ON THE ORIGIN OF ILLNESS

DAVE BEXFIELD

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BY MEANS OF A HIDDEN INFECTION

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There is a reason rates of chronic disease, autoimmune conditions, cancer, metabolic disorders, chronic inflammation, obesity, and mental illness have been rising dramatically with each recent generation. A hidden infection lurks within most of us. Humanity, unknowingly, has been trying to tame it for millennia, but a perfect storm accelerated its uncontrolled spread in the 1950s. Its presence was betrayed by the widespread use of medications meant to treat diabetes and weight gain. Those drugs, known to benefit over 175 health conditions, are starving the infection of its fuel: glucose derived from carbohydrates. Our diets and exercise routines are doing the same. The evidence hides in plain sight within a pattern that has long flummoxed scientists: why women in the prime of their lives are disproportionately affected by chronic illness. The answer to that disparity lies within an overlooked trace mineral—the elusive missing puzzle piece that not only explains the health crises engulfing the world but also dispatches countless medical mysteries and unlocks novel treatments. This is the origin of illness.

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—Preface—

HOW CAN I respectfully and professionally inform the world's leading researchers, medical journals, and public health institutions, that much of the settled science that they have relied on for decades is flawed, built on misinterpreted data? How can I explain to the ever-growing legions of people struggling with medical issues that their suffering likely has a common cause? And how can I tell the seemingly healthy, particularly those in our youngest generations, that a hidden menace lurks in most of us?

I have not been hasty in coming to this conclusion. It took 5 years, 500 citations, and one correction to a foundational assumption to change everything. The swirling medical mysteries of today—the rising rates of mental illness and cancers in the young, the steady climb of autoimmune diseases and metabolic disorders, the increasing threat of dementia, soaring chronic inflammation and pain, the curious upsurge of autism and ADHD, the unrelenting scourge of long Covid, even the wild successes of the newest weight-loss medications—all have a shockingly straightforward answer. An answer hiding in plain sight for a century.

My name is Dave Bexfield, I'm 57, and I'm a profoundly disabled health advocate from Albuquerque, New Mexico. People like me, with no formal medical education, are not supposed to have the aptitude to solve an enigma of the ages. But fresh eyes on a problem can produce unexpected results. As I apprehensively follow the same path of revolutionary pioneers of the past, from Hungary's Ignaz Semmelweis to Australia's Barry Marshall, I am going to be called crazy, delusional, a nut case. Another internet crackpot with a harebrained theory and a soapbox. No one is going to take my discoveries seriously, much less examine them with any rigor. History demands that my outreach will be futile, all but doomed to fail.

I am sorry to disappoint history.

Science repeatedly reminds us that correlation is not causation, to not get blinded by connections, no matter how logical. All of us, from seasoned researchers to amateur sleuths, have been looking in the wrong places, laser-focused on red herrings, convincing ourselves that the wave of sickness overwhelming our planet in recent years must be due to something introduced into society in the past three decades. Researchers have theorized that it is a complex interplay of factors, most likely a combination of unhealthy changes in our eating habits, a dangerous rewiring of our genetic gut microbiome, and the unrealized hazards of modern chemicals.

Rational. Sound. Commonsense. And wrong.

The answer lies within another curious trend that has stubbornly defied explanation: the unmistakable target on the backs of women. Women are far more likely to be diagnosed with autoimmune diseases, type 2 diabetes, Alzheimer's, severe depression, osteoporosis, long Covid, and a constellation of other illnesses. But why? It's a puzzle that has thoroughly bewildered

scientists, the medical mystery of mysteries. Theories abound, but nothing has been found to be airtight, conclusive.

For good reason. It has never been the fault of changes to lifestyle, the environment, or genetics. If you're detecting an uncommon level of confidence and a sense of certainty in my writing, that's because after years of research and chasing empty leads concerning my own health situation, I unexpectedly stumbled on a fatal error in one of the foundational pillars of modern medicine. That ultimately led me to the answer. Resolving the disparity between men and women was the elusive final puzzle piece, one that not only explains the health crises engulfing the world but also dispatches countless medical mysteries and unlocks novel treatments. In time, it will be regarded as the holy grail of medicine.

Such a discovery was believed to be unattainable, but please hold any applause or finger snaps for me personally. Although I was responsible for connecting the final dots, I am not the hero of this story. The true heroes are the researchers and specialists who have toiled for years, often spanning entire careers, to collect the necessary evidence. They are also the journalists and investigative reporters who have tirelessly devoted their lives to seeking and exposing scientific truths, all for little pay and even less fanfare. And they are the stubborn advocates, from patients to caregivers, whose ceaseless work and persistence—trying to sound the alarm that something isn't right, often through thick clouds of sickness—kept hope alive despite disbelief and endless gaslighting. Pay attention to all their names as you read this; humanity is forever in their debt.

Before we proceed, I have several more requests.

- This will be one of the most challenging things you'll ever read. If it hasn't already, your Semmelweis reflex should soon be kicking in, the knee-jerk reaction to reject and dismiss new ideas that conflict with existing beliefs. I experienced the Semmelweis reflex myself researching this, wasting months fighting with my own personal prejudices, ignoring evidence that was incessantly tugging at my shirt like an impatient 2-year-old. Every future MD is cautioned to beware of confirmation bias, groupthink, theory-induced blindness, and belief perseverance. What you are about to read will trigger them all, and even the brightest, most open-minded will find it difficult to prevent. But please try.
- Withhold assigning blame. As much as we all want to find fault (and there is plenty to go around), this travesty has been in the making for generations, long before any of you started practicing medicine or got thrust into an unexpected fight with a chronic illness. That also means the following discussion is devoid of conspiracy theories: there are no government cabals or malicious scientists with devious agendas, no talk of lab leaks or evidence of subterfuge from the pharmaceutical industry. Everything that could go wrong, went wrong, perfect storms colliding with one another. That's all.
- Lastly, please sit down before reading, and not just because it is exceedingly long. This is lengthy for a reason. The following pages are going to wholly rewrite the framework of medicine, upending countless fields of study. What they reveal will be simultaneously both glorious and horrifying. I'm sorry. It is not an exaggeration to say that by the time you reach

its conclusion, it will be one of those moments in life that occupy a permanent corner of your brain reserved for searing events that irreparably change how you see the world.

While I'll do my best to guide you through this tangled forest of discovery with clear logic and plain English, please note that at times it will get into the technical weeds and explore complex theories to better satisfy doubting Thomases. I intend to address all your questions, even those you never considered asking. Be patient. And please extend the same grace Darwin asked of his own readers in *On the Origin of Species*—he warned that errors would have crept in, that his work was necessarily imperfect, and that he trusted readers to have some confidence in his accuracy while awaiting fuller work to follow. I ask the same. For scientists, I apologize in advance for the unrestrained sureness in my language and the sweeping conclusions that accompany it. It's not ego or arrogance. Thanks to science, this impossibly complex puzzle fits together only one possible way.

If you are still with me, congratulations, you've cleared the first hurdle. That, or you have a scientist's level of curiosity, an instinct that something genuinely doesn't add up. Either way, welcome.

Buckle up and hang on. And maybe hug a loved one; you're going to need it.

—Chapter 1—

Round Pegs, Square Holes & Dumb Luck

For much of the past century, technology has pushed astounding advances—space travel, nuclear energy, personal computing, artificial intelligence—and has done so at such breakneck speeds that terms like "Moore's Law" have entered the public vernacular. But there has been one glaring, inexplicable exception: medicine.

For decades, the world's brightest scientists and researchers, despite the aid of trillions of dollars and vast resources, have had an abysmal track record in finding the cause of, diagnosing, treating, and curing countless health conditions. Indeed, "most healthcare interventions" (94%) are "not effective according to high-quality evidence" found the highly regarded *Cochrane Reviews*[1] after a 2022 systematic meta-analysis. Mysteries continue to churn unabated around autoimmune disease, chronic illness, mental health, metabolic disorders, and cancers. (This April, *Cochrane* concluded[2] that anti-amyloid Alzheimer's drugs show "no clinically meaningful effect.") It's gotten so bad that there is an insider term to describe the consistent failures, "Eroom's Law," the phenomenon where the speed of developing a new drug continues to slow while costs increase exponentially, the sardonic reverse of Moore's Law.

On the surface, it makes no sense. These same scientists can solve a once-in-a-century pandemic in a matter of months, but groundbreaking success in any of these other areas, with few exceptions, has eluded our best minds since time immemorial. The lack of progress is hard to comprehend, with at least 10 million practicing doctors all supported by the most ambitious AI outreach imaginable. Still bupkis. It's borderline comical, more a Gary Larson panel from *The Far Side* or a clever cartoon in *The Atlantic*. Picture a sea of men in white coats analyzing every minuscule aspect of a round peg, plumbed with a mess of wires, surrounded by bubbling test tubes, high-powered microscopes, and whiteboards clogged with complicated formulas, chemical equations, and graphs. For the caption, one scientist makes a bold proclamation: *Eureka! I'm now 100% confident that we are* **this close** *to getting closer*. Oh, there is one more element to the illustration. In the corner of the laboratory is a faded, forgotten poster, edges curled, of the goal: a square hole.

That peg is never going to fit and those wishful cures, barring dumb luck (more on that in a moment), are going to remain forever elusive. All the ice bucket challenges in the world won't move the needle. Unlimited funding won't change the calculus. As *The New York Times* has noted,[3] AI's ability to generate genuinely new scientific ideas remains deeply contested. Without realizing the data is compromised, scientists and every machine they feed the problem into will continue to toil on an unsolvable problem. It is impossible to complete this jigsaw puzzle of the ages if your dog ate the final piece. The modern scientific practice of evidence-based medicine is not equipped to uncover and identify fatally flawed research. Instead, it doubles down on popular, accepted hypotheses, creating an unbreakable feedback loop, as misinterpreted data reinforces misinterpreted data.

What I've discovered is going to seem far too overreaching to be real, a laughable theory that stretches the bounds of plausibility to its very outer limits. It is also going to feel too convenient, too simple, and too obvious to have been overlooked by experts. ("If there was a smoking gun, we would have found it by now," researchers inevitably say when interviewed about any unsolved health conundrum.) On every level, it is going to sound unbelievable. That's what happens when every possible medical disaster converges at once—when the most derided, disbelieved, and dismissed health claim of the last century turns out to be very much anchored in truth.

The health carnage we have been witnessing unfold around us (and to us) is all being driven by a solitary infection. A bacterial infection of the Borrelia genus. Lyme disease. It's always been Lyme disease, and it has been toying with our health for thousands of years. The third rail of medical controversies—the most mocked health condition of our time and the butt of endless jokes (even *The Simpsons* dismissed the disease as psychosomatic in an early episode[4]) truly is *everywhere*, duping doctors, fooling scientists, and contaminating research studies with its lurking presence.

That rising feeling of revulsion, anger, disdain coursing through you? *That's* the Semmelweis reflex.

Lyme disease?? It's unfathomable, I know. It violates modern medicine, I know. But before you dismiss what I've just written as an impossibility, a single invader responsible for the damage described in these pages, consider what a successful treatment might look like. It would appear miraculous, treating seemingly everything under the sun. Throw a dart: cardiovascular disease, sleep apnea, Alzheimer's disease, chronic kidney disease, substance abuse, autoimmune diseases, psychosis, non-alcoholic liver disease, cancer. It would even address diabetes and the obesity epidemic. Such a drug, no doubt, would shock the medical establishment and take the world by storm, triggering medication shortages.

Suddenly, that theoretical, prospective treatment doesn't sound like such an impossibility, does it? That "dumb luck" I was talking about earlier? There's a clear reason why the blockbuster GLP-1 drugs—Ozempic, Mounjaro, Wegovy, and others—have had such head-scratching success across such a wide swath of health issues.

They are all effectively treating variants of Lyme disease.

How I Got Into This Mess

Before we get into how I solved this colossal puzzle, I probably should introduce myself and explain how I got into this mess. In 2006 at the age of 37, I was diagnosed with the autoimmune condition multiple sclerosis. Like many who get diagnosed with a chronic disease, it was a shock. It was also frustrating. At the time there were few helpful resources, and those that existed painted a bleak future. As a professional writer, I had the skillset to change that dynamic and created the optimistic, science-heavy website www.ActiveMSers.org (tagline: be active, stay fit, keep exploring). It quickly grew into one of the largest MS websites in the world, my story featured in countless magazine and newspaper articles. But it was David "The Haggler" Segal's 2014 piece in *The New York Times*[5] about my unrelenting, ultimately successful, 4-year battle with my $2 billion healthcare insurer Presbyterian Healthcare Services—they had repeatedly denied my participation

in a clinical trial despite my having failed all existing treatments—that first suggested something about me was different. The treatment worked, putting my disease into years-long remission, and I got reimbursed in full (including interest and taxes), amounting to over $500,000. But then my disease woke up again.

By 2021 my health was once more in freefall, UTIs were inescapable, and creeping psychosis threatened my grip on reality. If something didn't change, I would soon need full-time care. Then, on a Sunday morning in early fall, I came upon an opinion piece in *The New York Times* by columnist Ross Douthat[6] about his experience with Lyme disease. All my symptoms aligned,[7] from a swollen knee to facial palsy to tachycardia. I had even been hiking in Lyme, Connecticut, where I got bitten by a tick, a full year before my MS diagnosis. Now it was beginning to all make sense.

For the record, long before I made this discovery, I fiercely believed Lyme was a contained, localized threat, little more than a convenient scapegoat for the unexplained. When I started investigating how it was remotely possible for me to be misdiagnosed with multiple sclerosis for 17 years, I just wanted answers, some level of accountability. After a tireless three years of searching, I finally got those answers… and far, *far* more than I had bargained for. I uncovered a series of catastrophic mistakes in scientific research, errors that bleed into innumerable hypotheses and theories—conclusions that have evolved to form the very foundation of modern medicine. I didn't intend to discover any of this, I *shouldn't* have discovered any of this, and yet here we are. With the health of our planet at stake.

Now if you think there is no way on God's green earth that an amateur medical sleuth with zero budget, zero scientific experience, and zero published studies has conducted the necessary in-depth research to prove that Lyme is at the root of countless health conditions, you would be correct. I haven't. That's because today's scientists and their predecessors have already done (and overdone) the research.

With millions upon millions of studies about the disease published in countless medical journals, Lyme is by far the world's most researched health condition. Scientists, unknowingly, have been mining and parsing critical data on the disease for over a century. The proof has already been unearthed—it is overwhelming, indisputable, an embarrassment of riches. But wholesale paradigm shifts in medicine follow a predictable pattern: overlook, ignore, and dismiss all evidence that contradicts long-held opinions. Some science historians consider this type of resistance[8] "the single most formidable barrier to scientific advances, and so disturbingly regular as to call for a partial restructuring of the modern scientific enterprise." Examples flood the annals of medicine, with one that particularly stands out.

In the mid-1800s, when Dr. Ignaz Semmelweis urged fellow doctors practicing in maternity wards to disinfect their hands in between patients—the mortality rate of mothers at the time was an egregious 18%— they scoffed, blaming bad air and miasma. The junior doctor pleaded with his contemporaries to look at the evidence, that in his ward the mortality rate had dropped to less than 2% after handwashing. They couldn't be bothered. Incensed and determined, Dr. Semmelweis and a handful of supporters pressed on, writing letters to anyone who might listen, from directors at

prominent clinics to leading medical journals. Shoulders shrugged. After more than a decade of being dismissed, the frustrated physician wrote a 524-page tome[9] detailing his findings in depth while disproving a mountain of other claims. It mattered little. In 1865, colleagues lured him to an asylum and had him committed. Two weeks later, the 47-year-old died from an infection identical to the type he had been warning about. Only decades after his death did doctors eventually come around to his ideas and start routinely washing their hands. Dr. Howard Markel, a historian of medicine at the University of Michigan who writes for PBS NewsHour, has noted that,[10] "Today, in every school of medicine and public health, his name is uttered with great reverence."

The reason I've retold this familiar story? I, too, have written to the international medical community warning of a hidden epidemic that scientists have missed. And I've also written a defining book. Together they upend much of modern medicine.

Sit Down Before Reading

When I embarked on my medical memoir *Sit Down Before Reading*,[11] I was not trying to emulate Dr. Semmelweis. I had only planned to break down and expose how easily Lyme disease could be confused with MS, working on the assumption that my misdiagnosis wasn't a mere outlier in the MS community. Others certainly must have Lyme like me, I reasoned. So, with full transparency, I started publishing chapters in real time for members of my nonprofit to read; I had figured at most a couple dozen installments. But the more I investigated, the more glaring inconsistencies, unbelievable coincidences, and implausible conclusions I discovered, reams of published research irreparably corrupted.

I kept writing, I kept researching, and what I continued to uncover just kept getting worse. But who was ever going to believe me? I had to be as thorough as possible, leave no question unanswered. In order to understand how scientists were being deceived by their own research, I had to become fluent in a broad range of topics: statistical theory, genetics, nutrition, epidemiology, neurology, world history, infectious disease, cardiology, rheumatology, pharmacology, and much more. I also had to familiarize myself with how scientists of the past tried to discredit researchers like Dr. Semmelweis, studying their playbook of ultimately flawed arguments and reasoning.

By the time I reached the memoir's conclusion, three years later in October of 2024, *SDBR* had stretched to an exhaustive 52 chapters, matching Dr. Semmelweis' work both in length and depth. I had connected virtually *every* dot: when Lyme disease started, how it spreads, why it went undetected, how it fooled scientists, how it presents, and why it responds to unique, unrealized types of treatment. I identified the missteps, the miscalculations, and the grave mistakes made over the years. I also made sure to show all my work, documenting in detail how I solved each puzzle (as well as revealing my own false starts). There are over 1,000 studies referenced, some published a breath ago, others that date back more than a century. The scope of research cited and broken down, from obscure speeches at medical conferences 50 years ago to random seminars given by Nobel laureates, is breathtaking in its granularity. I left nothing to chance.

And yet, exhaustive documentation and ironclad logic have never been sufficient currency in the court of scientific opinion. I knew no one was going to believe me—that's how modern science operates. But that reminded me of what celebrated astrophysicist Neil deGrasse Tyson has often noted: "The good thing about science is that it's true, whether or not you believe in it." If I wasn't going to be able to change minds, I had to put my faith in science and trust that it would do it for me.

For expert advice from a researcher who eventually succeeded at changing minds, I turned to Dr. Barry Marshall, the Nobel-prize-winning Australian[12] who discovered that a bacterium was chiefly responsible for ulcers and gastric disease, not stress or spicy foods, the prevailing thought before he fingered Helicobacter pylori. (His ideas were originally called crazy and received incredible pushback.) Try your hardest to dismantle *your own* hypotheses, he advises budding researchers, and see if they can survive. So, I put on a demolition hard hat and got to work. I even designed a novel, five-step process (SHARDs)[13] to pick apart and appraise medical theories so that they could weather the tightest scrutiny.

When you try to blow something up, there can be unintended consequences. Instead of turning my hypotheses into rubble, my attempts to lay waste to my theories only strengthened them. They would not break. Was I doing something wrong? I then tried using the same barometer on existing medical theories and was aghast at what happened next. Towering, long-trusted pillars that hold up much of medicine started collapsing under their own weight. The decimation was staggering. Horrified, I struggled to comprehend how this could happen. The answer would soon become clear.

Following in the footsteps of today's data-driven scientists, I had spent 40 chapters trying to conclusively connect some MS patients to Lyme disease by teasing out clues from the tiniest of details: bloodwork, spinal fluid, advanced MRIs, and the like. I was confident that such a connection existed, but the best picture I could extract from my microscopic dive was always fuzzy, inconclusive. I was prepared to admit defeat. Then I saw it, an errant string. I pulled. And kept pulling. Dear God.

To understand what is about to unfold, you need to first forget everything you think you know about Lyme—the ticks, the rash, the blood tests, all of it. Next, suspend what you've been taught about health and medicine. You need to approach this with a fresh, inquisitive mind unburdened by medical dogma—dare to question everything, including long-accepted hypotheses. Finally, put aside the kind of biases and preconceptions that have ensnared the most talented of scientists—cleanse yourself of hubris and certainty.

Avoid these pitfalls and you'll have in your possession what amounts to the long-sought skeleton key to today's medical mysteries.

The Unraveling

The unraveling of modern medicine began innocently, with a nondescript study published in the spring of 2022. Trying to assess the global spread of Lyme disease, Chinese researchers (Dong, et al.) released a far-reaching meta-analysis[14] investigating the presence of Borrelia burgdorferi

antibodies in the world's population. After narrowing 4,000+ studies to 89 for inclusion (spread among 28 countries and six continents), scientists were astonished to discover that an estimated 14.5% of humans have evidence of an active or recent infection of the bacteria that causes Lyme disease. More than 1.1 billion people. For perspective, the CDC reports 36,000 people are officially diagnosed and treated for Lyme disease each year in the United States.

The first axiom of SHARDs, the "S," is that Sound Science makes Sense. Even though the government agency admits they believe the true number to be closer to a half million (476,000 is most often cited), and others project a similar unofficial caseload in Europe where it is even harder to get diagnosed, the math still makes absolutely no sense. There's a gobsmacking difference between estimates.

This should have given researchers pause. Red flags should have been flapping furiously, alarm bells clanging with urgency. *Something is wrong. Something is very wrong.* Instead, the study that had initially garnered worldwide press, faded like a temporary tattoo. Even the chief scientific officer of a leading Lyme advocacy group dismissed the 1.1 billion estimate, "debunking" the research[15] with reasoning—the sample size of 158,287 couldn't possibly be representative of the global population—that completely disregards basic statistical theory.[16] Instead of approaching the 1.1 billion figure with curiosity, the findings were dismissed outright as an impossibility. How could infections be so widespread given the limited habitat of blacklegged ticks, the specific vector believed to be responsible for spreading Lyme disease? They didn't line up with the conventional and accepted expectations of the disease being regional and confined. And just like that, a study that could have torn off the veil of a Lyme epidemic was set aside like so many others before it.

This particular, seemingly innocuous, misstep was set in motion decades and generations ago, calling to mind the butterfly effect. Lyme has been synonymous with ticks since its discovery in the 1970s, the connection between tick and disease never questioned. Unexpectedly, this single assumption—to get infected with Lyme one must be bitten by an infected tick—set off a catastrophic chain of events, cementing its ability to continue wreaking chaos on human health unchecked.

How could this be?

Countless health conditions are diagnosed by the process of elimination. When Lyme is dismissed as a possible cause and eliminated as a suspect—due to, say, an absence of travel to areas endemic for Lyme disease or evidence of a tick bite—physicians, understandably, turn their focus to other alternatives. Everything is predicated on ticks spreading the disease. But if that assumption is wrong, the diagnostic model doctors have long trusted falls apart.

If anyone had bothered to investigate that 2022 meta-analysis with any vigor, they would have discovered scores of people with antibodies to Lyme disease who live nowhere near tick habitats. And that would have led to an inescapable conclusion, one supported by unbiased laboratory diagnostics from around the world: Lyme disease must be spreading and proliferating without the aid of ticks.

This changes everything. Where are all these cases? They should be straightforward to find. With more than a billion people infected with the bacterium that causes Lyme, one of the most destructive diseases in human history, a tidal wave of cases should have been flooding health systems with patients desperate for relief. But that hasn't been happening.

Or has it?

—Chapter 2—

The Great Imitator

Nicknamed "the great imitator" for its ability to mimic more than 200 health conditions, Lyme doesn't particularly look like anything. It looks like *everything*. Fatigue, brain fog, joint pain, rashes, depression, digestive issues, weakness, anxiety, numbness, heat sensitivity, fever, sleep problems, pins and needles, chest pain, psychosis, heart conditions, eyesight issues, breathing challenges, and so much more. If Lyme simply is being mistaken for other health conditions, it makes sense to start looking at the most obvious suspects, beginning with a behemoth.

Ten percent of the planet's population, some 800 million people, are estimated to have at least one autoimmune disease[17] (13% of women, 7% of men). Despite huge investments and concentrated study, to date no one has any clue where these autoimmune diseases come from, how to properly diagnose them, how to best treat them, or how to cure them. Collectively, the 100+ autoimmune diseases thought to exist are an enigma, a riddle that has stumped doctors for decades. The litany of symptoms attached to autoimmune diseases conveniently also happen to mirror those of Lyme uncannily. *Too* uncannily.

The very idea of autoimmunity was originally contentious and highly controversial. The existence of antibodies to fight off toxins and bacteria was first discovered in the late 1890s, and at the turn of the 1900s, German biochemist Paul Ehrlich realized that if the body could produce "magic bullets" to kill foreign invaders, some might contend that those same weapons could theoretically turn inward and destroy the body's own tissues. Nonsense. As one of the pioneers of modern medicine, he argued that a healthy organism must have internal regulatory mechanisms to prevent self-poisoning, coining the term *horror autotoxicus*. Ehrlich later won a Nobel Prize in 1908 for his work on immunity. The following year he developed an effective treatment for syphilis, cementing his reputation in the medical community. "Ehrlich's prodigious talents in the laboratory — he was called a virtuoso of test tubes — were matched by a combination of intuition and deduction that marked him as a genius," reports *The New England Journal of Medicine*.[18] "He was the father of hematology, a revolutionary immunologist, and the creator of the field of chemotherapy."

But there was a festering problem.

Scientists, after searching for half a century, still couldn't explain unusual antibodies that accompanied a multitude of health issues. Despite intense research with increasingly advanced technology, they could never identify an invader. If a stealthy bacteria or virus were responsible, certainly they would have found it by now. It was as if they were chasing a ghost. But Ehrlich's theory, despite misgivings, still had strong support long after his untimely death at age 61 (he suffered from deep depression and a series of strokes at the end of his life). In 1954, as scientists celebrated the centennial of Ehrlich's birth and his concept of *horror autotoxicus*, his mentor Ernest Witebsky declared,[19] "No living organism would be capable of producing—or would *dare* to

produce, if you wish—an antibody against constituents of its own body, for this would be incompatible with life."

Even so, an alternative theory had been gathering steam. An overactive immune system, a remnant from the earliest days of humans trying to survive in a hostile world replete with myriad infectious diseases, might be misfiring, causing the body to inadvertently attack itself. This hypothesis was in direct conflict with both Ehrlich's conclusions as well as Darwin's theory of evolution—the infectious threat from the earliest days of humanity had subsided, meaning "autoimmune" cases should have steadily subsided as well—pockets of frustrated scientists embraced this paradoxical and divisive opinion.

Researchers first linked Hashimoto's thyroiditis, a disease with antibodies that appeared to target the body's own thyroid tissue. Of course, that's why an attacker couldn't be found, reasoned scientists. Other theories soon piled on, and momentum was building. The concept of autoimmunity shortly became settled science, one whose consequences, as we'll see, would prove catastrophic. Any hypothesis that threatened this theory was swiftly squelched, mocked, and derided. That included a landmark discovery that would have changed the course of history.

