hundred years for the medical community to learn about it so, if you don't do this, who will?"