When *Time* magazine reported in 1957[20] that Philadelphia bacteriologist Rose Ichelson had found spirochetes[21] in the spinal fluid of MS patients, inferring that "multiple sclerosis is caused by the spirochete, and early attack on it should lead to cure or alleviation," it started a medical controversy. Even though she was "100% sure" spirochetes were behind the disease[22] known as MS, her predominantly male contemporaries dismissed her discovery as wishful thinking and her corroborating microscope slides of the bacteria as "scratches on the glass.[23]" Ichelson died a few years later, at her Philadelphia office in St. Luke's and Children's Medical Center, working on a vaccine. A pioneer, who should have had a Nobel prize around her neck and statues built in her honor, lost to mainstream history.

Countless researchers since have experienced aggressive, punitive pushback when it comes to Lyme disease, including today's advocates like Dr. Alfred Miller, Dr. Steven Phillips, and Dr. Daniel Cameron. All three have released powerful books to predictable eye rolls: Cameron's *An Expert's Guide on Navigating Lyme Disease*,[24] Phillips' *Chronic: The Hidden Cause of the Autoimmune Pandemic and How to Get Healthy Again*,[25] and Miller's just released *Borrelia the Hidden Imitator*.[26] Labelled as quacks, sanctioned by medical boards, ostracized by their peers, they've tried to raise the alarm only to be ignored by the medical establishment. But it's not just the "Lyme loonies" who question the autoimmune hypothesis.

The Autoimmune Paradox

Evolutionary biologist Paul W. Ewald, director of the Evolutionary Medicine program at the University of Louisville, wrote *Plague Time: The New Germ Theory of Disease*,[27] arguing that pathogens are at the root of many chronic illnesses, and that autoimmunity is the very antithesis of evolutionary biology. A growing number of troubled scientists agree. "No autoimmune diagnosis has been shown to confer any sort of beneficial survival trait," concerned researchers remarked in

2010,[28] echoing the undercurrent of unease scientists are having with the entire idea of autoimmunity. "Under these circumstances, one would expect any faulty gene or network of genes associated with an autoimmune condition to be selected against, especially since many autoimmune conditions strike during the reproductive years." And yet today those same conditions are skyrocketing.

Why hasn't the medical establishment taken the fears of these researchers more seriously? For starters, the idea of stealth pathogens causing destruction isn't new, it has had supporters for decades. International autoimmune conferences routinely feature presentations and papers investigating infectious agents and their potential role in disease. The awareness is there, but the case is weak. Proof is hard to come by—the suspected pathogens are notoriously hard to culture, and therefore near impossible to reliably detect—making fingering an offender beyond challenging. If you can't find the bacteria, mainstream medicine assumes it isn't there. Then there is the problem of scope. The number of suspects is so vast, with so many viruses and bacteria to consider, it gets a collective: "so what?" Without narrowing the wrongdoer backed by compelling, even mildly suggestive, evidence, it's just another hypothesis to tack on the board next to all the other plausible theories. But scientists are trying.

Take respected MS researcher Dr. Gavin Giovannoni, who is so convinced that multiple sclerosis is caused by an infection that he recently left clinical practice to focus on proving his infection hypothesis.[29] "To prove that MS is caused by a single factor or the interaction of several factors, we must explain everything we know about the disease's epidemiology," he wrote on his Substack blog[30] in February of 2025 titled "Rising incidence of MS in women: what is [it] telling us about the cause of MS?".

I've worked with Dr. Giovannoni in the past as part of my MS advocacy. He is everything a brilliant doctor should be: inquisitive, passionate, unafraid of challenging the status quo. And he is approaching the problem smartly, recognizing that any solution must convincingly address that intractable gender question. "If EBV is the cause of MS," his prevailing theory, "how does EBV explain why relapsing-remitting MS (RRMS) is becoming more common in women?" But he, like generations of scientists before him, is stuck on the perplexing problem. "I don't have the answer to this MS mystery. If any of you have any ideas, please let me know."

It's the same gender disparity puzzle that has flummoxed researchers studying other autoimmune diseases and chronic conditions, from rheumatoid arthritis to fibromyalgia to long Covid. Stanford researchers excitedly announced[31] in 2024 that they may have cracked the case, pinpointing the Xist molecule—found only in women—as one of the reasons there are disease differences between the two sexes. Inspired science, reasonable hypothesis, wrong conclusion. Unbeknownst to the Stanford-led international team of researchers (headed by Dr. Diana R. Dou and senior author Dr. Howard Chang), Dr. Giovannoni, or any of their contemporaries working on the same puzzle, science has granted us only one possible answer.

Lyme disease has few peers as a medical enigma, but one unique feature of Borrelia burgdorferi—realized a quarter century ago—sets it apart from virtually every other organism on the planet.

Researchers James Posey and Frank Gherardini at the University of Georgia found in 2000 that B. burgdorferi has no need for iron,[32] a metal that is essential to all other life. Instead, the bacteria requires unusually high levels of manganese,[33] a discovery made thirteen years later in 2013 by scientists at Johns Hopkins University, Woods Hole Oceanographic Institution, and the University of Texas. The authors deserve recognition by name: J. Dafhne Aguirre, Hillary M. Clark, Matthew McIlvin, Christine Vazquez, Shaina L. Palmere, Dennis J. Grab, J. Seshu, P. John Hart, Mak Saito, and Valeria C. Culotta. Together, these studies will go down as two of the most informative breakthroughs in the history of medicine.

Thanks to this research, the explanation for why women are disproportionately affected by so many chronic illnesses becomes simple, elegant, obvious—so obvious, even a fifth grader could figure it out—if you just knew where to look. But blink and you'll miss it.

Everyone blinked. Everyone missed it.

Connecting the Dots

The gender disparity puzzle was, by far, the most difficult for me to solve. It took me well over a year, chasing down false leads and discarding too many tenuous hypotheses. At first, the iron/manganese connection seemed more like a curiosity, a bit of trivia that might surface on *Jeopardy!* rather than something groundbreaking. By Chapter 48, I was resigned to accept Stanford's explanation of Xist even though it left too many holes, one being its inability to account for the neutral gender disparity in childhood and in older age. If Xist was the answer, diseases that are dominated by the female gender shouldn't deviate with age. But they do.

Midway through my memoir's final chapter, though, I realized my mistake—I had been overthinking the problem the entire time—and corrected course. The result? I found the final missing link that seamlessly and conclusively ties everything together, dissolving medical mystery after medical mystery in succession. I was so astonished at the time that I could only compare my elation to legendary NC State coach Jimmy Valvano's excitement after shockingly winning the 1983 NCAA men's basketball championship in an unforgettable upset at The Pit, Albuquerque's storied sunken arena. Like Jimmy V, I was just looking for people to hug in Albuquerque.

I'd hug each one of you right now if I could, doctors especially.

Even if someone had managed to figure out the gender brainteaser, it wouldn't have mattered. The game our physicians have been playing is essentially rigged. Think the 1950s TV game show Twenty-One or the 1919 Black Sox scandal. The researchers never stood a chance.

To begin to understand how it could be that generations of specialists—experts trained to drill into the deepest depths of a specific problem—have yet to put the requisite puzzle pieces together, we must confront an uncomfortable fact. The formulaic way doctors have been taught to problem solve has flaws. Evidence-based medicine, or EBM, is not as unassailable as everyone thinks it is, a known issue among researchers[34] who broadly continue to fully trust the method nonetheless. "The main problem with the EBM approach is the restricted and simplistic approach to scientific

knowledge." That, and it also has a tragic blind spot, one that Lyme disease has taken advantage of with devastating effectiveness. And I discovered, by total happenstance in the waning pages of what had become a 52-chapter memoir, that there is only one way to see it.

"Researchers have been playing Connect-the-Dots for the better part of a century, repeatedly drawing lines between revealing clues," I wrote in Chapter 41,[35] applauding their tireless detective work. "They just never stopped to take a step back to contemplate the resulting picture. And once you see it, you'll never be able to unsee it."

To see the bigger picture, you must connect the unconnected.

If that approach sounds both familiar and esoteric, it should. "You can't connect the dots looking forward," said Apple founder Steve Jobs in his fabled 2005 Stanford commencement speech.[36] "You can only connect them looking backwards, so you have to trust that the dots will somehow connect in your future." But Jobs was not the first to recognize this. The most pioneering scientists, philosophers, and inventors throughout history—Leonardo da Vinci, Isaac Newton, Charles Darwin, Marie Curie, Benjamin Franklin, Nikola Tesla, Jane Goodall, Thomas Edison, Copernicus, to name a few—share some combination of highly uncommon traits: polymathy and systems thinking.

Some 500 years ago, da Vinci mastered the art of "connecting the unconnected," a creative thinking strategy where seemingly unrelated concepts and ideas are intentionally, even forcibly, linked in the hope that it might spark new insights and possibilities. Famously, he realized that sound travels in waves after observing water rippling and bells ringing, tying the two concepts together. Da Vinci, like Jobs, was a systems thinker. Instead of focusing on the minutiae of a problem—concentrating on individual components in isolation—he continually stressed the importance of seeking the bigger picture, to look for patterns and correlations to realize the connectedness of things. Only then, by extracting the unknown through these "thought experiments," he believed you could uncover relationships that may have been overlooked, dismissed, or outright rejected by others.

It is all but impossible to solve this medical riddle of the ages without following, intentionally or by total fluke, the lead of science's greatest minds.

Here's how I did it.

The Big Picture

For me, problem solving starts with the two Cs: curiosity and creativity. I have a level of curiosity bordering on the obnoxious. Sometimes that has served me poorly. Touching an iron to see if it's hot, grabbing ahold of an electrified fence to see if it is indeed electrified, unclogging a running blender with a knife only to be rewarded with the task of cleaning an entire berry smoothie off the ceiling. But too often I have been rewarded. Failed experiments always inform, educate, enlighten.

I approached my Lyme disease problem initially with a narrow focus, trying to suss out how the disease could get conflated with multiple sclerosis. When I kept running into dead ends, I made a wholesale change of tactics. To stretch my imagination—and hypotheses—to their very limit, I

suspended most of what I knew about MS, Lyme, and medicine in general so that I could approach the problem with fresh eyes. I avoided popular theories, instead letting science steer me to answers, not the siren call of the loudest opinions. I also made a point not to let ambiguity, uncertainty, or even clear contradictions bother me, taking the leap of faith that I could address them later if I happened to have landed on a promising path.

This proved enormously challenging. Juggling multiple, tenuous hypotheses simultaneously meant keeping them all aloft until they either supported one another seamlessly… or they crashed down in unison, scuttling everything. This required me to pause publishing my real-time memoir to write the final chapters concurrently, toggling back and forth between a dozen lengthy chapters, knowing that at any moment my theories could collapse onto themselves, instantly turning the words already written into fantasy fiction.

I started with an outrageous hypothesis. What if Lyme's very nickname, the great imitator, is a misnomer? What if Lyme technically isn't imitating any of those diseases—what if it simply *is* those diseases?

Earlier I had scoffed at the idea of Lyme disease being mistaken for autoimmune conditions, thinking it ludicrous. Maybe a handful, but certainly not the entire lot of them. We couldn't possibly have gotten it *that* wrong. Except that no matter how hard I tried, I couldn't make that logic work. It turned into a clear, binary choice. Either autoimmune diseases exist, or they don't. Either Darwin's theory of evolution is irreparably flawed, or it isn't. I gambled on Darwin being right.

[I promised myself I would not harp on past mistakes made by the scientific community, but I must make an exception here. How did outright defying Darwin's famous theory and bucking Ehrlich's foundational conclusions with a poorly examined hypothesis—the body inexplicably attacking itself, essentially evolutionary suicide—pass the smell test? Evolution should have weeded out these supposed genetic misfires, but they've only been progressively increasing, not unlike something one might see with a *steadily spreading infection*. While a select brave few pushed back, generations of doctors shrugged and didn't question the autoimmune hypothesis. Mindbogglingly infuriating. Rant over.]

You don't have to be a polymath to see how doctors got bamboozled. For example, take the most common form of Lyme, Lyme arthritis. The symptoms of Lyme arthritis are nearly indistinguishable from those attributed to rheumatoid arthritis[37] and they are frequently confused with each other. The only way physicians can separate the two diseases: weighing the likelihood of a tick bite (based largely on travel history and any resulting rashes) and problematic diagnostic tests with known shortcomings (deeply flawed Lyme testing and unreliable "rheumatoid factors" used in RA).

Depending on how joint pain presents—scientists have identified more than 100(!) forms of arthritis[38]—they make educated guesses as to the cause. If a symptom arises that doesn't fit with the original diagnosis, it's revised. Rheumatoid arthritis with skin issues turns into psoriatic

arthritis. If, say, brain lesions are later discovered, a diagnosis of multiple sclerosis gets tacked on. As more health issues are revealed, patients get diagnosed with more conditions, a practice I coined "disease stacking."[39] Because Lyme can present so differently, that stack of questionable diagnoses can grow to look like a Jenga tower; about a third of those with an autoimmune condition have more than one[40] and it's not uncommon to have four or more. But the problem with Jenga towers? They eventually collapse.

The principles behind Occam's razor—the simplest solution is often the right one—would suggest that a single culprit is behind autoimmune conditions, which share remarkably similar symptoms, rather than a fragmented array of more than 100 separate diseases. Indeed, they are so similar that they have spawned research into their alikeness. (Colombian researcher Dr. Juan-Manuel Anaya has even labelled the coincidence the "autoimmune tautology" and presciently suggested a common origin.[41]) But the specialists who investigate and treat these diseases ply their trade in their own isolated bubbles; rheumatologists know little about the inner workings of Lyme disease and, likewise, Lyme experts are unfamiliar with the many nuances of arthritic conditions. And therein lies the crux of this travesty.

The answer has always been right there, *right there*, hidden in plain sight.

—Chapter 3—

Hidden Figures

Recall the well-known parable of the blind men and the elephant, where each sightless man touches part of the elephant—the trunk, a tusk, the tail—before confidently concluding that they are each grasping something different. When diseases are studied in their own separate silos, obvious connections, like parts of an elephant, get missed, misinterpreted. Closer inspection of the fuzzy science supporting any one of the autoimmune diseases thought to exist highlights this problem, but the research on lupus encapsulates the issue in stomach-churning fashion.

Due to the incredible variability in how it presents, lupus has been historically challenging to diagnose. In fact, the vast array of symptoms attributed to the autoimmune condition has earned lupus a familiar nickname: the great imitator. But the parallels between the disease and Lyme extend far beyond identical nicknames. For instance, the use of certain antibiotics in lupus—notably tetracyclines, sulfas, and penicillins—are used with caution[42] because they could trigger a severe disease flare. Lupus patients are warned to steer clear of these antibiotics. In contrast, those exact same antibiotics are frontline treatments for Lyme and cause the exact same debilitating exacerbation of symptoms. Only Lyme specialists have a clinical name to describe this predictable and expected side effect: the Jarisch-Herxheimer Reaction, a transient inflammatory response seen when antibiotics are introduced to spirochetal diseases.

By all accounts, lupus and Lyme appear to be variants of the same disease, only viewed through vastly different prisms. That helps explain why an astonishing 40% of lupus patients test positive for Lyme by ELISA,[43] results labeled as false positives, despite the fact that cross-reactivity between lupus antibodies and Borrelia antigens is itself a striking finding. But doctors have long been taught to ignore these test results, particularly in cases when tick involvement is implausible. After all, the epidemiological evidence appears overwhelming that these two diseases are separate and distinct from one another. In the United States, the hotbed for Lyme cases is in the Northeast, while lupus is most common in the South—and particularly less so in the New England region. Perhaps most instructive is the racial makeup of patients. People with lupus are disproportionately Black, a glaring disparity that cannot be explained if ticks are solely responsible for spreading Lyme. Why would ticks be uniquely attracted to African Americans?

Instead of tabling my hypothesis because of these obvious conflicts, I let them out on the longest of leashes, pressing forward. If I was right, those discrepancies would eventually get resolved. I then turned my focus to the distinct, curious patterns that seem to be hallmarks of autoimmunity, with one leaping out to me like a thunderclap: psychosis.

Having an autoimmune disease doubles the risk of mental disorders.[44] In fact, mental health issues appear to be a potential side effect of every autoimmune disease. Every. Single. One. From rheumatoid arthritis and lupus to alopecia and Crohn's disease. And, I can attest from personal

experience, detailed extensively in my memoir, multiple sclerosis (just-released 2026 research[45] found a near fivefold increase in dementia for patients with the disease). If Lyme disease is behind psychotic symptoms in autoimmune disease, is it also driving other mental illnesses? A hypothesis that at one point seemed preposterous now had serious legs. And on June 1, 2023, those legs carried me straight to a *Washington Post* article[46] by neuroscientist turned science journalist Richard Sima. The headline about a catatonic woman suddenly awakening was remarkable enough. But it was the subhead that truly made me freeze. "New research suggests that a subset of patients with psychiatric conditions such as schizophrenia may actually have autoimmune disease that attacks the brain."

The patients in his piece suffered from lupus and, after years of being institutionalized, shockingly awoke once they were administered treatment for their inflammatory diseases. Inspired, I began investigating mental illnesses. In 2019, an international team of researchers from the Cross-Disorder Group of the Psychiatric Genomics Consortium had made a startling discovery.[47] The eight major types of mental disorders—anorexia nervosa, attention-deficit/hyperactivity disorder, autism spectrum disorder, bipolar disorder, major depression, obsessive-compulsive disorder, schizophrenia, and Tourette syndrome—were all apparently connected. They appear to share much in common,[48] helping to explain why the conditions frequently co-occur and cluster in families. A December 2025 study in *Nature*[49] supports this finding, only it extends across an eye-popping 14 psychiatric disorders.

I then forced another connection, connecting the unconnected, smushing together mental disorders and autoimmunity. The same patterns that blazed brightest in autoimmunity lit up here too, so much so that the two seemingly separate health conditions are considered major risk factors for one another. If you have an autoimmune disease, beware of psychosis; if you have a mental illness, beware of autoimmune diseases. The similarities border on the ridiculous, punctuated with the same vague generalities that inevitably describe the disorders being caused by "a complex interplay between genetics and environmental risk factors." The pair even, tellingly, have the same triggers. Both autoimmune diseases and mental health problems tend to surface following any one of three notable immune system disruptors: stress, trauma, and illness.

I mulled. Stress, trauma, illness. If that trio wakes up dormant cases of Lyme in its host, it should do so reliably and repeatedly. But again, since Lyme presents so differently, any pattern that might surface would appear as statistical noise, coincidence. Unless…

Unless there is *scale*, massive scale, to reveal patterns that otherwise would have remained hidden, such as mountains of stress from a global depression or significant trauma from a world war.

Or an all-engulfing illness from a pandemic.

Not a Pipe

There is no clear count of how many people have contracted Covid-19, with estimates ranging from 800 million into the multiple billions, but those numbers unquestionably provide scale. The airborne virus has been studied more than nearly any other single health condition with easily a

hundred thousand studies and counting published in just the past five-plus years. If sparks from an illness awaken Lyme disease, that research should be smoking with evidence of a hidden Lyme epidemic.

Sure enough, and it's a raging wildfire. "An important aspect of COVID-19 is a notable increase in the overall incidence and range of autoimmune conditions in individuals after infection," reported a 2023 study.[50] December 2025 research has confirmed that Covid-19 is associated with a "significant" threat[51] of new-onset autoimmune disease, an astonishing 49% increased risk. A separate 2023 study[52] found that a full one-third of Covid patients "develop neuropsychiatric symptoms, such as anxiety, depression, psychosis, brain fog, and suicidal behavior." But nothing points to Lyme's involvement like long Covid, the latest mystery to stump scientists.

Researchers have discovered a mess of clues, but to most it just looks like a mess. For instance, they've discovered that persistently elevated autoantibodies are frequently found in long Covid, typically a hallmark of autoimmune diseases. Indeed, new 2026 research has implicated autoantibodies[53] as the *primary drivers* of long Covid, finding "a causal link between autoantibodies and neurological symptoms" in the disease. But that's not a reliable biomarker, they've determined, as autoantibodies are also seen in a range of conditions,[54] from myalgic encephalomyelitis/chronic fatigue syndrome (ME/CFS) to (notably) chronic Lyme, and even in perfectly healthy people. Inflammation is rampant in long Covid, but inflammation is often rampant in other illnesses and is regularly seen in aging. (A January 2026 study,[55] predictably, connected neuroinflammation to Parkinson's, Alzheimer's, and Covid-19.) Then there's the persistent sex disparity still to resolve. Like most autoimmune diseases, ME/CFS, and chronic Lyme, women are noticeably more susceptible to long Covid. Why? It would all appear to be so puzzling, but appearances can deceive.

The CDC has documented more than 200 symptoms of long Covid,[56] including respiratory and heart symptoms, neurological symptoms, digestive symptoms, joint and muscle pain, and rashes. Conveniently, those symptoms match the description of chronic Lyme disease. The overlap between the two diseases is so remarkably similar, that leading Lyme disease researchers were nearly speechless[57] when they compared them side by side, leading one, Michal Caspi Tal of MIT, to remark, "In terms of clinical presentation, [long Covid and chronic Lyme] look like the same disease even though one is caused by a virus and one by bacteria."

Again and again, and again, researchers repeatedly see the connection, but they can't quite make the link, tripped up by what they believe to be a virus/bacteria paradox. It's akin to staring at a painting of a pipe only to confidently conclude that, "This is not a pipe." Except this isn't a cheeky Surrealist message challenging convention. Given that Borrelia burgdorferi is omnipresent worldwide and can lie dormant in its host for years until an immune system disturbance, long Covid is not so mysterious after all, I realized. It's simply reawakened Lyme disease. That's why some Lyme specialists have reported success treating long Covid with antibiotics—antibiotics that should have zero effect on a virus.

Yikes. I had just stumbled into a hornet's nest. While covered in honey. Naked. After spending two entire chapters dissecting long Covid (Chapters 45 and 46), I came to the realization that if even mild cases of Covid are enough to coax a lurking bacterial infection out of hibernation, *anything* that remotely shakes up the immune system should accomplish the same thing. In science, Reliable Results Repeat, the fourth tenet of SHARDs.

Those results repeat, do they ever. Again and again, and again.

And Again, and Again

There are more than 1,200 terms for marijuana[58] according to Wikipedia. With the way scientists creatively name diseases, Lyme cannot be far behind. The Wiki page for medical syndromes[59] lists 118 starting with the letter M alone, and, as one example, it includes Milwaukee shoulder syndrome—first discovered in 1981 in four elderly women living in Brew City—a strange arthritic disease that primarily affects women and is characterized by inflammation, a familiar combination that sounds suspiciously reminiscent of Lyme disease. But finding every disease and illness that might be misidentified wasn't my focus.

After narrowing my attention to just the aftereffects of potential immune system disruptors, I nearly fell out of my wheelchair. The wide array of mysterious "post" syndromes and post-infectious illnesses that appear following a jostle of the immune system is staggering, just as I had predicted. And without exception, they all mirror the symptoms, even the telltale markers, of untreated Lyme.

If the disease awakens following stress, it stands to reason that post-traumatic stress disorder—an amorphous catchall that disproportionately affects women despite its popular association with combat—likely is just another name for our underlying bacterial infection and should be intimately tied to autoimmunity. It is, confirms a 2025 study.[60] "The risk for developing autoimmune conditions, including SLE, MS, RA, and IBD, is significantly increased in the setting of diagnosed PTSD." Likewise, if trauma awakens Lyme, and giving birth is traumatic, cases of postpartum depression should line up. They do, reported a 2024 study,[61] which confirmed a "robust bidirectional relationship" between pregnancy-related depression and autoimmune disease.

Post-infectious irritable bowel syndrome is among the most common of these syndromes, affecting an estimated 10% of people following a gastrointestinal infection, striking women at far higher rates than men. The risk of developing IBS increases sixfold after infection and can remain elevated for years.[62] What follows mirrors the broader pattern exactly: persistent sub-clinical inflammation and autoantibodies. Researchers have found that IBS patients carry distinct autoantibodies that distinguish them from healthy controls,[63] including, notably, antibodies to bacterial flagellin, a protein Borrelia burgdorferi carries in abundance. The risk of psychiatric illness rises too—IBS patients carry a 3.6-fold increased risk of developing depression, anxiety, bipolar disorder, and sleep disorders, with elevated risk persisting for more than five years.[64] It is not a disease of the gut. It is a disrupted immune system that just happens to express itself there.

I tried to find an exception, any exception, to the trauma/stress/illness trigger rule. Science wasn't having it. Researchers kept making the connection no matter where I looked, always careful to note that correlation does not mean causation. I learned an awful lot.

There's something called breast implant illness (BII), which can arise after breast enlargement surgery. You don't have to guess at the constellation of symptoms that follow (some patients report similar symptoms after breast reduction surgery). Then there's sepsis. Up to half of all survivors go on to develop post-sepsis syndrome[65] (PSS)—"a complex mix of physical, cognitive and psychological symptoms"—a complication that looks identical to BII, including "overwhelming fatigue, chronic pain, muscle weakness and disrupted sleep" as well as severe cognitive problems. Even minor, self-inflicted injuries from body art—tattoos and piercings—can trigger a range of immune responses as this January 2026 study[66] highlights. It's ubiquitous and unmistakable, and more than a little unnerving.

It goes without saying that most people don't believe that breast augmentation triggers psychosis, fender benders cause RA, or that tattoos are at the root of MS. People still get plastic surgery, drive cars, and get inked. But there is one reliable immune system agitator that doesn't enjoy the same reasoning—one I'd have preferred to avoid entirely. I couldn't. The science wouldn't let me.

Vaccines.

Two Rights

On a May day in 1796, English physician Edward Jenner inoculated 8-year-old James Phipps with a vaccine for smallpox, the world's first successful vaccination. Since then, vaccines have saved countless lives—at least 154 million in just the past 50 years.[67] But today, public skepticism about their effectiveness and safety has taken center stage, with a growing contingent of people who believe they are responsible for the recent rise of chronic health conditions, particularly in children.

What's the truth? It's complicated.

The Lyme-vaccine connection should have remained undiscovered, but Covid-19 changed that. With more than 5.5 billion people vaccinated against the viral infection, there was finally the scale and necessary time lapse needed to start detecting patterns. If the Covid vaccine is triggering dormant Lyme disease, then a subset of recipients should come down with what resembles autoimmune disease and other health conditions tied to the bacterial infection, including long Covid (catching Covid not required). And emerging research suggests they just might, with formal studies underway. Researchers remain wary.

One 2022 study[68] found an array of concerning health issues, including the onset of autoimmune diseases, but cautioned that, "whether the association between COVID-19 vaccine and autoimmune manifestations is coincidental or causal remains to be elucidated." Additional case series support these findings, but they also universally conclude that no firm conclusions exist, and that regardless, getting a vaccine is statistically healthier than avoiding one. Researchers are desperately trying to avoid making conclusions that might disturb this minefield.

This is a unique situation where both parties are partially correct. To the relief of scientists, all evidence points to a solitary conclusion: vaccines, including newer ones developed by revolutionary mRNA technology, are generally safe and responsible for the improved health of billions. And to the vindication of those who've questioned vaccine safety, all evidence points to a definite connection between vaccines and unexplained health issues. The apparent paradox resolves cleanly once Lyme is in the picture.

Vaccines themselves are not the problem—they've never been the problem. But anything that stirs up the immune system is a potential trigger for awakening Lyme disease, making routine vaccinations regular triggers. You can't blame frightened parents for refusing to let their children get inoculated against infection when they see sporadic cases of illness and disease unexpectedly pop up after recommended shots. However, these parents inadvertently are doing their children a disservice. If they or their children are infected with a dormant case of Lyme, avoiding vaccines dodges only a single trigger. Stress from a teenage breakup, a fender bender in the grocery store parking lot, an errant soccer ball to the noggin, or a case of strep throat could each just as easily be a trigger. Or worse, so would coming down with the very illness the avoided vaccine was meant to shield the recipient from, a compounding threat when both the illness targeted by the vaccine *and* active Lyme disease develop.

Bottom line: if a single spark from a vaccine is enough to awaken a sleeping case of Lyme disease, it's not so much a question of *if* Lyme is going to make its unwelcome appearance in that person, but when. And it doesn't matter if you are a stay-at-home parent in New Jersey or a world-class athlete. Eschewing a vaccine—whether it's to protect against Covid-19, the flu, HPV, tetanus, or any of the other 20+ diseases they are designed to ward off—would then only delay the inevitable.

Here's where it gets seriously sticky. There has been a recent push to use the "gold standard"[69]—randomized, placebo-controlled trials—to "prove" which faction is right. It will only prove disastrous. Any competent scientist can play out what will happen next (aside from the ethics of withholding a vaccine for the placebo arm of the trial). The group that receives an authentic vaccine—any vaccine, but especially those that trigger a strong immune response—will experience more chronic health issues than the group that receives the placebo saline injection, leading researchers and the public to confidently—and erroneously—conclude that vaccines are the culprit. Without accounting for Lyme disease lurking in the populations of both groups, evidence-based medicine cannot solve this impasse, and the distrust of vaccinations will skyrocket, a crushing setback to one of the greatest medical advances in history.

But all is not lost. Unbeknownst to me at the time, da Vinci foretold science's failures and provided a seminal roadmap to navigate around them: Seek the big picture, find the patterns, discover the truth.

—Chapter 4—

The Connectedness of Everything

Don't put all your eggs in one basket, warns the popular idiom. So, I did exactly that. I now had a raft of hypotheses floating out there. The entire lot of autoimmune diseases are misdiagnosed cases of Lyme disease, most mental illnesses are rooted in the bacterial infection, and all post-infectious illnesses, including long Covid, have a common cause. Add in the vaccine connection, and I had a witch's brew of bold theories that was near impossible to swallow. Perfect.

Blindly trusting that my assessment of autoimmune diseases was correct, I applied that insight to all health conditions, using it as a barometer. Any medical issue closely associated with autoimmunity—whether an autoimmune disease routinely precedes a health problem or predisposes an individual to a future diagnosis of one—then meant Lyme disease must be playing an outsized role in said malady. Lyme might not be the sole cause, but it assuredly is a starring cast member. As I settled into the research, starting randomly with birth defects, my head started spinning.

"You can say that people with Down syndrome are the largest human population with a predisposition to autoimmune disorders," said Joaquín Espinosa, PhD, director of the Linda Crnic Institute for Down Syndrome at the Colorado University Anschutz Medical Campus in a 2018 interview.[70] Epilepsy, thyroid diseases, blood cancers, diabetes, heart issues, obesity, Alzheimer's, psychosis, celiac disease, and more accompany the disorder thought to have purely chromosomal origins. But is that the whole story?

Many children with Down syndrome have autoantibodies, discovered researchers in a study that looked at the role of heredity in Down syndrome.[71] "We were not surprised when 50% of children with Down syndrome from families harbouring an autoimmune disorder had antibodies." Scientists have long believed that these autoantibodies "can cause autoimmune diseases, and, increasingly, autoimmunity has been found to be associated with a wide range of diseases, such as cancer, infectious disease (such as COVID-19), cardiovascular disease and neurodegenerative disease," said a 2022 study.[72]

The autoimmunity pattern found in Down syndrome was merely a preview of other birth defects. A 2021 Norwegian study[73] looked at the health of more than 1.3 million children, 3,575 of them with cerebral palsy, as well as the health of the mothers and fathers, discovering that "mothers with any autoimmune disease had a 40% increased risk of CP in their offspring." But it was an October 2023 study[74] by Yu-Hsuan Joni Shao and Yi-Ming Chen—finding that a mother or father with an autoimmune disease notably increases the risk that their children will develop an autoimmune condition, the risk rising even more if both parents are affected—that is eerily consistent with in utero transmission of Lyme. "This study demonstrated broadly how autoimmune diseases pass

from parents to infants of both genders and separately quantified the maternal and paternal contributions to disease," concluded the researchers.

The CDC has already acknowledged that this is a possibility in Lyme disease, but the agency has long believed the bacteria spreading from mother to child is a rarity. Science, however, would beg to differ.

Replace "autoimmune" with "Lyme" and let that sink in. If a mother or father has Lyme disease, the risk that their child will develop Lyme soars. The research into the parental influence on autoimmunity, birth defects, and mental disorders is mind-bending. Lyme disease varieties must be spread without hikes in the woods or visits to grandmas in New Jersey. Spirochetes are being passed along by infected mothers, and fathers appear to play a roll as well. When years or decades later, some of those children develop the same autoimmune condition as Mom or Dad, it's considered to "run in the family." *That's* why so many autoimmune diseases (re: Lyme) appear to have a "hereditary" component.

Genetics and faulty DNA are not to blame. Moments later, I landed on the reason "environmental factors" also routinely get fingered as a contributing cause of innumerable health conditions. Unexpectedly, spousal health is a surprisingly strong predictor of a partner's future health conditions. "Some chronic illnesses such as diabetes mellitus, coronary heart disease, stroke, depression, and dementia are shared between couples," reported an October 2023 study.[75] For diabetes alone, the risk to the spouse increases up to 40%, leading researchers to speculate, as so many others have, that "the spousal concordance of chronic illnesses may be due to shared environmental and behavioral characteristics."

An appropriate, educated guess, but the environment or the behaviors spouses share aren't to blame, either. Once the protective halo of a "tick-borne" infection was removed, another transmission method crystallized. The unconfirmed fears raised in a 2014 study,[76] which found Borrelia spirochetes in semen and vaginal secretions of Lyme disease patients, appear to be well-founded. Lyme isn't just spreading by birth, it must also be spreading by either sex or saliva, or both—transmission routes that remain under-studied but are biologically plausible given Borrelia's behavior in other bodily fluids. (And because the disease can lie dormant for years, decades, it would easily evade detection, unlike other STIs that follow predictable epidemiological sexual network patterns.) Ticks, long synonymous as the lone vector responsible for passing along the bacterial infection, play, at most, a minor supporting role in the disease's global march.

The evidence from Brazil makes this undeniable. Researchers there spent 30 years documenting a Lyme-like illness[77]— eventually named Baggio-Yoshinari Syndrome—transmitted by tick species that don't reliably bite humans, in a country where the standard Ixodes ticks responsible for Lyme in the Northern Hemisphere don't exist. Twenty-five thousand cases reached a single São Paulo hospital anyway. Brazilian researchers named the syndrome after themselves because international journals refused to publish findings that didn't fit Northern Hemisphere dogma—the Semmelweis reflex, in Portuguese.

The exotic vector isn't exotic at all. It's the same one operating everywhere else.

Women are infecting men. Men are infecting women. And women are passing along spirochetes to their children. Lyme disease has been spreading this way for thousands and thousands of years. Medical experts from the world's best-regarded institutions have been looking at it all wrong.[78] No wonder scientists have concluded that there is a genetic component to so many diseases, but that the connection had been too fuzzy to be the sole contributor. No wonder scientists blame an out-of-whack immune system, but the presence of suggestive markers like autoantibodies has been maddeningly inconclusive. And no wonder scientists have insisted that there must be an environmental factor, but could never make conclusive heads or tails of the inconsistent geographic spread of diseases.

No wonder.

Understanding how Lyme spreads unlocked mysteries that should have remained forever hidden, as the disparate puzzle pieces started snapping into place at a pace so furious that I struggled to catch my breath.

A Fizzled Revolution

Now I could turn back and apply what I'd learned to the unwieldy tangle of hypotheses I was currently juggling. For instance, if genes aren't in play, and if Lyme spreads primarily by sex and birth, how does that reshape what we know, or what we think we know, about a specific disease, say lupus? So I buried myself in more medical hypotheses, only to realize that many are far more tenuous than I had originally believed.

For the last 20 years, scientists have been debating the influence of genetics, with some calling it an abject failure[79] as gene therapy continues to come up short[80] despite sky-high hopes. Perhaps even the entire approach is flawed, others argue. They don't reliably predict disease,[81] the "risk scores" are a crapshoot,[82] and heritability is grossly overstated.[83] We can map the genome, but the "Genomic Revolution" promised in the early 2000s has fizzled like other wannabe game changers of the era: the Segway, Napster, and the Microsoft Zune. Even so, investment in the technology continues to surge,[84] projecting to crest $175 billion by 2034 in the U.S. despite the sketchy footing.

There is an open secret among geneticists: genes matter far, far less than you might think, accounting for less than 2%[85] of the longevity equation. (It should be noted a new twin study released in January 2026 in *Science*[86] disputes this; their contradictory findings are explainable, which I address later.) Genes also are rarely responsible for cancer, as only 0.3% of the population carries genetic mutations[87] thought to substantially raise cancer risk. New research on identical twins[88] found that intelligence doesn't seem to be genetic, either. IQ differences between identical twins were largely explained by differences in education, not genetics.

The same flawed logic extends to obesity: a study of 86,000 Norwegian children concluded that genetics explains an estimated 79% of the mother-child BMI link and a remarkable 94% of the father-child link.[89] The researchers pointed to the paternal figure as especially compelling evidence

for genetics, since fathers don't share a uterine environment with their children. But fathers do share a bed with the mothers who transmit Borrelia. The "genetic" signal evaporates the moment you introduce an infectious explanation.

The pattern holds even at the extremes of cognitive performance. A 2026 multisite study of adults over 80 with youthful memory,[90] so-called SuperAgers, found that neither APOE, the strongest known Alzheimer's genetic risk factor, nor any contemporary genetic risk score predicted who achieved exceptional late-life cognition. If genetics drove extraordinary aging, the strongest genetic signal in all of Alzheimer's research would show up here. It does't.

Race is technically not genetic,[91] a fact known to experts in the field for the past 50+ years. Skin color is passed down through families and shaped by where your ancestors lived—but the racial categories we use don't correspond to any meaningful genetic differences. That got me thinking… If lupus is not genetic and is instead being driven by an infection, that would mean the infectious agents—the spirochetes that fuel Lyme—are being passed down through generations. And that realization helped unlock another grand mystery.

Lupus sufferers are largely Black. If that particular strain of Lyme disease is circulating primarily in the African American community, following the rule of ancestral geography, it almost certainly originated in Africa generations and generations ago. In the US, cases of lupus are concentrated in the South, which now makes complete sense. The distribution of lupus worldwide follows a distinct path instantly recognizable by anyone familiar with world history. Cases of the autoimmune disease are concentrated in former major hubs of the slave trade—Brazil, the Caribbean, London, the Middle East, and parts of Asia. According to the data, and the science, lupus has been spreading worldwide this way since the 1500s. (An added wrinkle: Central/West Africa is awash in ticks and Borrelia, which would make it a leading suspect where this strain of the disease originated in humans.)

I couldn't believe I was the first to recognize this distinct, obvious pattern. That's because I wasn't. In a 2011 study,[92] researchers at the Medical University of South Carolina mapped lupus prevalence in Afro-Caribbean and West African populations and explicitly traced the disease burden along slave trade routes from Sierra Leone to Charleston—the same geographic thread I had independently followed. They stopped short of an infectious explanation, attributing the pattern instead to "genetic susceptibility." But with Lyme in the picture, the pattern needs no genetic explanation at all.

I took it further, letting out my fishing line until it was nearly fully unspooled. Since race isn't genetic, other diseases with similar racial disparities, therefore, should follow the same pattern. They do, and effortlessly. They also should be closely related. They are.

Half of all patients diagnosed with lupus develop lupus nephriyis,[93] which can progress to chronic kidney disease, which affects an estimated 10% of the general population,[94] about 800 million, mostly minorities. Where are cases of CKD most concentrated? In old hotspots for the slave trade. Drilling into the studies of each disease leaves little doubt that the two are closely related. But if we

travel down this racially influenced rabbit hole, there is another unexpected twist, one that is poised to upend the entire field of genetics. Sickle cell anemia.

In the United States, about 1 in 12 African Americans carry the sickle cell "trait" and approximately 1 in 500 get diagnosed with full-blown sickle cell disease[95] (there are multiple forms, the anemia version the most common and severe). Like chronic kidney disease, it's not just a "Black" disease. In the US, cases ripple through the Hispanic community, a minority also disproportionately affected by lupus, and its distribution is concentrated, unsurprisingly, in the South (1 in 1,100 compared to 1 in 32,000 in Western states[96]). SCD occurs when a person inherits two mutated forms of hemoglobin, one from each parent.

According to our understanding of genetics, that would make it appear to be an inherited disease, passed down from Mom and Dad. But here's the twist: sickle cell shares so many similarities with lupus, from geography to symptoms, that practitioners are repeatedly warned[97] about how easy it is to miss the autoimmune disease in cases of SCD. It's not just lupus. A November 2024 study[98] found that people with SCD are noticeably more likely to have an autoimmune condition than the general population, and even those without a diagnosed autoimmune disease were often flooded with various autoantibodies.

Even so, geneticists and sickle cell experts will point to clear evidence of an "evolutionary benefit,"[99] as carriers of one copy of the sickle cell trait have an unusual degree of protection from malaria, common in Africa, where the gene is thought to have evolved about 20,000 years ago. That would then mean for my theory to be correct, that Lyme disease is behind sickle cell anemia, Borrelia spirochetes would have to somehow protect against malaria. That sounds absurd, of course. So naturally, they do.

In 2010, researchers made the astonishing connection.[100] Of the millions of cases of malaria reported annually, they found that there is "alarmingly, a high prevalence of concomitant infections" with malaria parasites and Borrelia spirochetes, specifically relapsing fever Borrelia, the African cousin of the Lyme-causing strain. Researchers then studied the combination using a mouse model and to their surprise, they "observed a 21-fold increase in spirochete titers, whereas the numbers of parasitized erythrocytes were reduced 15-fold," explaining the illusion of an evolutionary benefit. They also discovered that a malaria infection could wake up a dormant Borrelia infection, "the first evidence of a clinically and biologically relevant cue for reactivation," confirmation that an illness, from the likes of malaria to Covid, can indeed roust dormant spirochetes. But the connection between malaria and Lyme disease goes back nearly 2,000 years. Here is where the second tenet of SHARDs comes into play, History Harbors Hints.

Galen, the celebrated Roman and Greek physician, was a foundational medical researcher, advancing the fields of anatomy and pharmacology to neurology and pathology. He was among the first to document that the high fevers of malaria were able to cure psychosis, a revolutionary side effect later recognized by doctors in the 19th century. It was such a powerful tool to treat mental illness that malaria was purposefully introduced to patients suffering from insanity, a process called malaria therapy,[101] or malariotherapy, to treat the spirochetal disease neurosyphilis. Austrian

physician Julius Wagner-Jauregg developed the technique in 1917, and a decade later it won the Nobel Prize. Eventually it fell out of favor due to the high risks and the development of penicillin, which is effective in syphilis (and, surprise, penicillin is also standard of care in sickle cell disease,[102] a curious fact for a condition presumed to be entirely genetic).

The fever-as-medicine connection runs deeper than malariotherapy alone. As far back as 1868, German surgeon Carl Busch documented[103] that a patient's advanced facial tumor dramatically regressed after she contracted a severe febrile infection—the first recorded case of fever eliminating cancer. By 1891, American surgeon William Coley was deliberately inducing fever in cancer patients using bacterial extracts, achieving a 64% remission rate in otherwise inoperable cases.[103] There's a completely reasonable explanation this unorthodox treatment works.

Unbeknownst to doctors, those high fevers were likely treating Lyme disease. In 2017, researchers discovered that extreme temperatures can incapacitate Borrelia burgdorferi bacteria.[104] They become immobile at 102.2°F and die if temps reach a minimum of 106.9°F—for at least 2 hours. (But pyrotherapy,[105] artificial fever, for Lyme is highly controversial and rarely used because death of the host can be a side effect.)

The phenomenon hasn't faded with time. A 2023 review[106] documented fourteen cases of spontaneous cancer remission following Covid infection—including solid tumors and lymphomas that disappeared for up to a year without treatment—with fever identified as a key contributing factor. A 2022 case report[107] described complete remission of acute myeloid leukemia following severe Covid infection, with no cancer therapy administered at all. A 2025 case report[108] documented a non-Hodgkin's lymphoma tumor vanishing entirely within four months—again, without treatment, and again following fever. The immune storm that Covid triggers, it appears, occasionally takes out more than the virus.

With sickle cell's genetics origin now in serious jeopardy, I turned my attention to another genetic claim—arguably the most famous one of all—BRCA, the so-called breast cancer gene.

Breaking BRCA

In 2013, Angelina Jolie underwent a preventive double mastectomy after testing positive for a BRCA1 mutation, a decision that made BRCA a household name and was widely celebrated as a triumph of genetic medicine. Women with a BRCA1 mutation have more than a 60% lifetime risk of developing breast cancer,[109] compared to about 12% for the general population. If that isn't a gene causing cancer, what is?

Look closer. Fewer than 10% of all women diagnosed with breast cancer carry a BRCA mutation,[110] meaning the vast majority of breast cancer occurs in women with no BRCA abnormality whatsoever. What BRCA actually does is impair a cell's ability to repair damaged DNA,[111] leaving it vulnerable to errors that accumulate over time. It doesn't cause cancer. It lowers the threshold for it—a susceptibility factor, not a sentence. And what's damaging the DNA in the first place?

Jolie's story didn't end with the mastectomy. Years later she developed Bell's palsy—facial paralysis that doctors attributed to stress, a known Lyme trigger—followed by high blood pressure. She reduced her risk of one Borrelia-driven outcome while the infection continued its work unimpeded. The breasts were removed. The bacteria stayed.

Researchers studying the intersection of lupus and cancer[112] made a striking discovery: lupus autoantibodies are selectively toxic to BRCA2-deficient cancer cells—and SLE patients, flooded with autoantibodies, show a 37% lower risk[113] of the very cancers BRCA mutations are supposed to cause: breast, ovarian, and prostate. The researchers themselves raised the possibility that lupus autoantibodies may be partly responsible. But this isn't protection—it's competition.

The Lyme strain driving lupus is already running its own immune war, and that war happens to target the same cellular vulnerabilities that the Lyme variant behind BRCA exploits. The lupus-driven infection's cancers—leukemia, lymphoma, blood cancers driven by chronic inflammation—get first dibs. BRCA's cancers get crowded out. But here's where the evidence turns damning.

Earlier I detailed that lupus, sickle cell disease, and chronic kidney disease come from the same variant of Lyme disease. That would then mean that they all should offer similar defense against the cancers BRCA fuels. They do. In sickle cell disease, solid tumor rates fall 38%[114] while blood cancers rise, and in chronic kidney disease, prostate cancer rates drop[115] while urinary cancers climb. Three related conditions. The same narrow window of apparent protection. The same elevated risk elsewhere. That is not coincidence. That is a disease leaving its signature across every population it inhabits.

BRCA is not an isolated case. It is a window into a much larger problem—one that researchers have been reticent to look through despite years of mounting evidence.

Across decades of research, thousands of genetic variants have been linked to chronic diseases through genome-wide association studies, the massive population-level scans that form the bedrock of modern genetic medicine. These studies have identified associations with everything from Alzheimer's to lupus to heart disease to depression. The assumption underlying all of it is that these variants are inherited blueprints—the body's own code, passed from parent to child, quietly predetermining disease. The entire apparatus of genetic risk scores, predictive testing, and gene therapy rests on that assumption.

But what if many of them aren't inherited blueprints at all? What if they are something else entirely—the molecular footprints of a chronic, multigenerational infection, leaving behind marks that look, in every population study ever conducted, exactly like genetic destiny?

This is not speculation. Researchers have now documented that Borrelia burgdorferi systematically alters DNA,[116] with changes concentrated specifically in HLA genes—the very genes most consistently flagged as "genetically associated" with autoimmune diseases across dozens of conditions. Alter these genes across a population infected for generations, and what you produce, in every downstream study, looks indistinguishable from inherited genetic risk.

It isn't. It's an infection's rewrite of the host's operating instructions, passed down not through DNA sequence but through cellular memory that can be transmitted to the next generation.[117] A child born to an infected parent may inherit not a disease gene but an infection-altered epigenome—and every population geneticist who studies that child's family will conclude, incorrectly, that the disease runs in the family.

The methodology compounds the problem. Some genome-wide association studies used to identify these "genetic" associations have documented false positive inflation so severe[118] that one analysis found rates tens of thousands of times higher than the field's own acceptable threshold. The mountain of genetic disease associations appears to be built, in significant part, on a foundation that the field's own researchers have acknowledged is unstable.

The genetic revolution promised to decode the blueprint of human disease. We now have a good idea why it keeps coming up short. There are many strains of Borrelia all over the world, at least 20,[119] and they "show complex region-specific trajectories," confirms a November 2025 study published in *Nature*.[120] Additionally, there are 29 strains of Relapsing Fever,[121] which can also cause lasting neurological damage if left untreated.

We've been painstakingly mapping the molecular traces of Borrelia burgdorferi this entire time.

Sickness Spreads

Good luck trying to find a "genetic" disease that doesn't have close ties to autoimmunity.

Turner syndrome[122] is closely associated with autoimmune thyroiditis and inflammatory bowel disease, with autoimmune thyroid disease affecting roughly 40% of patients and celiac disease occurring at four to eight times the general population rate. Fragile X syndrome[123] is marked by mitochondrial dysfunction and oxidative stress—and metformin, of all drugs, has shown surprising promise[124] in alleviating its symptoms, for reasons that will become clear. Klinefelter syndrome[125] carries a 14-fold increased rate of lupus compared to typical males—the same lupus that predominantly affects women—alongside elevated rates of rheumatoid arthritis, autoimmune thyroid disease, insulin resistance, and metabolic dysfunction. In 2024, when researchers tried genetic mapping across autoimmune conditions,[126] they found patterns that "indicate widespread sharing of pathogenic mechanisms but not a single global autoimmune mechanism."

The mechanism is there, they just haven't been able to see it. And it extends to cancer, one of humanity's leading killers.

Autoimmunity and cancer are deeply intertwined. Study after study confirms and reconfirms that there is "a pervasive, largely positive association between … autoimmune and inflammatory diseases and subsequent cancer development."[127] Cancer and autoimmunity are so tightly interwoven that each research study appeared to come with a mandatory infusion of autoantibodies, the link so strong that those antibodies are being studied as diagnostic cancer biomarkers.[128]

The cancer-Lyme connection runs deeper than association alone. Among the cancers most linked to autoimmune disease: non-Hodgkin's lymphoma, bladder cancer, breast cancer, kidney cancer, and lung cancer not attributable to smoking. Maternal autoimmune disease specifically has been associated with increased childhood cancer risk. And in perhaps the most striking finding, doxycycline—the frontline antibiotic for Lyme disease—has been found to prevent breast cancer recurrence, eradicate cancer stem cells,[129] and inhibit cancer cell proliferation,[130] Scientists have attributed these effects to doxycycline's "anti-inflammatory" and "anticancer" properties. The simpler explanation has been right under their noses for fifty years. The full, unabridged argument is documented in the companion memoir *Sit Down Before Reading*.

Gasping for better news, I started to look everywhere for a silver lining, but mostly I found evidence of Lyme disease lurking in the shadows of countless health conditions. Everything is connected to autoimmunity, the bidirectional relationship unmissable.

ALS, likely[131]—and its spousal concordance all but confirms sexual transmission. Parkinson's disease, likely.[132] Autism, likely[133] (spoiler: it's not vaccines). Chronic kidney disease, likely.[134] Anorexia and other eating disorders, likely.[135] Obstructive sleep apnea, likely.[136] Migraine, likely.[137] COPD, the fourth leading cause of death worldwide, likely.[138] Hypertension, affecting nearly half the population, likely,[139] so likely that some scientists believe it is an autoimmune disease.[140] Even periodontitis—gum disease—is now linked to autoimmune disease and elevated all-cause mortality, a finding confirmed across nearly 445,000 participants in the US and UK.[141] The connections keep mounting and scientists keep finding them—"Surprising links between autism, Alzheimer's could change how we treat both,"[142] trumpets an April 2026 article in *The Washington Post*; each time they are universally surprised.

The avalanche of research supporting the autoimmune/Lyme disease connection is crushing, the evidence overwhelming. From mental illness to metabolic disorders, from cancers to long Covid, from genetic diseases to birth defects, and from addictions to a spate of devastating rare illnesses, even the epidemic of unexplained chronic pain,[143] looks to have an autoimmune basis. More revealingly, all are awash in autoantibodies and are deeply tied to what has been deemed "autoimmunity."

To make sense of this, now consider what it might look like if a stealth pathogen were responsible. If they've evolved to hide deep within human cells or tissues, forming biofilms (essentially protective shields) or enter a latent state, they would be nearly impossible to detect with standard blood tests. If such a pathogen were to hide *inside* a specific organ (like the thyroid), the antibodies sent to kill it would look like they are attacking the organ itself. That would mean that an "autoantibody" is actually merely a standard antibody just doing its assigned job against a hidden target.

Today's researchers have been dutifully trying to connect the dots—it's unquestionably all tied together—they're just trapped by a broadly accepted theory that defies common sense.

Medical history is replete with sensible-at-the-time-but-later-debunked theories trying to explain the unexplainable. Tooth worms as the cause of toothaches. Phrenology, or the belief that a skull's size and shape determine personality and intelligence. The bright idea that diseases are caused by "bad air" or miasma. Alas, the theory of autoimmunity, the body randomly attacking itself with unrelenting gusto, is poised to handily eclipse them all. It doesn't just defy Darwin's famous theory, it makes an outright mockery of it.

If autoimmunity and its meteoric rise in the past half century were real, it would represent an evolutionary paradox demanding clear explanation—not necessarily impossible, but requiring us to identify what selective advantage could possibly offset such obvious fitness costs. Studying the origins[144] of this misguided theory is a lesson in the price of making broad assumptions, a lesson that nauseatingly repeats in this story.

Generational

The way lupus gradually proliferated from the 1600s to today gave me the first solid clues as to how Lyme was impacting generations. I understood how the lupus strain of Lyme had bloomed worldwide through the slave trade, chiefly during the 1600s, 1700s, and 1800s, but it didn't explain the majority of Lyme cases, many concentrated in Europe, and later, North America. Since people, not ticks, are Lyme disease's primary vectors when it comes to infecting humans, I turned my attention to history's major movements of humanity, the involuntary relocation of Africans being the bellwether. I knew that Lyme had already been spreading in Europe for thousands of years—Ötzi the Iceman, a 5,300-year-old mummy unearthed in 1991 in the Alps, had the disease[145]—but it must not have been until the Great Atlantic Migration, beginning in the 1840s, that the ancient European plague spread in earnest Stateside. Evidence suggests that it quietly spread until it hit the epidemic trifecta: WWII, the Baby Boom, and the advent of international air travel.

While WWI certainly contributed, the second World War saw tens of millions displaced in Europe alone, many arriving on US shores seeking refuge. With Lyme spreading sexually, it stealthily infiltrated populations, its march accelerated by the unprecedented explosion of babies during the '50s and '60s. The free love movement of the late '60s and '70s didn't help matters. And then the final domino fell. As airplanes started crisscrossing the globe with abandon, any chance of containing the disease would have all but evaporated. At least that was my hypothesis. For the theory to hold, history had to cooperate. Specifically, four things had to be true.

- Signs of an emerging health problem should have started developing in the 1930s, the first substantial generation of Lyme sufferers. Even so, it would have largely gone unnoticed.
- Beginning in the 1950s, as Lyme's spread accelerated, red flags should have started appearing. Autoimmune diseases, once rare, would be ticking up along with less common medical issues.
- By the 1970s, virtually every type of health problem should be rising, including rates of chronic disease.

- From the late '80s onward, every subsequent generation should experience a notable, substantial increase in a medley of medical setbacks, the relentless spread of sickness felt most acutely in the youngest generations.

That's exactly, *exactly*, how the Lyme epidemic has unfolded. This evolution and spread of Lyme align so perfectly that our youngest generation has earned an ominous nickname: Generation C, for cancer, due to unexplained increases. And young women are much more affected than men. From ages 15 through 49, researchers have found that women have a cancer rate that is a stunning 83% higher than men.[146] Every recent generation has seen a dramatic uptick in cases.

A July 2025 study in *JAMA*[147] confirms the soaring rates of sickness. "From 2011 to 2023, the prevalence of 3- to 17-year-old individuals with a chronic condition rose from 39.9% to 45.7% … and from 25.8% to 31.0% within the general population. Rates of obesity, early onset of menstruation, trouble sleeping, limitations in activity, physical symptoms, depressive symptoms, and loneliness all increased during the study period." But evidence of this once simmering, now boiling epidemic goes back more than a century, and astute researchers like University of Copenhagen epidemiologist Thorkild I.A. Sørensen have noticed.

In a 2023 journal article, later covered in *Science*,[148] Sørensen pushed back, hard, on the idea that "cheap, highly processed, and calorie-rich foods, as well as increasingly sedentary lifestyles and growing portion sizes" in the 1970s is to blame for today's obesity epidemic and spate of chronic illnesses. He found proof that the world's waistline started growing steadily far earlier—beginning in the 1930s, decades before documented food processing and lifestyle changes—undermining the entrenched couch-potato/ultra-processed assumption.

The opposite should also hold true. Leaning on the third tenet of SHARDs, Answers Await in the Anomalies, mysterious pockets of wellness should exist, and they should have identifiable patterns.

If sexual contact is a risk factor for Lyme, it should follow that having zero partners would equate to improved longevity, and that the opposite—lots of partners—would amplify risk. The research on the topic is especially, ahem, revealing.

"Ministers, priests, vicars, nuns and monks live much longer, and healthier, than their flocks," scientists have discovered,[149] with mortality rates 25% or more lower than the general public. Not only that, "many of the religious groups had far less disease, including heart disease and cancer, than other people." It was proposed that this gulf was due to diet, but nope. "This has been discounted as many now work and live in the community and share similar diets to those of the average population." So, it's not environment, either.

It's just sex. Which explains why 2020 research[150] found that men with 10 or more partners were nearly 70% more likely to develop cancer than those reporting zero to one partners and, cementing the Lyme pattern, women nearly *91% more*. But researchers scoffed at the notion that sex could cause cancer. A Harvard scientist downplayed the association, citing that those with more sexual behaviors are likely engaged in riskier behavior. "While future research could find previously

unidentified risks in having a higher number of sexual partners, we already know enough to explain the connection."

Researchers think they know enough. After all, sex causing cancer would go against the theory of evolution. Like autoimmune disease. Oh wait.

Abstinence clearly is not a sustainable solution. And even so, as much protection a sex-free life can offer, it can't control for infected parents who pass the disease down. That requires multiple generations passing down disease-free children. So let's find them.

Since fellow humans pose the most risk, isolation would be a significant advantage in containing Lyme's march through an area, most likely islands, remote villages, or other regions difficult to access. Romantic partnerships primarily within the community, rather than with outsiders, would severely limit the sexual spread of the disease. That likely would mean an insular group or society, typically something found in a tight-knit, older, or religious community. Scientists also would not be able to distinguish a reliable pattern from dietary interventions, lifestyle, or environmental influences that would point to successful aging. To rule out genetics as a factor in aging well, siblings, children, and grandchildren no longer living in these areas, and therefore more exposed to contracting Lyme, would be less likely to reach 100. Lastly, the environment would be largely devoid of blacklegged ticks, a known vector for the disease. Being isolated and insular cannot protect against an enemy from within.

All of that describes, to a T, the mystical, elusive areas known as Blue Zones, regions of the world where people are measurably healthier and live longer. Renowned for their unusual concentration of centenarians, Blue Zones have been a source of study for years as scientists have struggled to crack their longevity code. Diet was thought to be the missing link. But just as in the study of centenarians, there is no apparent rhyme or reason for their unparalleled success at breaching the century mark. It's a cavalcade of confounders.

Centenarians and people living in these remarkably healthy regions are just as likely to reach 100 whether they follow a low-fat vegetarian diet or partake nightly in frozen TV dinners. They are just as likely to be couch potatoes as daily exercisers, just as likely to be loners as social butterflies, just as likely to be imbibers as teetotalers. In fact, spouses share in the longevity advantage of centenarians[151] nearly as strongly as biological relatives[152]—a finding that makes no sense if genetics were the driving force. It's not diet, social interaction, or the complete avoidance of carcinogenic vices. It's not sunshine, the gradient of latitude, or the absence of 5G cell phone towers. It's not lax hygiene in childhood that uniquely builds a stronger immune system. It's none of that, and that, in and of itself, is a pattern, a clue.

Now to tie it all together.

—Chapter 5—

Seeking a Utopia

Despite a plethora of diet books espousing the benefits of Blue Zone eating habits, the science has never quite added up. There's the seafood-heavy diet of Okinawans, the Mediterranean diet of Sardinians, and the vegetarian diets of the Seventh-day Adventist church in Loma Linda, California, diets all drastically different from one another. And there's another problem: the health benefits of living in many of these zones appear to be waning.[153] Although older residents still enjoy longevity, younger generations—with the exception of those residing in extremely closed communities—decidedly don't, as if the advantage has disappeared.

If their rising health issues are largely the result of the relatively recent assimilation with the broader world and not due to aging, we should find pockets of this phenomenon in those rare communities that have not yet assimilated. If such a place sounds like it would be an Indigenous community residing deep in the Bolivian Amazon or in the wilds of a Malaysian jungle, you would be correct.

In June of 2025, a new study by researchers at Columbia University[154] found that "inflammation, long considered a hallmark of aging, may not be a universal human experience." Reporting in October 2025 for *The Washington Post*, Dr. Richard Sima explained[155] that researchers have long believed persistent, low-grade inflammation—"inflammaging"—is a universal hallmark of aging, linked to diabetes, heart disease, and Alzheimer's. The Columbia study upended that assumption.

That study compared populations from two industrial nations (Italy and Singapore) with two Indigenous populations (the Tsimane and the Orang Asli) and got a bit of a shock. Despite high levels of expected inflammation from jungle parasites, this inflammation "did not increase with age and, crucially, did not lead to the chronic diseases that plague industrialized societies." Then there was the kicker. "In fact, most chronic diseases—diabetes, heart disease, Alzheimer's, etc.—are rare or largely absent in the Indigenous populations," even when their inflammation profiles superficially resemble those of older adults in industrialized societies.

A separate study published October 29, 2025, in *Nature*[156] backed these findings. "We were surprised that inflammation is not driving healthy aging. We think inflammation is driven by something independent from just the age of a person," said Claire Gustafson, Ph.D., assistant investigator at the Allen Institute and one of the lead authors of the study, in a statement.[157] "This is important because there's been research showing similar findings that inflammation and aging don't go hand in hand, and your immune system is just changing with age."

Now consider the fact that super agers living in industrialized countries—countries bursting with an array of potential environmental toxins and ultra-processed foods—rarely show age-related inflammation either. A 2015 study[158] found that when it came to aging in centenarians and supercentenarians, the "suppression of chronic inflammation [was] a major determinant of

successful longevity, which [was] relevant over a very wide age range up to extreme old age." A 2024 study out of Sicily[159] found the same thing. "Controlling [inflammatory] responses plays a significant role in achieving extreme longevity."

Let's take it a step further. If super agers are Borrelia free and if Indigenous peoples in isolated communities are Borrelia free (with inflammation primarily being driven by parasitic infections), then that would mean that most chronic illnesses in industrialized societies today are being caused by the bacterial infection. So let's seek out the anomalies.

Japan presents perhaps the most studied—and most misunderstood—longevity puzzle in the world. The numbers are staggering. Japan has the world's longest life expectancy at approximately 85 years and is home to some 100,000 centenarians,[160] by far the highest of any nation. Eighty-eight percent are women. Japan also has the lowest obesity rate in the developed world[161]—approximately 4% by international standards, compared to over 40% in the United States—and the lowest rates of heart disease and metabolic disease in the developed world.

Researchers have reflexively attributed all of this to diet. But the diet explanation strains under scrutiny. Japan also happens to be one of the most insular, homogeneous societies in human history. For centuries, the country was willfully closed to foreign influence.[162] Immigration remains stubbornly restricted and over half of Japanese citizens still oppose accepting more foreigners. Intermarriage with outsiders has historically been rare and socially discouraged. Japanese attach more importance to ancestry and ethnic identity than almost any other nation studied.[163] In the framework of Borrelia's sexual transmission, Japan's insularity is not a cultural curiosity. It has been biological firewall.

The emigrant data is telling. The landmark NI-HON-SAN study [164] tracked Japanese men in Japan, Hawaii, and California and found a precise disease gradient—lowest in Japan, intermediate in Hawaii, highest in California. The researchers themselves concluded this gradient "appears not to be completely explained by differences in dietary intake, serum cholesterol, blood pressure or smoking." Additionally, a 50-year follow-up study found no significant difference in total caloric intake [165] between Japanese Americans in Hawaii and Los Angeles and native Japanese in Hiroshima; same diet, dramatically different disease rates.

But when the fire burns too hot, even the best firewalls eventually fail.

Underneath the stunning statistics of centenarians, many disease free, lurks another reality. Borrelia is insidious, and Japan's insularity could only hold off the bacteria for so long. In the 1970s, rates of dementia were astonishingly low. Today, they are soaring,[166] with familiar comorbidities revealing the disease's presence: diabetes and high glucose, coronary heart disease, stroke, and osteoporosis.[167] Women are disproportionately affected.[168]

Lyme disease has fully infiltrated Japan. We know how this story ends.

The Arctic and the Inuit provide the most illustrative example of the disease's destructive power—extremely isolated, exceedingly insular, and free of ticks that cause Lyme, but once opened to the

broader world, gradually swallowed by modern society and its chronic diseases. Their existence has been known for centuries, but they've only been studied since the late 1800s. According to modern nutritional beliefs about high-fat diets and their connection to chronic diseases, at the time the entire region should have been full of unhealthy, overweight people with considerable health issues. The opposite was true. A 2008 study in *The Lancet*[169] tried to make sense of the unique health trajectory of this population. "Although malignant diseases were believed to be almost non-existent in Inuit populations during the beginning of the 20th century, the increasing life expectancy within these populations showed a distinct pattern, characterised by a high risk of Epstein-Barr virus-associated carcinomas of the nasopharynx and salivary glands, and a low risk of tumours common in white populations, including cancer of the prostate, testis, and haemopoietic system."

This research is deeply revealing. Since EBV was already circulating widely in the Indigenous community—and autoimmune diseases, chronic illnesses, and most cancers were strikingly absent—it's clear EBV isn't the feared bogeyman[170] many have claimed it is. (It's also not behind lupus, the latest disease researchers have erroneously tied to the virus.[171]) We now have supporting evidence for why EBV-negative children still get diseases[172] thought to be triggered by EBV, why EBV hasn't elucidated the gender disparity puzzle, and why all promising EBV trials keep failing (antivirals like acyclovir and valacyclovir have flopped and atacicept unexpectedly made disease worse). It's not EBV, and plowing money into EBV vaccines—enthusiastically promoted by the former FDA commissioner[170]—won't change the outcome. I kept digging.

"In Canadian [Inuit] no case of diabetes mellitus has yet been reported in the traditional-living central and eastern Arctic regions," reported a 1981 book.[173] Colorectal cancer was a rarity and "in earlier periods breast cancer was extremely rare, reported almost as non-existent, among both Canadian, Alaskan and Greenland [Inuit]." Ditto cancer of the uterus. Before 1967, lung cancer in Canadian Inuit was only found in women, as they "tended day and night lamps which emitted much heavy sooty smoke from the seal and fish oils."

The protective halo shielding the Inuit, however, didn't last. A health utopia—with virtually zero cases of diabetes, colon cancer, breast cancer, and unexplained lung cancer—does a 180. Despite scant evidence of cause and effect and the noticeable absence of Chick-fil-As on the tundra, the fingers of researchers, reflexively, all pointed to fast food, Lucky Strikes, and estrogen thrust upon the Indigenous group by the seepage of Western society. Because what else could it possibly be?

I asked that rhetorical question for a reason. To understand why, it helps to jump back in time some 175 years and revisit Ignaz Semmelweis and his ultimately failed quest to get his contemporaries to recognize the critical importance of handwashing.

History, fatefully, is repeating.

Butwhataboutisms

In the mid-1800s, there was a cavalcade of medical hypotheses trying to explain the dreaded childbed fever, a mysterious fatal condition killing an inordinate number of women after

childbirth. It had to be toxic air, scientists reasoned, citing miasma. Or the imbalance of the four bodily humors. Or the spread of putrid material. Or inherent patient weakness. Or inflammation of the uterus. Or trauma during childbirth. Or imbalances in the blood. Or insufficient uterine contractions. Or spontaneous generation. Or mysterious atmospheric conditions. Or overcrowding. Or weather patterns. Or the mother's psychological state. Or improper birthing positions. Or the shame of being unwed. Or inferior moral character. Or divine punishment. Or embarrassment of being examined by male doctors. Or, obviously, an unknowable combination of all the above.

There were some 30 theories[174] bandied about in total, with Dr. Semmelweis's reportedly coming in 28th, meaning that it could plausibly be a reason, but it was exceedingly unlikely given the preponderance of other, far more accepted and promising theories. Experts studying the outright rejection of the hand-washing theory discovered that even though the evidence was substantial, it was too simple to be true.[175] "The main reason for the resistance among members of the medical community was the paradigm shift that required one to accept that disease, such as childbed fever, could have just one necessary cause."

In the early 1980s, it happened again. When Dr. Marshall and his colleague Dr. Warren discovered that Helicobacter pylori was the source of ulcers and a significant risk factor for stomach cancers, the pushback was swift and overwhelming. Not a chance, fellow researchers insisted. The very idea that bacteria could survive in stomach acid was preposterous. It must be emotional stress. Or spicy food. Or excess stomach acid. Or Type A personalities. Or coffee. Or irregular eating patterns. Or genetic predisposition. Or poor sleep habits. Or alcohol. Or smoking. Or, again, a cryptic amalgamation of all the above. The pair's bacteria theory was ridiculed, deemed oversimplified, and ignored. Dr. Marshall then famously infected himself, curing the infection with antibiotics. Despite the clear-cut proof, scientists continued to dismiss the theory for more than a decade. Grudgingly, they came around, and in 2005 the determined renegades received the Nobel Prize.[176]

Skip ahead to 2026 and the landscape of infinite but-what-about medical theories has barely changed. What's behind the recent surge of sickness, from autoimmune diseases and metabolic disorders to cancers and mental illness? It must be the disruption of the gut microbiome—scientists are literally traveling to remote Indigenous communities in Paraguay[177] to collect fecal samples in search of the answer. Or ultra-processed foods. Or contaminated water. Or forever chemicals. Or GMOs. Or microplastics. Or toxic pesticides. Or vaccines. Or unidentified environmental poisons. Or artificial dyes. Or too little vitamin D. Or social isolation. Or the reactivation of the Epstein Barr virus. Or host-microbe interactions. Or physical inactivity. Or barriers to care. Or heightened awareness. Or mismatched genes. Or artificial lighting. Or negative emotions. Or 5G cell phone towers and electromagnetic radiation. Or chemtrails. Or fluoride toxicity. Or dental fillings. Or synthetic nanotechnology. Or, just maybe, it's psychosomatic, all in our heads.

Then there is the Parasitic Infection Theory (sometimes referred to as the Persistent Pathogen Theory or the Stealth Infection Hypothesis), suggesting that most chronic conditions are being

caused by a hidden, undiagnosed infection. That outrageous theory checks in at the outer reaches of sanity, somewhere between laughably implausible and certifiably deranged. Researchers have never seriously considered such a theory because the potential whataboutisms surrounding sickness are endless enough. There are about 100 trillion microbes in the gut microbiome alone,[178] ergo trillions of possible suspects in just the stomach. It would take an enormous amount of research and an endless parade of clinical trials to even begin to narrow down all the possibilities, much less disprove any of them. But a solitary rogue infection?

No, it couldn't be that simple, scientists have insisted. It must be a complicated confluence of factors.[179]

Boxing Out

Discounting an idea—handwashing, a lone stomach bacterium, a spirochetal infection passed down for ages—because it's too simple or too sweeping isn't remotely scientific. But it sure is popular. Today's suspicions mirror, *identically*, the attention paid to yesteryear's suspicions. They are all monumentally mysterious, near impossible to test thoroughly, and ubiquitous and inescapable. Theories from the past, like the misguided belief of miasma, share another cancerous commonality: they are all but impossible to disprove, ensuring their staying power, a virtual cudgel to any contesters daring to question them. "But what about…"

Even though evidence continued to mount supporting my Lyme hypothesis, it would be shortsighted and hypocritical to then outright dismiss mainstream (or contrarian) opinions. People have become invested in these theories for a reason; they all are, to a degree, plausible. So, I turned to the words of Sir Isaac Newton for guidance, that "truth is ever to be found in simplicity, and not in the multiplicity and confusion of things." To simplify the problem, I developed a "box out" method[180] to cut down on the cacophony of 1,000 alternatives, a way to narrow the pool of possibilities. Here's how it works.

Take rheumatoid arthritis, for example. Like so many other health conditions, prevalence and incidence of the disease has risen steadily globally over the last 30 years.[181] This has led to the broad and popular conclusion that the cause for such an increase must be due to something introduced recently, most likely unhealthy changes in diet (e.g., artificial or heavily processed foods) or environmental toxins from modern chemicals.

Eminently logical reasoning but fatally flawed. It skips a critical step. There are two unknown variables in this RA problem: 1) what is the cause and 2) why cases are rising. Two unknowns in a single equation make it unsolvable; there are infinite solutions. Without knowing the origin of RA, the reasons for its recent rise become nothing more than a maddening guessing game.

In contrast, a systems thinker like Newton, using big-picture reasoning, might instead ask, *What is the root cause of RA*? Even if you are wholly unsure what the answer might be, you can cull suspects. Since evidence of RA dates back centuries, all modern-day poisons can be eliminated—diet and pollutants may theoretically be contributors to the rise, but they are not the drivers—ergo, the cause must date back hundreds of years or more. As compelling as it sounds, the trillions of

potential microbiome disruptors don't fall into this category. Nor do pesticides, vaccines, forever chemicals, microplastics or any other 21st century problem.

Once you remove this noise, which you always can revisit later if evidence nudges you in that direction, you can more closely investigate a more manageable, finite group of theories. Only when you have a firmer grasp of what might be the source of RA—by going backward and connecting those dots—can you then extrapolate what may be spurring the rash of recent cases. Using the box-out technique allowed me to home in on Lyme as the most likely genesis of RA. I then was able to apply that hypothesis and overlay all that I already discovered about Lyme to see if my theory *in its entirety* still held. It did, including how it has been spreading rapidly through recent generations, which ultimately revealed why rates have been rising.

This is an oversimplified version of how a systems thinker approaches a complex challenge. Unlike evidence-based medicine, which provides affirmation as scientists incrementally check off the requisite boxes of a problem piece-by-piece, systems thinking takes on the entire problem by looking for patterns and forcing connections across myriad planes. Experiments and their failures, so many failures, are commonplace. The process, which requires an enormous leap of faith, begins as a chaotic, jumbled, fuzzy mix of seemingly unrelated findings before the pieces come together (ideally, if a theory is correct), eventually coalescing into a remarkably sharp picture. Until it does, to most it will look like a hopeless mess, but each of these disparate puzzle pieces are required as they build off one another. Isolated, they appear as blobs of random paint, snippets of theories supported by the weakest of associations, but together they form a masterpiece. For an example, inspect any Jackson Pollock canvas. There's a reason the conclusions of systems thinkers, which Mr. Pollock almost certainly was, are often viewed as crazy and crazier until they, almost magically, turn genius.

That explains why my audacious theories faced—and continue to face—an astronomical level of doubt and a barrage of whataboutisms (*did you consider* X, Y, *and* Z?). Which is healthy, because I outright invited it, and I continue to. Why court such dissent? When Dr. Marshall lectures young, aspiring Nobel laureates, he instructs them to try to dismantle their own precious theories to test them before sharing with peers.[182] Following his advice meant that everything I was theorizing had to survive the tightest of scrutiny.

The burden of proof necessary for my hypotheses to cut through the noise of breathless but flawed medical conclusions thus far has been Herculean. So many pieces of this epic puzzle had to come together just so to avoid an equally epic collapse. I had dumped such a broad array of my grand theories into a single box, that I'd boxed myself into the tightest of corners. For them to *all* be correct—Lyme disease is at the root of autoimmune disease, mental disorders, long Covid, many cancers, and more—any potentially successful treatment must, therefore, have a notably positive effect on all of them as well. That's insanity! But I welcomed that impossible benchmark of proof.

Before drafting what you are reading, I was unaware systems thinking even existed, yet as I look back on my memoir, evidence of that style of thinking is everywhere. My signature investigative tool, SHARDs[13] encapsulates essential elements of the unique problem-solving technique, from

looking back to connect the dots (the second tenet, History Harbors Hints[183]) to seeking out patterns (the fourth, Reliable Results Repeat). It's uncanny. I've never studied systems thinking or its famous advocates. I only figured out by happenstance that it's what I'd been practicing after watching Ken Burns's Leonardo da Vinci special[184] on PBS. Then I caught Steve Jobs's Stanford commencement speech weeks later because of a news story about its 20th anniversary. Both times I was transfixed. In their words I recognized my own struggle—and my own vindication.

I believed, even when the science blew me far off my intended course. And by the time I concluded *Sit Down Before Reading*, the dots—seamlessly, perfectly, magically—all connected. That includes the insane idea that the same treatments work on an enormous swath of health conditions.

—Chapter 6—

That's Insanity

Antibiotics are the first line treatment for Lyme disease. The tetracycline class is used most frequently—doxycycline and minocycline—along with IV ceftriaxone for severe cases. Other classes of antibiotics are used as well, including macrolides like azithromycin, sulfas like Bactrim, and penicillins like amoxicillin. I could festoon the coming pages with studies of their effectiveness in the menagerie of illnesses I've connected to Lyme, only that would hold little sway among today's physicians. When it comes to these particular antibiotics, doctors have been taught, dating back to med school, that any unanticipated therapeutic benefit should be chalked up to their general, wide-ranging "anti-inflammatory properties." Of course.

Infections, and not aging, drive inflammation. Lyme is an infection. Specific antibiotics treat Lyme, lowering inflammation from the disease. But since Lyme, unknowingly, is at the root of so many health conditions—from skin issues to osteoarthritis to cystic fibrosis—the assumption of their effectiveness falls to "unique anti-inflammatory properties" rather than their intended purpose as an infection-fighting treatment. Scientists have been trying to connect the dots, but it hasn't gone well. A limited number of studies are ongoing as to why these antibiotics oddly appear to be aiding neurological conditions, cancer, and cardiovascular disease, but such research is limited. It's even more restrictive in mental health illnesses due to the heightened concerns over antimicrobial resistance and reports of worsening psychological issues. Of course.

Recall that when antibiotics are introduced to Lyme patients, they can trigger a Herxheimer reaction, the temporary worsening of symptoms. Now imagine what happens when antibiotics that treat Lyme are tested in clinical trials. Patients who react after antibiotics are introduced get dropped—"do no harm"—and trials, if they don't get outright halted, become riddled with unwarranted uncertainty, leading to a foregone conclusion. "More research is needed." This issue is especially acute in mental illnesses, as I personally discovered.

There's a reason my medical chart had flagged Bactrim as problematic. I wasn't allergic to the medication, I was Herxing. Thankfully my primary care provider ignored those concerns after I approached him for help during my late summer bout with psychosis in 2021.[185] Indeed, a week of doxycycline pulled me out of my spiraling psychosis so dramatically that, at the risk of my personal safety, I boldly shook my wife Laura awake out of a deep slumber to tell her. My PCP then consulted with world-renowned Columbia University Lyme expert Dr. Brian Fallon, who encouraged my doctor to continue treating me with antibiotics, bucking standard protocol. It saved my sanity, and probably my life. More importantly, regaining control of my mind allowed me to later make the unbelievable medical discoveries you've been reading about here. Just one wee, minor issue.

Antibiotics are in no way a failsafe Lyme panacea.

A curious pattern has emerged among those diagnosed with the bacterial infection. Of those who initially appeared to have successfully beat back a bout of Lyme, almost certainly with antibiotics, many have fallen ill to other conditions I've directly linked to Lyme disease, as have their children and partners. You don't have to look hard for evidence, as old cases of Lyme litter the backstories of many celebrities. This pattern of sickness within families, famous or not, repeats and repeats. None of this is surprising.

A 2012 primate study[186] found that Borrelia burgdorferi persists in the majority of infected rhesus macaques even after the same antibiotic regimen used in human trials; the spirochetes survive, they just go quiet. What is surprising is when it happens in reverse, when someone gets diagnosed with a chronic health issue only to later discover it was Lyme disease all along. Late country singing legend Kris Kristofferson was misdiagnosed with Alzheimer's[187] in 2013. It took three years before doctors realized it was Lyme instead, an amazingly lucky turn. Even so, a monumental problem awaited.

Although doxycycline and related antibiotics appear to have a profound effect on the disease when given shortly after the infection first blooms, their effectiveness plummets when trying to eradicate entrenched, active spirochetes. Often patients experience startling initial gains, only to see the benefits slowly fade over time; it happened to me. For this reason, antibiotics are infrequently used to treat chronic Lyme or its clinical alias, "Post-Treatment Lyme Disease Syndrome" or PTLDS. What to do?

At the time, I had no idea. Nearly two years of antibiotics had kept me remarkably stable—my psychosis would start to return if I paused for a stretch—but to what end? By Chapter 51, I was resolved to let scientists do what scientists do, hopefully taking advantage of everything that I had already discovered to start on the path of developing better treatments. But when I realized my brain was still firing on cylinders I didn't know existed, still solving problems it had no business solving, I figured I'd keep writing.

One last chapter.[188] One final crack at the puzzle. Just to see.

One Final Crack

I knew I wasn't going to create a novel drug out of the ether to squelch Lyme. Not because I lacked the aptitude or the $1.3 billion it typically costs to develop such a drug, but because I was pretty confident that I didn't have to. If Lyme disease has been flattening humanity for as long as humanity has existed, odds were exceedingly high that effective treatments had already been discovered. Like most revolutionary discoveries, though, they largely would have been overlooked, deemed a fluke, or rejected. But with thousands of diseases to sift through along with an ungodly number of potential therapies, the biggest challenge initially was just narrowing the search radius. I had an idea.

As a leading advocate for MS, I started by investigating extraordinary claims in the MS world. One came right to mind: the astonishing reversal of disease by Dr. Terry Wahls, who went from wheelchair to walking in less than a year. Dr. Wahls leans decidedly more unconventional than

other MS experts, with a heavy focus on diet, electrical stimulation, and other alternative therapies and supplements. My confirmation bias long ago had deemed her Wahls Protocol, a mishmash of "Paleo principles" that treat "all chronic autoimmune conditions," a nonstarter. Now, though, her bold claims seemed tame compared to mine. Maybe it was time to revisit her protocol?

For months I toyed with non-diet combinations—one of the meds she took was minocycline,[189] an antibiotic often used to treat Lyme, a fairly important detail not widely publicized—only to come to a sickening realization after all my trials summarily fizzled: a successful treatment had to involve diet. Maybe all those breathless anecdotes I'd scrolled past on the internet claiming such-and-such diet cured their disease weren't hokum after all. But I still didn't buy it. Not yet. So I turned to one of my trusted MS experts, Dr. Brandon Beaber, for his various takes on nutrition and diet. One newer video of his, that I had originally ignored, stood out: Does Ozempic prevent MS?[190]

A 2024 study found that the new GLP-1 weight-loss/diabetes drugs—Ozempic, Mounjaro, Trulicity, etc.—lowered the risk of getting diagnosed with multiple sclerosis. By *a lot*, up to 85%. Dr. Beaber was unmoved for reasonable neurologist reasons, one being that it appeared to be "too good to be true," but these drugs fit the kind of treatments I was looking for in an autoimmune disease—obscene success coupled with dubiousness and disbelief. It didn't take long to confirm that I had found a smoking gun.

Reports of GLP-1's success in tamping down inflammation[191] and relieving pain in autoimmune disease were everywhere. Symptoms were getting suppressed in every health condition I had flagged as Lyme driven, from metabolic disorders to mental illnesses to addictions.[192] The drugs were associated with an 18% lower risk of major depressive disorder and a striking 39% lower risk of bipolar disorder.[193] Psoriasis plaques were melting away within two days of patients starting a GLP-1[194]—far too fast to be a weight-loss effect, leading researchers to conclude the drugs were acting directly on immune cells in the skin. A sweeping review released in January of 2025 found that these drugs were having a profound impact on an eye-popping 175 health conditions[195] and counting, lowering the risk of a host of issues including cardiovascular, kidney, and liver diseases, even many cancers. The drugs are even slowing biological aging itself; a randomized trial found semaglutide reduced epigenetic aging markers across blood, brain, heart, kidneys, liver, and metabolism simultaneously.[196]

Doctors, understandably, have been ecstatic. Scientists, understandably, have been baffled.[197] A former VP at Eli Lilly told the *NYT* in April of 2026, "that many diseases may share the same root causes." Even more revealing, researchers have found that the gut cells producing GLP-1 appear to detect pathogens directly,[198] releasing the hormone as part of the body's response to infection—suggesting these drugs may be amplifying a defense the body was already attempting to mount.

So now we have breathtaking results from GLP-1s combined with staggering anecdotes from people who have embarked on restrictive diets to treat their chronic conditions. The secret sauce must not be exclusive to GLP-1 drugs broadly, they must all be working on a common goal, neutralizing an invasive, intractable bacterial infection. But how? What was going on?

Instead of zeroing in on the unique weight-loss medications, I pivoted to the anecdotes. Answers, as I've discovered, await in the anomalies.

Food as Medicine

For most of my life, I have been a diet scoffer. I cannot begin to tell you how often I have been encouraged, with the flimsiest of evidence, to embark on a specialized diet to regain my health and vanquish my disease. Now I was doing exactly what I'd always scoffed at, investigating these diets, looking for patterns to connect them to GLP-1s.

The most successful weight-loss and diabetic drugs have a commonality: they all lower blood sugar. They generally achieve this by targeting literal sugars and future sugars, or carbohydrates, which the body converts to glucose. What diets do the same? Most of them, I discovered. Gluten-free, paleo, low-fat, the Zone, vegan, DASH, Ornish, all limit, to an extent, carbs and sugars. But there are certain diets, like Atkins, where carb reduction is the focus with few restrictions on fats and proteins. The most popular of these currently, and conveniently one of the most studied, is the ketogenic diet: a low-carb, high-fat, often protein-rich diet.

The ketogenic diet was developed more than 100 years ago to treat epilepsy,[199] but as a diet doubter, I had never given it much thought. The reason keto diets originally came about? Doctors were trying to mimic the success of fasting—used for centuries out of desperation to tame a litany of diseases, including debilitating seizures—without resorting to outright starvation. If I had only researched epilepsy earlier, I would have swiftly connected the dots of this powerful dietary intervention.

There are types of epilepsy that are thought to be autoimmune,[200] a telltale sign that we merely are looking at a variant of Lyme disease. My initial hunch was right, an effective treatment for Lyme has been around for years and years, since the 1920s. Alas, the diet is hard to follow, so most epileptics today use pharmaceuticals to manage symptoms instead. But regardless of treatment, the bacterial infection, no doubt, is still simmering, still a threat. Now for the critical question: Does the ketogenic diet treat everything GLP-1s and antibiotics appear to treat?

Emphatically, yes, so much so that a stubborn subset of respected doctors are heralding the intervention as a treatment for some of the most challenging and intractable chronic illnesses of our time. Harvard psychiatrist Dr. Christopher Palmer is a leading advocate and has dramatically improved the lives of his patients suffering from schizophrenia, bipolar disorder, severe depression, PTSD, and more using the diet. He's attributed psychological illnesses to metabolic disorders of the brain, as he recounts in his book *Brain Energy*,[201] noting that there are "clear connections between mental illness and disorders linked to metabolism, including diabetes, heart attacks, strokes, pain disorders, obesity, Alzheimer's disease, and epilepsy." Not coincidentally, that list is exactly what I've already connected to Lyme disease.

You don't have to look hard to find evidence of the ketogenic diet's potential, with it appearing to aid (in limited trials or case studies) everything from autoimmune diseases to cancer to long Covid. The evidence reaches even into glioblastoma, the deadliest brain tumor known to medicine, with a

median survival of just 11–14 months on standard treatment. A 2026 clinical trial combining a ketogenic diet with prolonged fasting and time-restricted feeding nearly doubled median survival[202] to 21.5 months—and quadrupled the three-year survival rate from 7% to 27%. Even periodontitis, gum disease long linked to systemic inflammation and autoimmune disease, shows measurable improvement with calorie restriction, with a 2026 clinical trial finding significant reductions in inflammatory markers in patients following a fast-mimicking diet.[203]

Doctors and researchers are so close to connecting all the dots. Dr. Palmer's work, and the videos of a patient advocate[204] diagnosed with schizoaffective disorder (the indomitable Lauren Kennedy West, who put her disease into remission after following his protocol), were invaluable in helping me make these final links. There was one more question, though, that needed answering, a question a young ER doc recently asked me about while he was wondering aloud why I was taking a GLP-1. After all, I wasn't overweight or suffering from diabetes. "What's the mechanism of action?"

Sun Tzu got it right in *The Art of War*. Once you know the enemy—Lyme disease—it all makes perfect sense. The GLP-1s, Dr. Wahls, the curative diets, all of it.

Know Thy Enemy

Researchers are uncertain as to what causes metabolic disorders. There are many theories, but it is believed to involve a complex interaction of a combination of things. Genetic factors. Hormonal imbalances. Nutritional factors. Environmental and lifestyle factors. Autoimmune processes. (Unsurprisingly, it's the same kaleidoscope of theories proposed for most illnesses.) Doctors also don't know why the ketogenic diet has had success in treating these disorders and other conditions like diabetes, besting low-fat diets.[205] There are many theories. Maybe by influencing metabolism, it's tamping down inflammation. Or reducing oxidative stress. Or improving cellular signaling pathways. Or it's something else.

It's something else. Per Occam's razor—properly applied—often the simplest answer is the right one. And it couldn't get much simpler.

Where do you suppose the microscopic corkscrew bacteria behind Lyme disease gets the energy it needs to survive? "It appears that this bacterium is unable to synthesize amino acids, nucleotides, fatty acids, or most other cellular building blocks," says research.[206] "The present metabolic studies determined that B. burgdorferi is capable of utilizing only a small number of different carbohydrates as energy sources." Specifically, sugars, or glucose, derived from carbs. This buffet of carbohydrates circulates in the blood and "B. burgdorferi uses those nutrients to increase metabolism and rapidly replicate."[207]

The latest research supports the connection between carbs and disease. An October 2025 study in the *International Journal of Epidemiology*[208] found that low glycemic index diets, featuring fewer and slow-to-release carbohydrates, were protective against all-cause dementia, while a carb-heavy diet led to a notably higher risk. Another October 2025 study[209] found "significant associations between higher intakes of total carbohydrates, total sugars, fiber, fructose, and glucose and an increased MS risk," leading the authors to recommend the development of "dietary interventions

focused on carbohydrate regulation to mitigate MS risk." But if MS is Lyme disease, what is the issue with relying solely on CDC-recommended antibiotics?

Although susceptible to certain antibiotics, Lyme disease spirochetes are crafty enough to evade and wait out such assaults, blunting their effectiveness. But there is one warfare tactic that it cannot dodge: the siege.

Marauding spirochetes have overrun the traditional defenses of immune systems and established residence in the body, rendering their myriad tactics to repel and evict the raiders moot. They appear to have taken over the castle with no plans to relinquish their conquest, setting up a stout defense. Except in an unexpected twist, these drugs and diets that reduce blood sugar are all effectively laying siege, denying the bacteria critical food rations. The shapeshifting, stealthy bacteria with the indestructibility of the T-1000 Terminator has a weakness.

By forcing the body to burn ketones instead of glucose by restricting carbohydrates, the ketogenic diet is essentially starving out Lyme disease. (Intermittent fasting also pushes the body into ketosis, temporarily accomplishing the same thing.) Similarly, GLP-1s are severely limiting blood glucose with the help of pharmaceuticals, cutting off necessary fuel. Even exercise, long touted by doctors as an effective intervention to most health conditions, accomplishes much of the same. A single exercise session can lower blood glucose for 24 hours or more,[210] while regular exercise consistently drives down A1C numbers.

The most dramatic proof of this requires no clinical trial. It happens naturally, at the bedside, in the final days of life. As appetite fades in dying patients, blood sugar drops. And in a documented subset of those with late-stage dementia, something extraordinary occurs: the person comes back. They recognize faces. They speak in full sentences. They crack jokes. They say goodbye. NIH-funded researchers, now capturing these episodes on video for the first time, call it paradoxical lucidity, and have spent years confounded by how a brain ravaged beyond repair could suddenly, briefly, function again.[211] One researcher compared it to a pot-hole-filled highway on the verge of collapse abruptly turning smooth, a theoretical impossibility. Except it isn't. It isn't a paradox or divine intervention. It's what happens when Borrelia is starved of its fuel. The spirochetes recede. The person resurfaces. Then the window closes.

That's why GLP-1s have been so successful at preventing and treating so many conditions, including cancer—and the evidence keeps mounting. At the 2026 American Society of Clinical Oncology meeting, a cluster of five real-world studies reshaped how medicine thinks about these drugs. A University of Pennsylvania analysis of 111,646 women[212] found GLP-1 users were 30-47% less likely to develop breast cancer, and the effect persisted after accounting for weight loss. A Cleveland Clinic study[213] found GLP-1s reduced metastatic progression across four solid tumor types—lung, breast, colorectal, and liver cancer—by 38 to 50%. A Roswell Park analysis of nearly 150,000 women[214] showed improved overall survival in breast cancer patients taking GLP-1s.

It's no surprise that researchers keep homing in on weight loss as the reason for the striking success of these medications, with them delivering an astonishing 41% reduction in cancers linked to

obesity.[215] GLP-1s reduce the risk of endometrial cancer—the fastest rising cancer in young women—by an eye-popping 58%. Risks have plunged by 50% or more for other historically deadly cancers: multiple myeloma, colorectal cancer, and pancreatic cancer. In acute myeloid leukemia, it was a bonkers 63%.[216] But researchers have noted something that keeps appearing across every GLP-1 study: the benefits are "independent of weight loss." Medicine keeps finding this and keeps being bewildered by it. The Borrelia framework is not bewildered. Starving the bacteria of glucose starves the tumor of its inflammatory driver. The weight loss is incidental. The bacterial starvation is the mechanism.

The cardiovascular findings are equally staggering—and equally unexplained by conventional medicine—dropping heart attack risk in some patients by as much as 54%.[217] A meta-analysis of 16 trials involving 23,467 participants[218] found GLP-1s reduced major cardiovascular events by 20%, with the strongest effects on stroke (28% reduction), heart attack (16% reduction), and heart failure hospitalization (18% reduction). Adding GLP-1s on top of existing cardiovascular medications produces a further 10-20% reduction[219] in heart attacks, strokes, and deaths on top of already excellent cardioprotective care. A Mass General Brigham study of nearly one million adults[220] found both major GLP-1 drugs reduced stroke and heart attack risk by 13-18%, with benefits appearing early, "suggesting that their protective mechanisms go beyond weight loss alone." A large real-world study of nearly 50,000 patients confirmed as much[221]: semaglutide's cardiovascular benefits tracked with the dose patients received, not the weight they lost; the drug is acting directly on the heart, independent of any slimming effect. Blood pressure, the single most common precursor to cardiovascular events, drops consistently on GLP-1s.[222] Recall that a landmark 2025 study of more than 9 million adults[223] found that 99% of all heart attacks and strokes were preceded by just four risk factors: high blood pressure, high cholesterol, high blood sugar, and smoking. GLP-1s directly address three of the four. Not because they treat cardiovascular disease. Because they starve the bacterial infection driving the inflammation that causes all three.

This also explains one of the most confounding athletic stories in recent memory. Serena Williams[224]—arguably the greatest athlete of her generation—could not lose weight after her pregnancies despite doing everything right. "No matter what I did—running, walking, I would walk for hours because they say that's good, I literally was playing a professional sport—and I could never go back to where I needed to be," she told Today[225] in August 2025. Joint pain and blood sugar issues compounded the problem. After starting a GLP-1, she lost 31 pounds and her lifetime risk of heart disease fell 70%; her health markers improved beyond what they were during her prime. "I wish they had this when I was on tour. I would've been really amazing."[226] So it was unsurprising to see Williams return to professional tennis[227] to play singles at Wimbledon as a wild-card entry. At age 44.

For women who are struggling with the same issue and resisting GLP-1s, Williams has long maintained that these drugs are not a shortcut.[228] Instead, they a potential life-changing intervention against a bacterial infection that has been silently undermining health for years.

The evidence increasingly backs this up. A large-scale study of nearly 18,000 people with hip or knee arthritis[229] found that high-impact exercisers—runners, tennis players, skiers—were about half as likely to need a hip replacement as those who avoided such activity. If mechanical pounding destroyed joints, the opposite would be true. The damage isn't coming from the outside. It's coming from within. A decade-long Finnish trial published in the NEJM confirmed as much[230]: patients who underwent arthroscopic surgery to trim degenerative knee cartilage—one of the most common orthopedic procedures in the world—fared *worse* than those who received a sham incision, with more pain, accelerated osteoarthritis, and higher rates of knee replacement. Medicine has been operating on the symptom while the cause goes untreated.

I've tried to sound the alarm. In April 2024, I wrote to Dr. Gary Dorshimer, a veteran Philadelphia sports medicine physician and longtime team doctor for the Eagles and Flyers, warning that Joel Embiid's Bell's palsy—facial paralysis that appeared days before the playoffs—was almost certainly neuroborreliosis: Lyme disease attacking the nervous system. Dr. Dorshimer graciously wrote back and said he passed the letter along. Two years later, Embiid has missed 491 games in 12 NBA seasons[213] —more games missed than played—with a cascade of conditions the Borrelia framework predicts precisely: facial palsy, persistent migraines, chronic knee inflammation, and most recently, appendicitis requiring emergency surgery days before the 2026 playoffs. My letter went unheeded. The cost of inaction is measured in careers, in seasons, in lives. The promise of action is measured in generations.

The true power of GLP-1s will be so much greater if the drugs are found to be safe in pregnant women and children, and if they are, look for the risk of birth defects and childhood diseases to plummet. The implications of that outcome are almost too large to state. But here is the rub.

The Rub

These drugs prevent Lyme from gaining purchase, holding the disease in check, but they don't necessarily eradicate or reverse it. They severely restrict its fuel, keeping the bacterial disease in a kind of stasis, unable to do much additional damage. If the medication is stopped and no other interventions are pursued, spirochetes will resume feasting on carbohydrates. Without a pharma governor, any weight and health issues spurred by Lyme will likely return, meaning GLP-1s need to be taken continuously. (A study published in *JAMA Internal Medicine*[231] in late November 2025 confirms this.) Also problematic: they are obscenely expensive, health insurers limit their use, and, as with most medications, there can be uncomfortable side effects.

Meanwhile, the ketogenic diet features its own unique set of challenges. No matter how potent a ketogenic or other carbohydrate-restrictive diet may be when done properly (an extremely tall order as I've discovered), 8 billion people are not going to embark on an extended carbohydrate-restrictive regimen to starve out the infection, never mind the devastation such a societal shift would do to the food, beverage, and restaurant industries. It is a notoriously difficult diet to sustain, and it must be followed religiously if the goal is to starve the disease into submission (no cheat days). Successful dieting often requires dedicated coaching, close monitoring, pricier groceries, and the commitment of an entire household, all nonstarters for many.

Virtually all health interventions recommended by doctors today are addressing, unknowingly, Lyme disease. Dermatologists prescribe more doxycycline and steroids than any other medication. Healthy diets and regular exercise combat blood sugar.[232] Social engagement and getting better sleep both reduce stress, a known Lyme trigger. Vaccines and avoiding head injuries are minimizing the risk of major immune system disturbances. Most medications target Lyme symptoms, from trying to decrease inflammation to helping to manage fatigue. Then there are the weight loss and diabetes meds, including metformin, an inexpensive diabetes drug that has been touted as a "wonder drug" for its positive effects on a range of health conditions.[233] They are all just tamping down the disease's fuel source.

Metformin deserves a closer look. Researchers have noted its remarkable effects across dozens of conditions far beyond diabetes[234]—cancer, aging, neurodegeneration, autoimmune disease—and have repeatedly tried to confirm those effects in clinical trials, repeatedly coming up short. A 2020 meta-analysis of nine randomized controlled trials[235] adding metformin to cancer therapy found it failed to improve outcomes, then flagged the most likely culprit: the dose used in the trials may have been insufficient, with drug concentrations at standard diabetic doses potentially too low to produce the direct cellular effects observed in preclinical studies. A fibromyalgia trial currently underway[236] is testing explicitly low-dose metformin as a neuroinflammatory intervention.

In each case, researchers are running the right experiment with the wrong hypothesis, at the wrong time, measuring the wrong outcomes, at doses that may never have been sufficient to test the actual mechanism. GLP-1s work best at preventing cancer, autoimmune diseases, and other health conditions from forming in the first place, and the same logic applies here. If metformin works by restricting glucose availability—starving Borrelia of its primary fuel source—then these trial designs all have fatal flaws and were doomed before they began. No wonder their lackluster results. Provide the drug as a preventative, and when treating disease, push to maximum tolerated dose. The results will speak for themselves and confirm what the observational data has been quietly suggesting for decades.

There are more mammoth complications. Because Borrelia appears to spread sexually and possibly through other forms of close contact, in addition to in utero, to curtail its spread, all parties ultimately must be treated, or it will just be passed between romantic partners. Worse, there's evidence that blood transfusions and organ donations may be problematic,[237] as research has shown a 90% increased risk of autoimmune disease after a single transfusion as well as a two to fourfold elevated risk of cancer after an organ transplant.[238] This could simply be Lyme reawakening due to an immune system disturbance caused by the patient's existing health condition, but if someone requires a life-saving infusion or transplant, with most donors unknowingly infected, Borrelia tagging along wouldn't be surprising. (This does call into question the growing trend of infusing yourself with young blood to rejuvenate older tissues. Younger patients may have multiple strains of Borrelia. Maybe don't do that.)

Eradicating Lyme, now entrenched in society, will require patience, unwavering persistence, and scientific breakthroughs in the form of vaccines and novel interventions.

For scientists reading this still hoping upon hope that it's not chiefly Lyme—that there is another explanation for all that I've uncovered—there is a final arbiter, a final test, to validate prospective alternative theories. Can your hypothesis plausibly explain the gender differences in autoimmune diseases, why women are far more likely to be afflicted than men? And why women are more likely to get Alzheimer's, suffer from PTSD, and be diagnosed with chronic Lyme?

Earlier I challenged courageous scientists to try to crack this conundrum for themselves. As attentive readers may have noticed, I've been leaving a trail of breadcrumbs throughout this missive—leads and hints for researchers to follow up on and investigate. Why? As a critical thinking exercise. I'm not asking you to believe in me and my discoveries, I'm asking you to believe in the science behind them.

There's a reason I've called the answer to this puzzle the holy grail of medicine. It does far, far more than settle those questions. With startling efficiency, it vacuums up medical mysteries that have stumped healers for millennia.

—Chapter 7—

Extraordinary Evidence

One of the most challenging things to do in all of science is to see the unseeable. Modern medicine relies on an incredible array of technology to see the tiniest of organisms, peer into the deepest recesses of the brain, and scan the most minute parts of the human body. But when something truly is unseeable, when it cannot be directly observed, another tool must be deployed: scientific inference, the ability to deduce what must be true by indirect evidence. For example, we knew black holes existed more than 200 years before we observed one for the first time in 2019. Researchers conclusively solved the puzzle of continental drift not by watching continents move, but by analyzing earthquake patterns, fossil distributions, and magnetic striping on ocean floors. And planets outside our solar system, even entire planetary systems, can be inferred merely by the wobble of a star due to gravitational tugs.

Semmelweis and his handwashing theory? Ichelson and her discovery of spirochetes in MS patients? Alfred Wegener and his continental drift theory? In these cases, researchers couldn't reliably observe the offending bacteria, much less continents moving, leading them to ridicule and dismiss their theories out of hand. After all, there were plenty of other far-more-plausible theories to give weight to before these stretches of the imagination. At the time, these discoveries couldn't quite meet the Sagan standard: Extraordinary claims require extraordinary evidence.

Popularized by Carl Sagan, the astronomer and planetary scientist, Extraordinary Claims Require Extraordinary Evidence (ECREE) is a principle holding that bold claims demand proportionally robust evidence. It's a bar rarely met, even by the masters. But if Darwin, who studied theology in college, could do it with limited formal scientific training—his theory of evolution relied primarily on scientific inference and observation—so could I, even without access to a dedicated research lab, much less a basic microscope. That meant finding, with essentially zero resources, the holy grail of medicine.

So, I did.

Borrelia burgdorferi is one of the only organisms on the planet without a need for iron, indeed it appears to lack iron entirely, and instead relies exclusively on manganese for functions other bacteria typically use iron for. Lyme disease requires unusually high amounts of the trace element for its survival and metabolic functions. These remarkable features are unique to Borrelia spirochetes.

That knowledge is the skeleton key needed to unlock the swirling mysteries that have vexed the world's brightest medical practitioners for generations.

Understanding how the human body collects, absorbs, distributes, and stores manganese was the first step needed to solve this puzzle. Generally, it is thought that people require small amounts of

the element to function, as it is believed to play a crucial role in numerous bodily functions, including metabolism, bone formation, wound healing, and immune function. Manganese for the body comes from food—whole grains, leafy greens, nuts, legumes, and shellfish are common sources—which is then absorbed in the small intestine. Upon absorption, it enters the bloodstream and is distributed throughout the human body, the highest concentrations stored in the bone, liver, kidneys, and pancreas. But no corner is off limits, as the trace element can cross both the blood brain barrier and placental barrier.

If Borrelia is on a mission to acquire manganese, women therefore must have higher levels since they broadly have more disease. But why? That meant I had to learn what influences manganese levels, specifically what causes them to rise and fall. The answer led to that proverbial aha moment, akin to Newton's apple, Fleming's penicillin, or Einstein's relativity. Everyone had been overthinking the problem, trying to tie women's susceptibility to disease to immunological differences, hormonal factors, genetic factors, microbiome differences, even anatomical differences. It is none of those.

Revealingly, levels of manganese and iron are inversely coupled.[239] Iron levels influence manganese levels and vice versa. This interplay is key. Higher iron intake reduces the absorption of manganese, while iron deficiency increases manganese absorption.[240] Women have lower levels of iron and noticeably higher levels of manganese during their reproductive years due to menstruation.[241] Menstrual cycles cause the body to lose blood, and with it, iron. My eureka moment is tied to, of all things, Aunt Flo, a monthly visitor I've never personally met. Girls typically get their first period, or menarche, starting around age 12 or 13, about the 6th grade, but it's not uncommon in 5th graders (hence my earlier clue that a fifth grader could figure out this puzzle). Then, every month for the next 35-40 years, women reliably absorb ample quantities of manganese, the essential mineral Lyme disease requires.

This is the elusive, final puzzle piece, the one that connects every last dot.

Every Last Dot

The sickness that is swamping the world can be traced to Borrelia burgdorferi's ceaseless quest to obtain manganese. It's an elegantly simple solution to one of the world's most confounding problems.

This explains why women are more often afflicted by autoimmune diseases, and why those health issues often surface during the prime of their lives. They have much more manganese stored than men do due to menstruation; it's that straightforward. And because manganese functions as a trace element—operating at concentrations measured in micrograms—even modest differences in absorption produce outsized biological effects. Borrelia needs manganese and consistent access to stored manganese, and menstruating women provide exactly that, reliably, month after month, for decades. It's the same reason cases of long Covid, chronic Lyme, and Alzheimer's are the primary domain of women. And stroke. And type 2 diabetes. And heart disease. And osteoporosis. And

depression. And so many other health issues caused by Lyme, including obesity. The connection never breaks. It does, however, pause.

Researchers have long speculated that a complex interplay of hormones and remarkable female bodily adaptations—carefully honed through evolution—are responsible for diseases uniquely calming down during pregnancy, a benefit that continues through breastfeeding. Not exactly. During pregnancy, the immune system deliberately suppresses itself to protect the fetus from being rejected as foreign tissue. That temporary immunological truce limits Borrelia's ability to trigger the full cascade of immune responses that drives chronic disease symptoms, which is why so many conditions quiet down. Not because manganese disappears, but because the immune alarm system is intentionally muffled.

Disease modifying therapies work for the same reason. They don't treat the underlying infection—they suppress the immune system's ability to mount a full inflammatory response to it. The alarm rings less loudly. Relapses decrease. But Borrelia continues its work unimpeded, which is why disease modifying therapies must be taken indefinitely, why they reduce relapses without stopping progression, and why discontinuing them reliably triggers a return of symptoms. Medicine has spent decades perfecting the art of muffling the alarm. What you are reading explains what has been triggering it.

The manganese picture during pregnancy is actually the opposite of what you might expect. Blood manganese levels rise two to three times above pre-pregnancy levels, as the body mobilizes manganese from internal stores and increases dietary absorption to support fetal development. Far from starving Borrelia of its fuel, pregnancy gives the spirochete greater access to the element. Not coincidentally, cancers of all types are disproportionately diagnosed in women during pregnancy and in the year afterward[242]—breast cancer, melanoma, lymphoma, leukemia—exactly what the manganese model predicts. Colorectal cancer belongs on that list too: pregnancy-associated cases in Australia climbed roughly 6 percent annually from 1994 to 2013,[243] and such cases are notorious for delayed diagnosis, often mistaken for the bowel changes and anemia that pregnancy itself produces. Unsurprisingly, excess manganese also increases the risk of preterm delivery[244] and boosts the odds of postpartum depression.[245]

Other outliers, like childhood autoimmune diseases, typically are explainable after closer examination. Type 1 diabetes, an autoimmune disease, would appear to conflict with the manganese hypothesis, as it is typically diagnosed in childhood, before girls get their first period. It doesn't fit the traditional pattern of autoimmunity—the gender ratio is close to even—but manganese straightens out that apparent contradiction. Boys and girls have similar levels of the trace mineral,[246] which also explains why most birth defects caused by Lyme fall closer to a 50/50 gender distribution.

To help understand why cancers are rising precipitously in young people, look no further than the unique signature of Lyme—manganese is the magic unifier. Without this knowledge, scientists are going to keep fingering the usual suspects.

That explains the panicked study published in November 2025's *JAMA Oncology*,[247] with researchers hypothesizing that ultra-processed food might be contributing to rising early-onset colon cancer cases in women under 50. That same month, *The Lancet* published a breathless trio of papers[248] decrying the rise of ultra-processed foods and their "association" to many diseases—seemingly ignoring the whole correlation isn't causation mantra of science—came to an expected conclusion. "Deteriorating diets are an urgent public health threat that requires coordinated policies and advocacy to regulate and reduce ultra-processed foods and improve access to fresh and minimally processed foods."

Dr. David S. Ludwig, a Harvard specialist in nutrition, must be aghast. A month earlier he had argued in both *The New England Journal of Medicine* and *The Washington Post* that researchers are falling into a familiar trap,[249] relying on the same type of weak observational studies that originally linked high-fat diets to chronic disease. Low-fat diets were no panacea as obesity skyrocketed, and "now ultra-processed food has replaced fat as the new dietary villain" with equally flimsy evidence.

But it was another revelation that left no doubt of Lyme's involvement. "Researchers have observed what's called a 'birth-cohort effect' with colorectal cancers, meaning that successive generations — in this case, beginning with those born in the 1950s — are showing progressively increased risk," said a *New York Times* article reporting on the study.[250] "People born in 1990 are twice as likely as those born in 1950 to develop colon cancer, and four times as likely[251] to develop rectal cancer."

A generational spike of cases beginning in the 1950s screams Lyme. So, of course, it's got to be something introduced into the environment around that timeframe. "Much of the ongoing research is focused on changes to the gut microbiome and inflammation associated with modern products like ultra-processed foods, microplastics and antibiotics, the study said."

When scientists latch onto an idea, they latch on like a pit bull with a case of lockjaw.

Even the nuances of how manganese accumulates in the body are illuminating. Researchers have long observed a flattening of the gender gulf in chronic illness[252] as patients age into their 60s and beyond. This flattening makes sense, as women typically enter menopause in their 50s and stop menstruating, throttling back their regular manganese absorption. This also helps explain why late onset MS patients are more frequently male,[253] yet for some conditions, osteoporosis for example, older women are still far more susceptible. Why? The half-life of manganese may hold clues.[254] While the trace element lingers for only a few hours in the bloodstream, its half-life in bone is 8-9 years, far longer than other storage areas.

But what about schizophrenia and autism? If they are being caused by Lyme, why are these conditions disproportionately affecting males? For this hypothesis to hold up, spirochetes must therefore be after manganese in an area of the brain suspected to be responsible for causing the mental disorders, and male brains must store more of the trace mineral in that exact location. Science supports both fronts. The *globus pallidus* indeed stores the lion's share of manganese in the brain,[255] and its volume is notably larger in males.[256]

It all lines up. And that's a fatal problem for competing hypotheses.

Any suggestion that the source of today's maladies is caused by something else—EBV, gut microbiome disruption, metabolic disorders, a coinfection (e.g., babesia, bartonella), or the theory du jour—must clear at least one hurdle. It must be able to explain an illness's gender disparity. Sure, it would be more reassuring if an alternative theory could also check all the boxes Lyme disease checks, but the critical importance of manganese in Lyme disease effectively boxes out every other competing theory. It clarifies not only the sex ratio, but also the age of onset, any generational differences, the location of myriad health problems, and so much more.

In a modern medicine twist, Lyme disease has evolved from zebra to horse. Doctors have been taught to think of the disease as rare, exotic, and easily dismissed—the diagnosis of last resort for patients with mysterious symptoms who have exhausted every other explanation. "When you hear hoofbeats, think horses, not zebras," the medical aphorism goes. But in the case of Lyme, medicine got it exactly backward. The horse has been standing in the room for decades. Doctors kept diagnosing zebras.

There absolutely are zebras. Not every mysterious illness is Lyme. Not every gender disparity, every disease flare, every unexplained cancer traces back to Borrelia. The manganese connection doesn't eliminate other causes—it just makes them less likely. But when the pattern is consistent, when women are disproportionately affected without explanation, when the same constellation of symptoms keeps clustering across conditions that supposedly have nothing in common, the horse is almost certainly in the room.

Most rare diseases skew female. Doctors should immediately suspect the horse, yet they see only zebras. And those zebras doctors keep diagnosing? Upon closer inspection, many aren't even zebras. They're unicorns—mythical creatures that don't exist. Tragic cases of mistaken identity, dressed up in diagnostic clothing that obscures the beast underneath.

Test it out on your favorite health concerns, even those with seemingly known causes. Just look for the revealing signs of Lyme's involvement, the medical equivalent of a star's wobble.

For instance, it is common knowledge that cigarette smoking causes lung cancer, and that men are most affected. This makes sense. Men are more likely to smoke and, when they do, they smoke more cigarettes daily than women. Lyme is not the driver of these cancers. However, in cases of lung cancer that do not involve smoking, the gender disparity flips: women are twice as likely to be diagnosed as men. Trying to explain this confounding difference between sexes, researchers suspect radon levels,[257] a curious hypothesis. If men and women breathe the same air, pollutants should affect both sexes equally, and cases of lung cancer between genders should be similar, even if a minority of cases are being caused by Lyme. But that's not happening, as demonstrably more women than men are affected.[258] There simply are not enough cases of lung cancer caused by pollution to significantly narrow the gender disparity. Ergo, it's largely Lyme.

How powerful is this holy grail of medicine? It lays waste to other theories with the efficiency of John Wick, exterminating them with simple logic.

For example, men and women contracted Covid at equal rates, yet women are more than twice as likely to develop long Covid.[259] Similarly, men and women received Covid vaccines at similar rates, yet women are far more likely to experience health complications afterward.[260] In both cases, there shouldn't be any difference between the sexes. Lyme disease and its pursuit of manganese is the only way to resolve this gender gap, meaning it's at the root of long Covid and the extensive list of health problems blamed on vaccines. The damning proof is in the response of young children. Rates of long Covid in pre-pubertal children show no significant sex differences[261]—the gap only emerges at puberty, precisely when girls begin menstruating and accumulating manganese.

This also puts to rest the ill-conceived hypothesis that people who don't recover from Lyme have some sort of "post-treatment" syndrome and not an active case. That gender disparity still exists, meaning that spirochetes are still on the hunt for manganese. The disease is very much active. This same logic applies to any of the other "post-infectious" syndromes now commonplace in the medical charts of women.

Manganese. Its role in Lyme disease is the ultimate box-out, the final arbiter, the extraordinary evidence demanded by extraordinary claims.

Telltale Patterns

Identifying patterns is central to systems thinking, a critical tool to understand and deconstruct complex problems. I don't seek out these patterns; they appear to me. Often this happens over time, organically, when enough reinforcing data points synchronize to make me go "huh." It might take weeks, months, even years, as my brain chews on disparate morsels of information, constantly processing in the background of daily life. It's typically not intrusive. But just as often, these epiphanies arrive during that late-night hazy time just before falling asleep. It turns out this is known as the science-tested hypnagogic state,[262] and many scientists, artists, and creatives—Newton, Einstein, Tesla, Beethoven, Edison, and Dalí are just a few examples—have credited hypnagogia for helping to make their discoveries and masterpieces. So that ideas wouldn't get lost to deep sleep, a common trick used by these men was to hold an object that would clang to the floor as they drifted off in order to capture potential eureka moments.

When I suspect a potential connection, I'll do some quick research of published studies, emailing myself on the spot if I find anything relevant. Perhaps I'll jot down some quick notes on my phone app to help jog a memory. I investigate with more rigor only when enough studies support my suspicions. Often those hunches don't immediately pan out—the science doesn't add up the way I expected it to—leaving them to languish in my inbox. That is, until they are urgently needed.

I first registered the manganese connection more than three years ago—I was so uninspired I didn't bother emailing myself—but it made an impression. It wasn't until June of 2024 that I started to put it together. A cluster of emails concerning the trace mineral arrived in my inbox June 13th. In mid-July there was another cluster, and then another in mid-August, before the floodgates burst open in the following weeks with more than two dozen.

Today there are thousands of emails with links to various research studies stuffing my inbox, poised to assist. They span the gamut, from research that bolsters my findings to studies that appear to refute them. The latter always intrigues me, as I am constantly on the lookout for anything that might expose a weakness in my conclusions. To date, every potential gotcha I've come across has had a fatal shortcoming.

For instance, when *The New York Times* reported in February 2025[263] that a drug similar to Ozempic had failed to treat Parkinson's disease in a trial, I was skeptical. A "rigorous," randomized study "showed absolutely no benefit or slowing of the course of the degenerative disease after 96 weeks." Observers called it "hugely disappointing" and "a sobering moment" after earlier epidemiological studies exhibited real promise. People on GLP-1s were getting diagnosed with Parkinson's far less frequently than others not on the medication. So why did it fail so conclusively?

Researchers were testing exenatide (Byetta), a first-generation GLP-1 drug given twice daily for diabetes. It should have helped, at least a little. But for this trial, the injection was administered only once a week, with researchers reasoning that even at low doses, some positive effect should surface. Without knowing why these drugs are so effective—the study's researchers would have been unaware that they are starving out Lyme disease—letting spirochetes feed six out of seven days wasn't going to produce much more than transient bacterial hunger pangs. (In contrast, recent diet research in Parkinson's[264] has found low carbohydrate diets lower the risk of the disease, while ultra-processed food increases it.)

While finding missteps like this can be reassuring to my own hypotheses, the most rewarding experience for this amateur scientist is the burst of elation that comes when a pattern fully crystallizes and the dots connect, exposing a revelation that promises to upend existing, long-entrenched medical beliefs. In my memoir, I referred to these eureka moments as "hallelujah booyahs,"[265] or HBs for short.

What I've exposed thus far, documented in detail in *Sit Down Before Reading*, is merely the dawn of what is certain to be the most incredible era in medical history, a health renaissance. Since writing the final pages of my memoir nearly two years ago, I've continued to, unintentionally, stumble on the telltale, distinctive patterns of Lyme disease, and often in areas I never expected to be connected to the bacterial infection. I need to share what I've recently discovered (all while writing this)—an unexpected, dense thicket of HBs that conclusively throttles what we thought we understood about health. Like most of my other discoveries, these will be met with disbelief, extreme skepticism, even outrage.

At times science can be profoundly uncomfortable.

Hallelujah Booyahs

After *The New York Times* published a piece in March 2025[266] on the challenges of treating acute and chronic back pain—Lyme disease is synonymous with lower back pain—I found compelling evidence, underscored with women bearing the brunt, that the bacterial infection plays an outsized role in the leading cause of disability worldwide.[267] Chiropractors aren't treating back

injuries due to poor lifting techniques; they are treating our favorite bacterial infection. No wonder their unconventional methods often raise eyebrows—they are the original Lyme practitioners. (That also means anything connected to back pain is suspect, particularly the mysterious, so when a March 2026 headline in *The Washington Post*[268] called attention to "the surprising link between back pain and a sensitivity to loud noises," maybe it's not so surprising.)

Then four months after the *NYT* piece published, a headline in *The Washington Post*[269] raised questions: "Allergies seem nearly impossible to avoid—unless you're Amish." Amish communities are largely isolated and famously insular, hallmarks of so-called Blue Zones. Investigating further, evidence of Lyme's involvement surfaced, the patterns unmistakable with an abundance of autoimmunity issues in people suffering from allergies, all piled predictably on the female gender. Other conditions also grabbed my attention, premenstrual syndrome in particular. PMS has a noticeable connection to manganese during the luteal phase of the menstrual cycle, the phase when the health of females is most at risk.

While there are so many fist-pumping hallelujah booyahs yet to be uncovered—news recently broke that GLP-1s dramatically reduce fractures,[270] an unsurprising finding given that manganese is largely stored in bone—I landed on a deeply unsettling HB, one I initially debated sharing. It is bound to be both controversial and a distraction, but I am newly smarting over a similar discovery that I opted not to disclose in my memoir for the same reasons.

When I first realized that vaccines were awakening dormant cases of Lyme disease in 2023, I knew that every illness I had linked to the infection would eventually crop up in studies. But oddly, cases of cancer had not yet been reported in any medical journals. I had planned to warn readers to expect it and not to panic but then became worried that if such studies didn't soon surface, people would jump to conclusions claiming a coordinated coverup, overshadowing the evidence I had found that vaccines are safe. So I punted.

That was a mistake. In July of 2025, Italian researchers, apprehensively, made the cancer connection.[271] Then in September, a South Korean study[272] reported, as predicted, that cancer rates indeed increased after Covid-19 vaccinations, spurring researchers to question the results and vaccine skeptics to claim vindication—justifiably, albeit incorrectly—just as I had feared. (In contrast, because vaccines can prevent illnesses that are likely to awaken dormant cases of Lyme, vaccines are also linked to a lower risk of dementia[273] and all-cause mortality.) So this time I'll not hold back; this next discovery impacts the health of too many for me to ignore. (Besides, both scientists and AI would have eventually made the connection after correcting for faulty data; I ask only that you follow the evidence where it leads.)

In February, a headline in *The New York Times*[274] gave me pause. "Nearly One in 10 U.S. Adults Identifies as L.G.B.T.Q., Survey Finds," a rate that has tripled since 2012. But it was the paper's accompanying chart on the generational differences of those identifying as LGBTQ that lit up my *Lymedar*, mirroring the distinct generational pattern of Lyme disease. Investigation revealed that all the other markers for the bacterial infection lined up in lockstep. I even found suggestive proof of where the atypical gender development might originate—in the thyroid—and when it occurs.

For most, it appears to manifest before birth, with both sexes equally affected. But among bisexuals, who typically discover their dual sexuality in adulthood, gender prevalence is predictably dominated by women. Alarmingly, bisexual women die an estimated 37% sooner than heterosexual women[275] (lesbians 20% sooner). This astronomical deathrate is not explained by sexual discrimination alone, as researchers have proposed.

I expect many of you to be especially dubious of these latest discoveries, so I've included more detailed research at the conclusion in Appendix A. The science behind these discoveries never breaks. The manganese-Borrelia connection is so entrenched in modern medicine that it plays a clandestine role in countless studies, appearing in research that otherwise would seem to be completely unrelated to a bacterial infection.

—Chapter 8—

Shell Shocked

Although many of you likely are feeling shell-shocked—a term from WWI that, aptly, is more likely the result of Lyme rather than munitions, established earlier—maybe an epidemic of this size shouldn't have been completely unexpected. The Plague of Justinian, The Black Death, The Spanish Flu, all swept across the globe. Even Nostradamus predicted the rise of an ancient plague, one that conveniently was foretold to occur right about now, in the mid-2020s. "A great pestilence from the past returns, no enemy more deadly under the skies," reads a quatrain.

It all sounds unreal, a wave of sickness you only read about in history books. But the more you try to dispel these findings—it can't possibly be *this* bad—the more you will only reaffirm them. The evidence swirls around us in research and the press, teasing us with bold headlines daily.

Just in a two-week period in early September 2025: "Couples are more likely to share psychiatric disorders, but why?," "Weight loss drugs can halve heart patients' risk of early death, study finds," "Treatment for psychosis may be 'fundamentally flawed', study finds," "Could glucose be the key to next-generation cancer treatments?," "It's not your imagination. Your period may be making your depression worse," "People who live to 100 have a unique relationship with disease," "Ozempic really could turn back the clock on your biological age," "A forgotten cancer is rising in young people, and experts are puzzled," "Heart attacks may be linked to bacterial infections, study finds."

This stream is endless, with each maddeningly fuzzy medical mystery suddenly coming into focus, eminently explainable, when viewed under the lens of Lyme disease. As past studies are gradually reinterpreted (an immense project that AI can assist with), everything will start making sense.

"Experts found that patients of adolescent mental health services who were treated with the antibiotic doxycycline were significantly less likely to go on to develop schizophrenia in adulthood compared with patients treated with other antibiotics," excitedly announces the news release[276] about a recently-published surprising November 2025 study in the *American Journal of Psychiatry*.[277] Except it's not so surprising when doxycycline is the standard treatment for Lyme disease.

"While roughly the same number of prepubertal girls and boys experience migraines, the prevalence more than doubles for women after puberty," reads a September article in *The Washington Post*[278] on why women oddly experience more pain than men. Except it's not so odd, it's just that menstruating women have more of the trace element Lyme disease requires to cause damage, manganese.

Recently there has been increased attention on the dramatic rise of autism worldwide, particularly in the United States. It's not spiking because pregnant women are taking Tylenol, as recently suggested by some,[279] but it's not unexpected that there appears to be a correlation. When

pregnant moms report taking the anti-inflammatory for chronic back pain,[280] disorders like Ehlers-Danlos syndrome,[281] or other pain relief not associated with injury, that suggests an active case of Lyme, elevating the risk of birth defects including autism. New research published in December of 2025[282] excitedly argued that a "staggering number, more than half of autism cases, could be prevented with the right interventions, [proposing] a 'three-hit' theory suggesting that genetic susceptibility combined with environmental exposure and prolonged period of physiological stress contribute to autism." University of California at San Diego's Robert Naviaux and his research team believes that it "may be better understood as a metabolic and inflammatory syndrome shaped by both biology and environment." The bottom line? "At the center of his theory is the cell danger response, a temporary survival state triggered by perceived threat." An autoimmune-like response overreacting.

Inflammation, a mix of genetic and environmental factors, the body turning on itself? That sounds painfully familiar. And yes, there is a manganese connection. "In a case-control study of autism spectrum disorder (ASD) and non-ASD controls, thyroid dysfunction and Mn exposure were associated with increased risk of ASD[283] and increased severity of ASD symptoms." It's just a flavor of our bacterial infection.

So when Justin Timberlake revealed his struggles with Lyme[284] at the end of July 2025, calling it "relentlessly debilitating," it wasn't a shock, it was just another sadly predictable data point. As dormant cases of the bacterial infection increasingly awaken, though, JT's diagnosis will stand out as a rarity. Most will not get a proper diagnosis. And most will not be so public. Instead, illness will rain onto your friends, family, children, partners, perhaps yourself.

Whether you realize it or not, all of us have personally experienced the devastation that Lyme brings. And we will continue to. It is inevitable, inescapable. But it is not unstoppable.

Dismantle or Defend

In the span of a few months this past year, I helplessly watched a neighbor struggle with IBS, a close colleague get hand surgery for a suspected arthritic condition, a cousin get diagnosed with Parkinson's in his early 50s, a dear friend struggle with an infection that would not resolve, a long-time friend complain of unexplained vision issues, another dear friend fight an unexpected case of early onset breast cancer, and my closest confidant bedbound for weeks due to back pain. And in that same timeframe, incredibly, I was forced to say forever goodbyes to a father who perished from Parkinson's disease, a longtime friend who passed from liver cancer at 57, a dedicated husband who slipped away with dementia, a father of 7-year-old twins who died in his early 50s from a heart attack, and a new mother who succumbed to a raging, uncontrollable infection at 40.

Her name was Sarah Cady Sartorius.[285] She had intended to read my memoir when the demands of motherhood lessened. If she had, she would have read my opinion that sepsis is a direct byproduct of Lyme disease. She never got the chance. I've lost my first and closest friend diagnosed with multiple sclerosis, a woman with the radiance of a thousand suns, and I am crushed beyond

imagination. The letter I wrote to her parents after her passing is one letter I hope to never write again.

Her death from likely sepsis and its cytokine storm was not an outlier, a one off. It was a bellwether. NASCAR driver Kyle Busch[286] died in May of 2026 at 41—his family confirmed that severe pneumonia had progressed into sepsis, resulting in rapid and overwhelming complications. Actor and singer Billy Porter, who has lupus, and pop icon Madonna survived recent bouts. It's going to get infinitely worse with inaction.

Sepsis isn't just any infection, and it recently took another colleague of mine in his 40s, punishing him with a series of strokes before he passed. And Lyme's fingerprints are all over it.

According to the CDC,[287] "most people who develop sepsis have at least one underlying medical condition, such as chronic lung disease or a weakened immune system." It's a relentless killer, responsible for 20% of deaths worldwide says the World Health Organization.[288] And it has baffled scientists for ages. "Despite years of research, the pathophysiology of sepsis is still poorly understood, and there is not yet a specific treatment that targets the immune response to sepsis."[289] (To date, intravenous ceftriaxone has been most reliable.[290]) The initial infectious trigger could be anything, from pneumonia to a car accident to a "playground scrape."

Hmm. This all sounds suspiciously like our villain. Does the geographical pattern fit, with Western nations leading the charge? It sure does.[291] Does autoimmunity increase susceptibility and mortality? That's the title of a 2022 journal article.[292] Are the GLP-1 weight-loss drugs associated with reduced sepsis and organ injury? Again, that's *literally* the title of a 2024 study summary![293] Even the ketogenic diet improves a slew of clinical measures[294] in septic patients. And research has confirmed[295] that levels of iron and manganese, notably, are "significantly lower" than other elements in critically ill sepsis patients compared to healthy controls. But the smoking gun? The inevitable aftermath following recovery from a bout of the illness.

As in long Covid and so many other "chronic" conditions, patients with post sepsis syndrome[296]—an official diagnosis that affects upwards of *half* of those who get diagnosed—experience a vast range of debilitating, lingering symptoms. The tired description of undiagnosed Lyme is by now so familiar that you should be able to recite the telltale signs[297] by heart. Fatigue, joint pain, rash, swelling, brain fog, depression. Symptoms that predictably never go away,[298] as "many say that life is never the same after sepsis."

Of the five tenets of SHARDs, I've called the last perhaps the most important: Dismantle or Defend, do not Defer. This call-to-action strikes at the heart of why most revolutionary medical theories take years, if not decades, to reach a level of acceptance. Deference, the commonplace capitulation to established hypotheses, means questionable science, once established, rarely gets challenged.

At this point I had planned to ask for your help, stressing that the health of humanity cannot wait. That passively waiting for technology to catch up isn't a strategy. That waiting for concrete, ironclad evidence in the form of wriggling spirochetes—a bar that will eventually be met in time—is a

recipe for doom, as death and disability will follow without respite. That now, this moment, is one of the gravest crises facing civilization. And then I realized I shouldn't have to rally the troops.

Nothing unifies and inspires like a common goal to preserve your own health and the health of loved ones. Action takes courage, conviction, resolve—attributes that soar during the most challenging of times. And we are in a historic tempest.

We now know the carnage overwhelming humanity is largely being driven by Lyme disease. Without directly addressing the bacterial infection, which, if this evidence is correct, has now spread to most people on planet Earth, all efforts to improve human health will fail. Hope, though, is nearing a rolling boil.

Our existing quiver of emerging options to fight Lyme disease—antibiotics, carb-reducing diets, and GLP-1s—can help mitigate the surge of sickness overwhelming society until better choices arrive. Better still, another potential treatment for Lyme disease is already here, hiding in plain sight.

Achilles' Heel

You're probably aware of the counterintuitive findings that coffee—considered a vice by many—is good for you. Potentially really, *really* good. The Mayo Clinic goes so far as to call coffee, "An unexpected ally for wellness." One of their doctors, Donald D. Hensrud, explained its benefits this way.[299] "In coffee drinkers, there's a decreased risk of type 2 diabetes, Parkinson's disease, liver disease, certain cancers including liver cancer, depression and suicide, kidney stones and gallstones, and overall mortality." New reports suggest that drinking 3-4 cups daily could add 5 years to lifespans.[300] There's also evidence that it lowers the risk of Alzheimer's disease, stroke, mental illness, and cardiovascular disease. Sound familiar?

The wide range of bewildering benefits track with the astonishing successes seen in GLP-1. Similar outrageous wellness benefits have been seen in the consumption of red wine, tea, and dark chocolate. Doctors aren't exactly sure why. Antioxidant protection is their best guess. Potentially their anti-inflammatory properties. Resveratrol? Caffeine? Maybe flavonoids?

Tannins are a type of flavonoid, and the latest research of flavonoids couldn't get much hotter[301] with their potential to promote healthy aging, prevent chronic diseases (like cancer, cardiovascular, and neurodegenerative diseases), and improve brain health. But there is an important caveat. "There is a long-standing puzzle: flavanols are poorly absorbed by the body (the fraction that actually enters the bloodstream after ingestion). If only small amounts reach circulation, it remains unclear how they exert measurable effects on the brain and nervous system."

It's not about what *is* being absorbed—tannins are known to act as anti-nutrients by binding to proteins and minerals, reducing their absorption—it's all about what *prevents* manganese from being absorbed. From past research, I knew that iron played a role, but I had neglected to investigate further. I was dumbstruck.

Non-heme iron, found in legumes, seeds, nuts, dark leafy greens, fortified foods, and certain dried fruits, uses the exact same transporter as manganese, DMT1, muscling it out.[302] Meanwhile, fiber physically binds to manganese,[303] preventing its absorption. Now there is a clear reason why it reduces the risk of heart disease, stroke, chronic diseases, and many cancers. Tetracyclines, the class of antibiotics that treats Lyme disease, prevent manganese absorption through chelation.[304] Calcium in high doses, which has been linked to lower rates of osteoporosis, breast and ovarian cancers, and more, easily crowds out trace amounts of manganese.[305] And tannins. Tannins, the so-called anti-nutrients,[306] are found in the highest concentrations in coffee, red wine, tea, and dark chocolate.

Look more closely at the foods that manipulate manganese and you'll see the core elements of what is considered the world's healthiest diet backed by science: the Mediterranean diet. It works by denying Borrelia access to its required manganese, its startling effectiveness no longer a mystery. Regular consumers of these goods, with robust daily consumption, enjoy health benefits that have confounded scientists for decades.

No wonder the Mediterranean diet has been the belle of the diet ball, preventing cardiovascular disease, lowering blood pressure, dramatically lowering the risk of type 2 diabetes, reducing the incidence of cancer including breast cancer, and reducing all-cause mortality by nearly a quarter. No wonder Europeans and their proclivity for wine and coffee have enjoyed a health edge over Americans in recent years. And no wonder studies often appear so conflicting.

Scientists have debated the "French paradox"[307] for decades, recently coming to the conclusion that it must be a mirage, a myth. How could the French, with their cholesterol-laden, high-fat diets and copious consumption of red wine, enjoy such low rates of heart disease? Now we know—healthy fats are fine (re: the success of high-fat keto diets and the copious use of olive oil in Mediterranean diets) and regularly drinking tannic-heavy red wine starves Lyme of its manganese. Tannins simply prevent the absorption of the trace element Lyme requires, essentially neutralizing the spirochetes and preventing them from doing much damage to their host.

Given what we've all been taught about these vices, this is hard to swallow. But try to find a red wine study that shows negative health effects. They don't exist, as researchers have discovered. "There is no evidence of an association between moderate red wine consumption and negative health outcomes," found an exhaustive 2023 review of 74 studies.[308] Most were positive, a grand total of zero were negative. "A beneficial effect of moderate red wine consumption was consistently seen for mortality and dementia, along with certain cancers (e.g., non-Hodgkin lymphoma) and cardiovascular conditions (e.g., metabolic syndrome)." A Rutgers University article[309] even cited an especially remarkable finding from one Danish study: "People who drank three to five glasses of wine per day had a 49% reduction in mortality rate than people who never drank wine."

If you investigate the effect of consuming food and beverages with tannins in chronic diseases, you'll get the confirmation your doubting brain needs. A 2017 study examining the consumption of red wine in MS[310] patients found lower rates of disability with patients who drank more than four glasses a week compared to abstainers, but dosing matters. Patients who drank just 1-3 glasses of

red wine per week accumulated brain lesions faster. Similarly, two independent studies found that the high consumption of coffee[311] (exceeding 900 mL daily) was associated with "substantially" reduced risk of developing MS.

Even studies of tannin-laden dark chocolate and tea, despite both being rich sources of manganese, have shown impressive health benefits. In September 2025, research dropped that showed that cocoa extract supplements could reduce aging-related chronic inflammation,[312] which followed up on research that found chocolate "significantly reduced death by cardiovascular disease." And tea, which originated nearly 5,000 years ago in China as a medicinal tonic. has long been attached to better health, confirmed by repeated studies including a 2025 study[313] that found that "the evidence is solid for the prevention of cardiovascular diseases, obesity, diabetes, and some types of cancer" as well as "the prevention of cognitive decline and muscle loss."

Denying Borrelia manganese appears to lead to a cascade of health benefits. This led to an intriguing brainstorm, which, if it were true, would remove all doubt, fully exposing Borrelia and its signature path of destruction.

The recent death of Dr. Jane Goodall, the celebrated primatologist, brought to the forefront the surprising interconnectedness of species. She and Darwin saw something others could not. What they discovered was brilliant, arguing that we have an innate kinship with all animals. But what if they had taken their research even further and pushed these connections to their absolute limits?

We have mountains of scientific support through human studies not only for how Lyme spreads and the kind of damage it can cause, but also for the two primary types of interventions that can slow the disease: denying the bacteria carbohydrates and manganese. Now let's drastically expand that premise. Borrelia's reach shouldn't stop at us.

Although humans sit atop the food chain, ticks—likely the original vector for Lyme—don't particularly fancy where they get their next blood meal. That means all land-dwelling mammals should be at risk of Lyme, the disease not only spreading by ticks, but also, once the host is infected, by sex and by birth. These animals should be sick like us and, by extension, the same factors that protect us should also apply to the animal kingdom.

Two huge populations—humans and mammals—with identical illness patterns as well as defenses would be devastating evidence. But these distinctive patterns have never been compared and evaluated in research. After all, medical specialists and biologists don't typically swap notes.

They should have.

—Chapter 9—

Of Mice and Men

There was a natural place to start my investigation of the animal kingdom: with mouse models used in medical studies. My first task was to find out everything I could about these laboratory-grade rodents. Mice and rats share 85-90% of their genes with humans, which is a primary reason that a staggering 120 million are used in research annually.[314]

There are tens of thousands of strains of mice used in experiments, but only a few hundred are used regularly, mostly inbred strains to be genetically consistent. The earliest of these strains date back to the early 1900s—C57BL/6, or Black 6, is a popular one—and variations of these lines are the ones most used today. They are generally rather healthy, but can still spontaneously develop chronic illnesses, autoimmune-like disease, and cancers on occasion, much like people at the start of the 20th century. These mice also experience low-grade inflammation,[315] or inflammaging, which scientists have long connected to the aging process. Female mice, predictably, display stronger immune activation.[316]

If Lyme disease is indeed driving inflammation in humans, suggested by the absence of inflammaging in the Tsimane, it must be doing the same to most laboratory mice. After all, any mouse strain used for medical trials needs to have a founding pair. With mice being among the animals most likely to carry Lyme disease bacteria[317]—more than 90% test positive—finding Lyme-free examples would have been iffy a century ago, and a near impossibility today. That means that any research using these laboratory mice has been compromised. But there is a notable, revealing exception.

Like isolated Indigenous peoples and super agers, there are strains of mice that rarely show signs of inflammation due to aging: axenic, or germ-free, mice. This striking anomaly is why Nobel-nominated bacteriologist James A. Reyniers[318] warned in a 1959 report[319] that "the science or art of detecting contamination is always the limiting factor and is at best a temporary situation." In essence, if current testing methods cannot reliably identify the existence of an infection, there can be no assurances that one isn't present, which is why he advocated for using germ-free specimens in clinical trials. For a time, other scientists recognized this issue,[320] as many pathogens "still turn up in laboratory animals and represent unwanted variables in research." But as today's lab mice are almost "too healthy," poorly reflecting the increasing health issues of humans, many scientists instead have pushed the idea to make these research subjects more representative: use free-range lab mice,[321] which are reliably sicker and, arguably, a more accurate predictor of health outcomes (not to mention vastly cheaper to study than sterile axenic mice). On the surface, this makes sense.

"Flawed studies may have smothered life-saving insights and interventions," cautions a 2023 piece in *The New Yorker*,[322] noting that treatments might appear to have failed if experiments on healthy mice are misleadingly ineffective. "Others have likely sent researchers down scientific dead ends."

Using "wild" mice would prevent that, the article reasons, citing wildling research that accurately predicted the dangers of high-fructose corn syrup.

Herein lies the crux of the problem, and easy answers are nonexistent. Borrelia burgdorferi spirochetes feed on glucose; high-fructose corn syrup would have been a feast from the gods for the bacteria. Studies using these mice would then guarantee that any clinical trial using them would be infected with an underlying, undetectable threat, providing unreliable, potentially dangerous results. Conversely, if you *don't* use them and unknowingly treat a patient with Lyme disease, which now is more likely than not, catastrophic results could also follow. In 2006, an experimental treatment for autoimmune diseases and lymphoma[323] nearly killed six healthy volunteers after an unrelenting cytokine storm set in shortly after the drug was administered—TGN1412 was confirmed safe in earlier animal studies after being given doses that were 500 times stronger than the patients received—leading to multiorgan failure and causing lifetime health issues.

Germ-free mice are not the answer. Free-roaming wild-caught mice are not the answer. Mice strains from the early 1900s are not the answer. Without curing active Lyme infections in both mice and men, no clinical trial using rodents will ever be truly accurate. (And when we succeed in eliminating the bacterial disease, the need for laboratory mice—projected to be a $5.8 billion market[324] by 2031—will rightfully and mercifully plummet.)

The necessary groundwork was complete. If most humans are infected with Lyme disease, and most mice are infected with Lyme disease, it stands to reason that the bacterial infection, over tens of thousands of years, has overrun not only humanity, but also the animal kingdom. Mice are just the proverbial canary. Now came the hard part. But thankfully, science gifted us a set of tools to ferret out Lyme's involvement.

A Riddle of the Ages

I needed to approach this imposing problem differently than I did with humans. Most mammals do not menstruate, many have carbohydrate-rich diets, and my most reliable Lyme marker can't assist. Although mammals will exhibit autoimmune-like symptoms, a hallmark of the bacterial infection and an identifiable red flag in humans, autoimmunity is not roundly recognized or tested in animals. Cancer, however, is. With Lyme disease being the primary driver of many cancers in humans, other mammals should be equally susceptible.

This is going to get uncomfortable.

Since the 1950s, notably the same timeframe the concept of autoimmunity took hold, scientists have believed that both age and mass contribute to the genesis of cancer, a foundational principle in how it arises. Theoretically, a single abnormal cell mutates before it begins dividing uncontrollably, over time leading to the creation of tumors.

"If every cell division carries a certain chance that a cancer-causing somatic mutation could occur, then the risk of developing cancer should be a function of the number of cell divisions in an organism's lifetime," say researchers.[325] "Therefore, large bodied and long-lived organisms should

face a higher lifetime risk of cancer simply due to the fact that their bodies contain more cells and will undergo more cell divisions over the course of their lifespan."

But that's not the way it plays out in reality. That leads us to Peto's Paradox.[326] In 1975, epidemiologist Richard Peto observed that some large animals—such as whales and elephants—enjoy shockingly low cancer rates, in direct conflict with how cancer is thought to form and evolve. Ever since, scientists have struggled to understand why, another riddle for the ages thought to be unsolvable.[327]

So let's solve it.

We need to begin by boxing out other causes of cancer temporarily, particularly those caused by viruses or environmental contaminants. These are known problems, often restricted to specific vulnerable species or regional hot spots, that we can acknowledge and work around. How? Instead of looking at which animals routinely get cancer, which is unclear due in part to shorter lifespans of those in the wild (so many potential life-threatening hazards), let's investigate which animals *rarely* get cancer and live notably long lives. Those are the anomalies.

The human experience with Lyme provides the necessary trail markers we need to follow, starting with isolation. If ticks are the primary vector required for Lyme disease to infiltrate a species, we must first venture to animal "Blue Zones," the most inhospitable regions on planet Earth for the arachnids, starting with the oceans and the poles; ticks are largely absent from ocean environments and struggle to survive in extreme arctic conditions. If this hypothesis is correct, cancer rates in mammals trolling the oceans and residing in arctic climes should be remarkably lower than anywhere else on the planet.

They are. Whales, dolphins, walruses, and others who call the oceans and arctic climes home have remarkably low rates of cancer,[328] among the lowest on the planet. There are exceptions of course, like the beluga whales who live in the heavily polluted St. Lawrence Estuary,[329] but belugas who live in the Arctic are nearly cancer free. Now let's remove the safety net of frigid waters and look at mammals confined to dry land, specifically subterranean mammals who live exclusively under it, where ticks are blissfully absent. That's the darkened, isolated habitat of the naked mole rat and its relatives. Do they get cancer?

"Naked mole-rats seem to defy ageing and appear immune to cancer," reported the BBC[330] in 2022, noting that they have "extraordinary characteristics" that are intriguing scientists, including astonishing resistance to chronic diseases and extremely long life spans. In fact, as they age, "there are no significant changes in cardiac function, body composition, bone quality or metabolism," further befuddling researchers. Could the absence of Lyme disease alone account for their extraordinary health?

More than plausible. Complete isolation from the ticks that spread Lyme disease would confer an incredible health advantage. But most mammals restricted to terra firma aren't so fortunate. They all should be ideal fodder for infection. To understand why that's not the case, we need to pivot to

another reliable way to avoid the ravages of Lyme disease: insulation. Not the kind that keeps out the cold—the kind that keeps out Lyme.

Diet.

Upending Medicine

I've broken down why whales and aquatic-adjacent creatures lead such healthy, long lives, and why isolation provides a powerful defense. But there are other animals, like the aforementioned elephant, who enjoy minuscule cancer rates, and they roam the same patches of earth as Lyme-infected ticks. They appear to be a mishmash of varied species, an unlikely assortment—from rhinoceroses and giraffes; to goats and deer; to beavers and squirrels. Their cancer resistance, as well as that of other lucky members of the animal kingdom, has defied explanation, but there appears to be a trend. They are vegetarians.

"Herbivores' meatless diet cannot explain the differences in cancer resistance, which have accompanied species segregation since the Jurassic era," reflect puzzled researchers.[331] Dozens of hypotheses have been explored in depth, but none have taken root, with many scientists believing that it is species dependent, a complex interplay of factors with a protective evolutionary component.

Or not.

Darwin would have been dismayed that his theory of natural selection was so poorly misunderstood and misused to prop up so many fatally flawed hypotheses. Blaming defective genes. Believing mental illness is being passed down through generations. Suggesting that most cancers form randomly, an out-of-the-blue DNA hiccup. All are an affront to Darwin's signature theory—and all would have been selected out over time.

Big animals or small ones, evolution doesn't play favorites. In a broadside to modern medicine and decades of research, cancer is in no way a game of chance, a mix of bad luck and regrettable lifestyle choices. Cancer is largely not a genetic issue passed down from family. The answer lies in its prevention, and it is exclusive to plant-eating mammals, herbivores.

"It has often been proposed that diet may play a role in interspecies differences in cancer rates or lifespan," muse scientists, with the seeming frequency of sunrises.[332] It does, and in an enormous way, but again, researchers have never been able to connect quite all the dots. Borrelia's reliance on manganese connects them.

Carnivores have the highest risk of cancer among mammals, omnivores—who eat both plants and animals—fall in the middle, while herbivores are exceptionally resistant. The key lies in what they eat. Contrary to most human diets that focus on restricting foods—cutting carbs, cutting sugars, cutting calories—the cancer-starving diets of many herbivores hinge not on elimination, but on consuming tannins, the same group of compounds found surprisingly beneficial in human diets. While their plant-based diets provide non-heme iron, a reliable manganese transport blocker also enjoyed by omnivores (eating meat, carnivores absorb heme iron, which does not affect

manganese), high tannic diets are unique to the vegetarians. And the herbivores with diets highest in tannins enjoy the most protection from cancer and other diseases.

Squirrels, who often live in the thick of blacklegged tick territory, frequently show antibodies to Lyme disease,[333] but rarely experience any ill effects. That checks out. Acorns, a primary food source for these rodents, are chock-full of tannins, so much so that they cannot only be tremendously bitter and unpalatable, but also toxic to other animals.[334] Deer and goats produce specialized tannin-binding proteins in their saliva[335] to handle tannic vegetation, while elephants and rhinos[336] consume so much plant matter of all types that robust tannic levels are maintained. Beavers get their tannin fill from tree bark.[337]

Just how protective are tannins? To my knowledge, no modern-day animal that consumes a consistently high-tannin diet shares an elevated cancer risk—with one exception. Koalas. Eucalyptus is loaded with tannins, but koalas still frequently develop cancer due to a known retrovirus[338] commonly spread within the species. That's the entire list. Even the limited number of omnivores with tannin-filled diets, like bears, enjoy their cancer-protecting benefits.

Cancer, a disease that has perplexed healers since 3000 BC, largely has a common cause. Borrelia. The evidence—across all mammals, across all scenarios—could not be any clearer. There are no paradoxes. No genetic overreaches. No environmental uncertainties. No unfortunate lifestyle choices. No random mutations by chance. Just bacteria damaging DNA.

That's all.

Now that we know the genesis of many types of cancer, our manganese holy grail has one more surprising gift to offer. Hope.

Another Unexpected Weakness

Curious about what specific teas provided the most health benefits, I investigated popular types—black, green, oolong, white, and herbal—predicting that the healthiest would have the most tannins and the least manganese. I was taken aback. Green tea, with the least amount of tannins and easily the most manganese, was consistently deemed the healthiest in study after study, from having a protective effect on the recurrence of breast cancer[339] to promoting gut health and lowering blood sugar.[340]

I had been ruminating on this striking manganese-tannin paradox, my frustration mounting, when an unbelievably timely study out of Northwestern University and Uniformed Services University[341] published just as I was writing this section in November of 2025. It was led by Northwestern's Brian Hoffman and USU's Michael Daly, but the entire research team deserves to be named in full: Andrés F. Londoño, Ajay Sharma, Venkatesan Kathiresan, Jared Sealy, Robert P. Volpe, Cene Gostinčar, Utpal Pal, and J. Stephen Dumler. These researchers found that Borrelia burgdorferi are constantly adjusting manganese levels to find the sweet spot; as manganese levels increase, MnSOD levels drop and vice versa. Too little manganese weakens the bacteria's defenses, but too much can be toxic. "Future drugs could starve the bacterium of manganese, disrupt its

ability to form protective manganese complexes or even push it into toxic overload," the scientists excitedly reported. "Any of these approaches would leave *B. burgdorferi* wide open to attack by the immune system."

This was the research I needed to resolve any remaining inconsistencies.

The resolution to the apparent green tea paradox lies in Borrelia's exquisitely narrow manganese tolerance. Green tea's manganese content—the highest of any common tea—doesn't just nourish the bacteria, it can overwhelm it. Other teas starve Borrelia by blocking manganese absorption with tannins. Robust consumption of green tea floods it into toxic overload. Two different mechanisms, the same disruption of Borrelia's carefully maintained equilibrium.

This also explains why green tea's health benefits cluster so strikingly in women. With chronically elevated manganese from the iron-manganese seesaw of menstruation, women give Borrelia a more reliable food supply, meaning green tea's manganese overload hits the bacteria harder. A 2024 systematic review[342] confirmed what the manganese mechanism predicts: tea's protective effects at decreasing all-cause mortality are observed particularly in women—from reducing female hormone-dependent cancers[343] to a 35% reduction in lung cancer risk in nonsmoking women to reducing depression in postmenopausal women.[344] Its benefits extend to everything from inflammatory bowel disease to metabolic syndrome. But perhaps most telling: a study of nearly 10,000 elderly Chinese adults found that daily green tea consumption was associated with a 38% increased risk of hypertension in men[345]—but no effect whatsoever in women.

Women are unknowingly flooding Borrelia with manganese. Men, largely, are merely feeding it.

Daly calls manganese the Achilles' heel of Borrelia burgdorferi, echoing my conclusion from 2024 that the bacteria has a weakness. Unknowingly, this weakness is already being researched and exploited. Days before the Northwestern study dropped, *The New York Times* ran a story[346] with the subhead "Longevity labs, 'immortality islands' and grapeseed pills are part of China's national project to conquer aging, despite sometimes shaky science and extravagant claims." It might not be as unfounded as it might appear. In 2021, Chinese researchers had linked the flavonoid procyanidin C1, grapeseed extract, to dramatically increased lifespans in mice in a study published in *Nature Metabolism*.[347] What's loaded with tannins and attached to a bundle of (unconfirmed) health benefits? Grapeseed extract.[348]

Limiting manganese—either by restricting manganese-rich foods in one's diet or restricting its absorption—or overloading the bacteria with manganese are areas ripe for treating Lyme. While tackling manganese absorption—taking iron supplements (approved by your doctor), consuming more calcium and fiber, drinking tannic beverages, and avoiding manganese-rich foods—will limit the damage Lyme can do, it will not eradicate the disease. Even so, it is an intervention that people could take advantage of immediately without significant effort. The science-backed success of the Mediterranean diet is Exhibit A.

Overloading with manganese promises to be far more complicated.

Researchers have long believed that excessive, chronic exposure to manganese—first discovered in industrial workers and miners in the 1800s—can lead to a condition known as manganism, with symptoms that present as a syndrome called parkinsonism because it closely resembles Parkinson's disease. (It's not uncommon for miners to later develop full-blown Parkinson's after contracting the syndrome.) But if PD is merely a variant of Lyme, everything about manganism gets upended. Let's quickly break this down.

Manganese drives disease in Lyme, and women reliably have more manganese and, hence, more health issues. If men absorb more manganese, say through occupational exposure in mines or construction, they too should have higher rates of disease, just as menstruating women do. But critically, these miners should exhibit a broad array of symptoms, not just those associated with manganism. Did scientists get fooled again?

Recently, researchers have noted curious contradictions in studies of manganese exposure,[349] finding that it is virtually indistinguishable from Parkinson's, and that "there is a spectrum of neurologic effects associated with chronic, lower Mn exposures." More confounding still, those symptoms expand to include psychosis (sometimes called "manganese madness"), impaired motor function, impaired steadiness, tremor, behavioral, and cognitive dysfunction. But the industrial worker data goes further. They are generally sicker, with a higher risk of a constellation of illnesses typically afflicting women, from lupus[350] (a prevalence 10 times higher than expected in the general population) to rheumatoid arthritis[351] (a prevalence 3-4 times higher) and a host of other autoimmune diseases.[352] And metabolic disorders.[353] And cancer.[354]

It's the same pattern as Lyme disease. Clearly, being moderately exposed to manganese is giving spirochetes exactly what they want. Could overloading with the element be part of the cure as suggested? New research provides an intriguing blueprint to follow.

Blueprint to a Cure

In a February 2025 study[355] released in the *European Heart Journal*, Harvard and Tulane researchers made a notable discovery. "Coffee drinking timing significantly modified the association between coffee intake amounts and all-cause mortality; higher coffee intake amounts were significantly associated with a lower risk of all-cause mortality in participants with morning-type pattern but not in those with all-day-type pattern." Seven months later another study dropped,[356] in the *British Journal of Nutrition*, that found that a combination of coffee and tea—2 cups of coffee for every 3 cups of tea (along with water)—was the sweet spot for substantially reducing all-cause mortality.

That novel combo would place maximum stress on the spirochetes as they try to juggle dramatic swings of the essential element, effectively starving Lyme disease of manganese in the morning with coffee before toxically overloading it with tea in the afternoon. Except, I discovered, manganese starving/overloading technically isn't such a novel approach. Members of the animal kingdom figured it out long ago.

"Whether foraging on pastures or rangelands, herbivores encounter plant species that differ in their concentrations of nutrients," explain Utah State researchers in a sweeping study[357] on herbivores

and their unique diurnal eating habits. "[Plants] also all contain various secondary compounds that at too high doses can be toxic, but at the appropriate dose many of these toxins may have medicinal benefits. The quantity of forage an animal consumes depends on the other forages it selects because nutrients and toxins interact." A 2025 study[358] supports this, finding that "the key lies in balancing tannin levels and type (e.g., condensed, hydrolyzable, phlorotannins) to optimize their positive effects without compromising animal performance."

For herbivores, it's all about the manganese yin and yang balancing act. Timing matters,[359] as concentrations of both tannins and manganese found in plants can vary over the course of a day. That would be challenging to replicate in a controlled environment like a zoo. And that's an obvious problem. Generally, zoos confer protection from predators, starvation, and miscellaneous life-shortening nastiness often encountered in the wild. But for long-lived animals with few natural predators who rely on plants to keep their Lyme disease at bay, zoos and their regimented feeding schedules should then be potentially deadly. And research says they are.

A study published in *Science*[360] found that elephants in zoos die significantly earlier than those in the wild or even tamed elephants who freely forage. "African zoo elephants had life spans of about 17 years, whereas those in Kenya's Amboseli National Park lived 56 years," researchers discovered. Meanwhile, "the median life span for Asian zoo elephants was nearly 19 years, but at Burma's Myanma Timber Enterprise (a logging enterprise), it was almost 42 years. Death rates for infant Asian elephants were especially high in zoos."

(Before any readers get the idea to experiment with manganese interventions on their meat-eating furry friends—who, like most animals, almost certainly are infected with Lyme—don't. They cannot safely metabolize the same compounds humans can, one reason you never want to let your dog or cat eat chocolate.)

Following the timely eating habits of herbivores might be a template to follow for effectively treating a Borrelia infection in humans. Coffee in the morning, tea throughout the day, and red wine in the evening would appear to be a tannic sandwich filled with toxic manganese. But being untested for this purpose, there could be unintended consequences. Tannins also prevent the absorption of iron and other nutrients, and may do the same with medications. And too much of these interventions can be not only problematic, but also detrimental, particularly the risk of iron overload, the complications of alcohol in general, and the unknowns of high doses of manganese.

There are certainly other treatment options to investigate as well. Unknowingly, many of us are already using some. After all, cranberry juice and cranberry supplements help prevent and treat UTIs,[361] something I suffered with, particularly during my psychotic spell, as it is a common problem in Lyme.[362] (Not coincidentally, new 2026 research[363] warns that severe UTIs are an overlooked risk factor for dementia.) While antibiotics swiftly fixed my bladder issues—as well as my swollen ankles, or "cankles"—permitting me to permanently stop self-cathing, tannic-laden cranberries likely would have provided some relief had I been more open-minded. Although I've publicly scoffed at such remedies in the past, no more. So when I read about how the compounds

in turmeric or rhubarb might fight off antibiotic-resistant bacteria[364] or how plant-derived acids can boost tetracycline antibiotics against superbugs,[365] I pay attention.

Researchers have discovered, again unknowingly, two more ways to put the hard brakes on Lyme, albeit at a premium price. The newest is CAR-T therapy,[366] which has generated buzz in the science community, but with it costing in excess of $350,000 per dose, it's not a terribly practical option except in life-threatening situations. At about half the cost, bone marrow stem cell transplants have proven over the past two decades to be remarkably effective at treating more than 75 health conditions,[367] putting them into remission that can last years. Blood cancers like leukemia and lymphoma; genetic blood disorders like sickle cell disease; genetic metabolic and immune-related disorders; other cancers including solid tumors; and autoimmune diseases like lupus and multiple sclerosis. Even those with HIV have seen long-term remission,[368] compelling evidence that the disease involves Lyme in some capacity. But the dreams of an outright cure are a mirage, which I learned firsthand.

In 2010 I underwent a hematopoietic stem cell transplant (HSCT) in a desperate attempt to slow my cratering health. My transplant, part of the NIH-sponsored clinical trial HALT-MS,[369] put my disease into remission for five blissful years before it ultimately failed (thankfully my crushing seasonal allergies haven't returned). Costing upwards of $200,000 with an uncomfortable risk of death, HSCT is not a practical solution for most. But the biggest issue: it doesn't eradicate bacterial infections; it only puts them into hibernation. (How it does that isn't entirely clear, but copious antibiotics are part of the treatment and recovery regimen.) When it inevitably reawakens, Lyme may return appearing as the disease doctors were trying to treat, but more likely than not, it comes back as another malady, which has alarmed researchers for years,[370] believing the new complications were from chemotherapy itself. HSCT survivors face a mess of threatening complications. Two-thirds develop at least one chronic health condition, a fifth develop severe or life-threatening conditions, and those surviving five or more years post-transplant face up to a ninefold increased risk of late mortality—and a 30% lower life expectancy—than the general population.

Those are sobering facts. Over time, HSCT is doomed to fail. Health conditions I've connected to Lyme disease routinely rear their heads after transplantation: Secondary cancers, cardiovascular disease, endocrine and metabolic disorders, bone and joint conditions, organ and sensory damage, long-term immune issues, and autoimmune diseases. While HSCT is clearly no cure, exploiting the Achilles' heel of Lyme disease, manganese, should lead to one, or at the very least, to a vast improvement in treatment options. But with timing such an important consideration, determining precisely when and how to safely overload with manganese, and when to starve the disease of the trace mineral, will require clinical trials.

There are so many outstanding questions. Are tannic supplements, like grapeseed extract, viable alternatives to coffee or wine? Is supplemental manganese, outside of green tea (perhaps oversteeped with lemon to maximize extraction), a practical approach and, if so, how much for each gender? And then there is the complication of the Herxheimer reaction. Any successful

intervention, including diets, will cause the die-off of spirochetes, potentially triggering symptoms that will initially appear as disease worsening, frightening doctors worried of causing harm.

This is going to be impossibly hard, but it's not impossible. Now, science—with the support of motivated governments worldwide and an international coalition of researchers, pharmaceutical companies, and volunteers for clinical trials—needs to come to humanity's rescue. And we, all of us, need to muster the strength to forgive, to rebuild our recently fractured trust in science and medicine.[371]

Just as Newton, Darwin, and Einstein built their iconic theories in fundamentally different ways, we needed to approach this problem differently, wildly so, from the standard practice of evidence-based medicine. The path forward will be difficult. But for the first time in history, we know where it leads.

—Chapter 10—

The Burden of Proof

Your head at this moment is almost certainly spinning furiously with disbelief, trying to make sense of what you've read, just as I warned in the opening paragraphs. With Lyme disease swamping not only humankind, but also the animal kingdom, the myriad health hypotheses embraced by so many for so long effectively crater one by one, undone by our holy grail of medicine.

Darwin couldn't "deduce" evolution in a lab; instead, he used abductive reasoning, or "inference to the best explanation." He gathered vast, diverse facts—from fossil records to finch beaks—and argued that natural selection was the most plausible "best explanation" that unified them all. His theory was accepted because it explained the evidence better than any competing idea. Similarly, without a plausible explanation to justify the gender disparity in chronic illnesses—for example, a study that finds women between the ages of 15 and 50 drink twice as much bottled water as men (opening the door a crack to support the theory that microplastics may be to blame) or that they consume twice the amount of ultra-processed food (giving renewed life to the microbiome theory)—any alternative to Lyme and its insatiable need of manganese is DOA.

Lyme disease is the simplest and most likely answer, its gender disparity solution effectively eliminating competing theories. It isn't just the best explanation, it's the only explanation that doesn't break, ever.

The Chinese study on Borrelia burgdorferi antibodies, the one that discovered 14.5% of the world's population was infected with the bacteria that causes Lyme, illustrates the flawed reasoning of the past with the efficiency of a boardwalk portrait artist. How can doctors remotely trust the tests for Lyme disease when more than a billion people, many of whom have never been bitten by a tick or travelled in tick-infested areas, test positive for the infection? Either those are "false positives," thereby confirming the testing for Lyme is woefully inaccurate, or the tests are at least somewhat accurate, confirming that the disease is spreading without the aid of ticks. There are no other possible explanations. And yet we are told to trust the tests and rest assured that the risk of getting infected is restricted to tick habitats. Even a fifth grader can see that both cannot be true.

Then there are the medical hypotheses that routinely break upon closer inspection. Take the Hygiene Hypothesis, the idea that a lack of early childhood exposure to infectious agents and parasites increases susceptibility to allergic and autoimmune diseases by suppressing the "natural development" of the immune system. Wealthier nations must be too clean! Except the poorest communities in these countries, living in crowded, often "unhygienic" conditions, tend to be sicker. It doesn't add up, so a paradox is slapped onto the hypothesis, and it continues to chug along, unchallenged.

If a hypothesis requires a parade of paradoxes to explain away inconsistencies, the hypothesis is irreparably flawed. The Middle East, particularly the United Arab Emirates and Saudi Arabia, has

soaring rates of chronic disease. That seemingly supports the Hygiene Hypothesis but, being located near the equator and bathed in sunshine, thought to confer health benefits, the region outright bucks the popular Latitude Gradient and the Sunlight hypotheses. Now, instead, consider that residents of wealthier nations have more upward mobility, allowing them to travel and spread Lyme far more efficiently than those living in poorer nations where air travel is more fantasy than reality. This truism goes back centuries, as a study of European nobility[372] found that "it was not unusual for aristocrats to experience higher mortality than average people, especially people who lived in the countryside." Blamed, of course, on glutinous excesses. Again, simple inference to the best explanation, no paradoxes needed.

The theory of Lyme disease being behind our most devastating health crises unifies it all. All of it. The skyrocketing rise of autoimmune diseases[373] and their connection to so many debilitating conditions, the growing presence of autoantibodies[374] even in seemingly healthy people, the startling discovery that most heart attacks in younger women aren't caused by clogged arteries at all,[375] the unexpected discovery that immune cells shed their own sugar coating to invade inflamed skin,[376] the surprising absence of diseases and cancers in isolated Indigenous communities,[173] why diabetes drugs are tamping down cancer[377] as rates are soaring in the young,[378] why there has been a resurgence of gout,[379] the reason why ADHD significantly shortens lifespans,[380] why cardiovascular, kidney, and metabolic diseases cluster so reliably together that medicine invented a new name for it—CKM syndrome, now affecting nine in ten Americans,[381] why a condition affecting 170 million women required a century and a name change to PMOS before medicine admitted it was never really about ovarian cysts at all.[382] and on and on. It all makes sense.

This marvelously complex puzzle of puzzles only fits together one way. The thread that connects it all—the illnesses flooding humanity and the desperate interventions to tame them—is Borrelia. But history has shown us how scientists think; it hasn't changed in hundreds of years.

Where's the supporting physical evidence that provides absolute proof?

There is a famous scientific aphorism that the absence of evidence is not evidence of absence, a foundational principle in logic, science, and law. The evidence has always been there, but because Lyme disease is thought to be contained—localized, and not widespread—any sign of its presence is dismissed, chalked up as a curious coincidence, not cause. The damning proof drapes medical research, an all-but-invisible cloak of evidence that is smothering in its breadth.

Borrelia found in multiple sclerosis in 1925[383] and again in 1957[21]—and again in 2000,[384] and again in 2023,[385] each time noted, each time set aside. Found in skin cancer in 1991.[386] Found in heart disease in 1991.[387] Found in skin disease in 1993,[388] even in patients who tested negative for Lyme. Found in connective tissue disease in 1994.[389] Found in psychosis patients in 1999[390] (symptoms resolved with antibiotics). Found in patients who died of sudden cardiac arrest in 2013.[391] Found in Parkinson's disease in 2003.[392] Found in Alzheimer's disease in 2004.[393] Found in sarcoidosis in 2018.[394] Found in ALS patients in 1990.[395] Found in dementia brain tissue in 2021,[396] fifteen years after the patient's Lyme was considered treated. Found in a leukemia patient in 2024;[397] cancer

lesions stabilized with antibiotics. Found in human fat tissue in 2026[398]—the first study to look (Yale School of Medicine, preprint).

And then, soberingly, in a single 2019 autopsy, Borrelia was recovered from all four organs simultaneously—brain, heart, kidney, and liver—in a patient who had received sixteen years of antibiotic treatment.[399] The bacteria have the tenacity of a wolverine, the cunningness of a fox, and the adaptability of a raccoon.

Borrelia has always been here, hiding in plain sight—like the twisty spirochetes on the cover of this treatise that you almost certainly didn't notice.

By now, a particular objection has likely crystallized in the minds of skeptical readers, especially those with medical training. It goes something like this: *Dave has a theory, and like any true believer, he's finding confirmation everywhere he looks. Lyme explains everything? That's not science—that's a hammer looking for nails.*

It's a fair objection. And it deserves to be addressed.

Cognitive bias is real, pervasive in medicine, and it has caused genuine harm. Doctors anchor to the first plausible explanation and stop looking. Researchers design studies to confirm what they already believe. Entire fields organize around a consensus that turns out to be wrong—and then defend that consensus long past the point where the evidence justifies it. I am not immune to this. No one is.

But here is what that objection quietly assumes: that the medical establishment, in dismissing a hidden infection as the root of chronic illness, is operating *without* cognitive bias. That researchers who have spent careers studying autoimmune disease, or mental illness, or obesity—that these researchers arrive at their conclusions fresh, unencumbered by decades of investment in their specialties.

That assumption does not survive scrutiny.

The bias critique cuts both ways. It always does. The question is never whether bias exists—it does, on all sides—but which theory, when held up against the full weight of evidence, requires the least amount of special pleading to survive.

Throughout this document I have identified, one by one, the places where medicine's reigning hypotheses don't align with their own data. Not fringe objections. Structural failures, the kind that, in any other field, would prompt serious reconsideration of the underlying model.

The microbiome theory asks us to believe that the catastrophic rise in autoimmune disease over the last half-century is explained primarily by changes in gut bacteria—while offering no convincing account of why those changes happened so dramatically, so universally, and so fast. It is a hypothesis in search of a cause.

The microplastics theory, the ultra-processed food theory, or any other theory for that matter, might explain a piece. None explains the gender disparity. None explains the Blue Zones. None

explains why isolated Indigenous communities, living without modern medicine but also without modern mobility, are largely spared. Offered the simplest of litmus tests—explain the gender gap—each one fails.

These hypotheses don't merely have gaps. They have gaps requiring additional hypotheses to paper over, which develop their own gaps, requiring further hypotheses still. This is not how correct theories behave.

Now apply the same standard to the Lyme hypothesis. Where does it break? Not where is it unproven; unproven is not the same as broken. Where does the data point *away* from Lyme rather than toward it? Where does invoking Borrelia as the hidden driver make the picture harder to explain, not easier?

I have spent years looking for that crack. I have not found one of significance. The startling amount dementia in Japan initially gave me pause, but Japan has made an outsized effort to maintain and improve public health—better quality diets, smaller portion sizes, regular exercise. Even though Borrelia has infiltrated the island nation, these interventions should hold off the disease for a time, but with old age comes weaker immune systems when the disease can gain a foothold. With the Japanese enjoying robust lifespans, rising dementia rates in the elderly tracks. Maybe it's not much of a crack after all.

There is, however, one anomaly I have yet to fully account for: the extraordinarily low cancer rates of bats. I haven't found a consistent pattern in the flying mammals. To thwart manganese absorption, fruit bats ingest tannins and those living in industrialized areas absorb enough manganese from the environment it might push Borrelia into toxic overload. (Vampire bats get ample iron, but it's heme iron, which doesn't affect manganese absorption.) But I'm not convinced; something else appears to be in play. Answers await here, perhaps novel treatments that thwart Borrelia. Of all mammals, bats hold the most promise to unlock one.

That's my list.

That is not the behavior of a biased mind finding what it seeks. A biased mind, searching long enough, eventually finds its confirmation and stops. A theory that holds under sustained, adversarial self-examination—one that keeps explaining new data it was never designed to explain, from GLP-1 drugs to Blue Zone longevity to the gender gap in autoimmunity to the surprising reach of diabetes drugs into cancer—is not behaving like confirmation bias. It is behaving like a correct theory.

So here is the challenge I would put to any researcher inclined to dismiss this on those grounds: dismantle it or defend it. Not "we haven't proven it yet." Not "the studies haven't been done." Find the contradiction. Find the population that should be sick under this model and isn't. Find the place where the manganese mechanism, the gender gap solution, the Blue Zone anomaly, and the spread pattern of chronic illness all point somewhere other than a hidden, globally disseminated bacterial infection. If you find it, I want to know. A theory with a real crack in it is better caught early.

But if, after honest examination, you cannot find it—that it just feels too overreaching or that it hasn't been validated by the institutions whose foundational assumptions it most directly threatens—then your objection isn't a rebuttal. It's a delay of the inevitable.

Kyle Busch, champion NASCAR driver, dead at 40 from sepsis. Eric Dane, dead from ALS at 53. James Van Der Beek, Catherine O'Hara, Anthony Head, Bonnie Tyler, Sam Neill—cancer took them all. And those still fighting: Chris Johnson, NFL star, ALS at 39. Danny Glover, Alzheimer's. Bruce Willis, whose mind built a career on precision and wit, currently lost to frontotemporal dementia. And Chesley Sullenberger—the pilot who held 155 lives in his hands on the Hudson River and brought every one of them home—now losing his mind to a disease medicine has spent decades and countless dollars failing to understand.

Medicine has been at these crossroads before. The evidence for handwashing was not subtle. The resistance to it was not scientific. It was human—the ordinary, understandable, catastrophic unwillingness to accept a paradigm shift in the profession.

Semmelweis was right. He died in an asylum, still right.

We refused to see then—but today we can. We must.

Learning to See

"To develop a complete mind: Study the science of art; Study the art of science. Learn how to see. Realize that everything connects to everything else." — Leonardo da Vinci.

When I first heard that famous da Vinci quote years ago, I didn't get it. When I started writing my memoir, referencing his "connectedness of everything" mindset, I didn't get it. When I ran across it again (and again), years deep into writing *Sit Down Before Reading*, I didn't get it. Now I get it.

Once the tent pole of autoimmunity collapsed, any medical issue with a close connection to the ill-conceived theory collapsed with it. It's unbelievable how a single misstep can cascade. With everything interconnected, it took one correction in medicine to upend it all, rewriting much of what we thought we knew about health. Genetics, nutrition, mental disorders, cancer, autoimmunity, chronic illnesses, metabolism, epidemiology, infectious diseases, and so much more. To understand just how deep this goes, choose any topic and then hold your breath when you consider the consequences.

Half of all people in the U.S. will experience some type of psychiatric disorder in their lifetime.[400] As mental illnesses vanish, a new, never-anticipated issue will arise, captured by a headline in the July 28, 2025, issue of *The New Yorker*:[401] "Mary had Schizophrenia—Then Suddenly She Didn't. *Some psychiatric patients may actually have treatable autoimmune conditions. But what happens to the newly sane?*" That's not so easily answered. After smothering psychosis for 20 years, Mary's schizophrenia lifted after cancer treatment. What does her future and reintegration into society look like now? How will the eradication of mental disorders affect suicide rates? Homelessness? Substance abuse—which was tied directly to late-stage borreliosis in a recent study[402]—and gambling addictions? Eating disorders? Gun violence?

For those actively suffering from Lyme, is it right to abandon, shun, blame, or punish them, perhaps with the finality of the death penalty, for having involuntarily contracted an unwanted bacterial infection, a disease wholly out of their control? Worse, our "rehabilitation" programs are not merely flawed, they often are actively, literally, fueling the disease. The diets of those incarcerated and the homeless—two communities with sky high rates of both mental health issues and chronic illnesses[403]—typically are laden with ultra-processed foods swimming in carbohydrates. Add the stress of living in prison or on the streets, combined with the ease with which viral illnesses spread in those populations, and it's no wonder few get better.

After my own bout with psychosis (accompanied with a now-explainable recurring bad back) and my complete detachment from reality, I know firsthand what it feels like not to be in command due to this infection. You have no clue, no concept, that you are in an altered or demented state.[404] Your addled brain will try to justify any action, no matter how convoluted. Thankfully I never hurt anyone or was institutionalized like Dr. Semmelweis, but that was just luck. I also had a wife who didn't give up on me, who didn't take the easy out and walk away. Many are not so fortunate.

When you are trapped in a state of psychosis, you are helpless against your mind's demons until it lifts, if it ever does. But sometimes, through treatment or time or luck, you can regain sanity. Only when that happens can you then reflect on your craziness, often with horror and regret.

When my sanity blissfully returned at the rather inconvenient time of 1 a.m. back in 2021, I had to tell my wife immediately, unsure if it would hold until morning. That was instantly followed by a string of sorrys for putting her through the scary hell of having to care for yet another family member with a mental illness (her brother was diagnosed with schizoaffective disorder). My experience isn't a one-off; history is riddled with examples. In the late 1600s, Sir Isaac Newton felt similarly after his 18-month bout of psychosis,[405] "characterized by paranoid delusions, insomnia, irritability, and loss of appetite," lifted. He did his own apology tour, and "expressed remorse to his friends for his accusations." Scientists are keenly aware of the savant/crazy connection,[406] noting that "throughout history, genius and madness have often dwelled together" and that "delusional psychosis and inspired creativity, ostensible antipodes of human experience, ironically also seem to be next-door neighbors."

Only it's not ironic. It's just Lyme disease.

There is another wrinkle to consider. Even if scientists succeed beyond their wildest dreams and triumphantly vanquish Lyme, there will be a societal cost, one that an opinion piece about autism[407] in *The New York Times* touched on. If we successfully eliminate autism, Maia Szalavitz writes, "we're also likely to lose a great deal of mathematical, scientific, linguistic, artistic, musical and humanitarian genius." She's right, and this concern extends far beyond the likes of Elon Musk and neurodivergency.

If Lyme has been driving this planet's creative genius, and history suggests that it has, it is also the backbone of civilization's most astonishing achievements. So many of our luminaries have muddied health histories, from autoimmune diseases to mental disorders. Would da Vinci have

become the da Vinci we revere without assistance from the bacterial infection? Would an even-keeled Vincent van Gogh still have painted his masterpieces? Would a healthy Darwin have made his incredible discovery? Would I have written this?

Doubtful. In rare cases, Lyme disease appears to be rewiring brains, opening a unique pathway for a select few to achieve the superhuman. With its extermination, there will no doubt be loss, but perhaps future scientists will eventually be able to unlock that elusive door that leads our mind to greatness.

Lessons From History

Despite overwhelming evidence that handwashing saved lives, Ignaz Semmelweis couldn't quite connect every dot to remove all doubt, allowing his discovery to languish for decades. But when a British naturalist made a discovery that threatened the very existence of God, he left no fossil unturned. To his surprise, the public swiftly embraced his theory, his controversial book selling out its first printing in a single day, becoming a bestseller. Today it remains a cornerstone of biological science. The title of this treatise, *On the Origin of Illness by Means of a Hidden Infection*, intentionally mirrors his.

Of all the historical parallels one might draw, my story uncannily resembles Darwin's—a sickly, self-educated scientist who revolutionized an entire field of study. His opus, *On the Origin of Species by Means of Natural Selection*, identified patterns and connected countless dots, just as I have. We both solved our grand mysteries using Inference to the Best Explanation. But the similarities between his life and mine run far deeper.

Darwin depended on his wife Emma for both caregiving and for painstakingly reviewing and editing all his work despite her significant misgivings. (She was aghast that her husband's evolution theory might undermine faith in God, her calling. Ironically, the hypothesis of autoimmunity does the same. Why would a benevolent God, who created such a magnificent specimen, allow it to turn on itself?) And then for years, Darwin couldn't personally promote his discoveries due to a mysterious, debilitating illness. An illness, researchers posited in a 2018 study, that looks exactly like Lyme disease.[408]

Throughout his life beginning as a young adult, Darwin was beset with unusual health issues that would come and go, leading many to call him a hypochondriac.[409] Scientists and historians have obsessively tried to diagnose his disease ever since,[410] landing on some 40 potential maladies, ranging from lupus to chronic fatigue syndrome to Crohn's disease to eczema, as well as acronyms familiar to anyone battling mysterious symptoms—PTSD, POTS, MCAS, ASD. But Lyme is the only condition that checks every box, argued researchers Erwin Kompanje and Jelle Reumer.[408] "Based on descriptions of Darwin's symptoms as found in his diaries, notebooks and letters, we conclude that Darwin suffered from a complex condition with multisystem symptoms, in which chronic (neuro)borreliosis may have played an important role." They proposed he was bitten by a tick, but his family tree,[411] assembled by researcher Dr. John Hayman, tells the full story. Many of his siblings

and maternal ancestors were also severely ill, highly suggestive evidence of the Borrelia infection being passed down for generations. He got it the old-fashioned way—from Mom.

I live the life of a disabled, possibly mad, definitely determined man tilting at the world's largest windmill. Lyme disease has ravaged me also, laying waste to my legs, leaving me unable to care for myself independently. Laura, my wife of 33 years, dresses me, helps me in the shower, prepares my meals, cuts my food, chauffeurs me to appointments, and pushes my wheelchair when needed. An active world traveler for years, today I infrequently leave home, and never without my wife. Pants have become a novelty since I cannot pull them up without assistance. If there is a silver lining, it is my hope that, like Darwin's experience, my ill health will turn out to be a boon to science and discovery.

In a show of extreme generosity, Laura has allowed me to toil on this project unimpeded, which began in the early days of the pandemic and, unsurprisingly, coincided with my soaring disability. A respected scientist with over 30 years of research experience, she has diligently reviewed all my writings despite her concerns that my findings might foster and promote the distrust of science. (Well, she has reviewed all of them except this one; I couldn't muster the courage to show her. She still is struggling with the idea that the goofy boy she met in a strip mall card shop over 37 years ago could have figured this all out. I don't blame her.) She even allowed me to rent an oversized P.O. box to handle the "impending avalanche" (my words) of fan mail; after several years, the number of letters received can still be counted on one hand.

None of my discoveries would have happened without my wife's unflagging support and commitment to a deeply incapacitated husband. None. Laura deserves accolades, and she would have justifiably earned a share of my future Nobel Prize, except for one minor problem. If awarded one for medicine, I would, respectfully, have to turn it down.

Last Words

In one of my last text exchanges with my good friend Sarah Cady, I was in full apology mode. While writing my memoir, my brain shut out distractions with such efficiency that anything unrelated was deemed superfluous. I shirked many husband duties, forgot to pay estimated taxes (once), and had to be reminded to eat. One year I even sort of forgot Christmas, which did not go over well with my wife. Anyway, I had been tasked with purchasing a gift for Sarah Cady's baby shower—a puzzle step stool customized with her son-to-be's name, Bode. When the stool arrived, I got a curious text from her about how the company had inexplicably misspelled his name. "What arrived says Body!" I didn't need the added facepalm emoji to know what had happened. "Save the Body stool," I implored, embarrassed. "It will make a hell of a story later about your absent-minded friend who won the Nobel." She gave it a thumbs up in approval.

Sarah Cady will never know that I fell short of that goal; I'm unlikely to win a Nobel Prize in medicine for this discovery. The Nobel Prize is given to a maximum of three recipients in any given field. Three other research teams properly deserve the recognition, accolades, and prize money: those involved in finding that Borrelia burgdorferi spirochetes are one of the only known lifeforms

not to require iron, those that discovered that they instead required prodigious amounts of manganese, and the team that found that too much manganese is toxic to the virulent bacteria, a discovery I predict will be the cornerstone of a cure. I would not have made my discoveries, the most critical ones, without their research. If I am going to win a Nobel, I am committed to ensuring it won't come at their expense.

(I also won't win XPRIZE Healthspan,[412] a $101 million global competition on longevity, to restore muscle, cognitive, and immune function by 10 years or more. I didn't apply. While recent research[413] unsurprisingly has found "robust and significant deceleration in longevity gains," it's actually worse than that. A study released in March of 2026[414] found "particularly alarming mortality patterns among those born after 1970," especially in cancer, concluding that, "examining the pattern in its totality reveals more of a systemic failure, for which there is no single explanation." The sure-to-be-shocking realization that this downward trend is being caused by a treatable bacterial infection could easily end up adding a decade or more to lifespans. Perhaps the organizers will feel inspired to honor the spirit of the contest.)

For humanity to crawl out of this Lyme disease abyss, it will require the world to come together—backed by brave, uncompromising leaders and bold researchers from all countries—to step up to meet the health challenge now threatening civilization. This is going to be an otherworldly, monumental challenge. About 90% of the annual $5.3 trillion healthcare spent in the U.S. is attributed to managing and treating chronic diseases and mental health conditions. Nearly $5 trillion. Annually. In one country. Ostensibly to treat Lyme.

It now is a race against time. For families struggling with chronic illness, cancer, and the litany of diseases caused by Lyme, every moment counts. Each day, more than 110,000 people die worldwide[415] from these chronic health issues, the equivalent of a midsized city vanishing *daily*. To those who have lost loved ones—reading the obituaries in my local paper is a painful reminder of the carnage this disease is inflicting—I am so sorry I wasn't able to deliver this sooner. To those in the throes of a health battle caused by this infection, know that there is hope.

Our youngest generations, Gen Z and Generation Alpha, will be the most impacted by Lyme disease, the warning signs already everywhere. "The number of college students reporting disabilities rose more than 50 percent over the last decade," reported *The New York Times* in March of 2026.[416] These young adults are not overly sensitive, fragile hypochondriacs (sheltered, of course, by excessively protective parents) intent on gaming the system. They are genuinely sick, suffering from an infection—and they will be the most driven to fully eradicate it.

As for my personal health, I am optimistic that I will recover, putting my faith in karma—and perhaps in something bigger. Five years ago, I was at best agnostic, but today? The inordinate number of coincidences required for me to make my discoveries have led to a new level of openness. So, perhaps I should pay attention when the universe speaks, which is frighteningly often in my world.

When I met Laura in the summer of 1988, I had one cassette tape that I played on our dates over and over in my powder blue Plymouth K-car: UB40's Red, Red Wine. It became our song, one that we awkwardly danced to at our wedding. Red wine, with its tannins, helps with Lyme disease. Was our song fate, destiny, written in the stars?

That's one mystery that will remain unsolved, unlike everything I've discovered over the last five years, spilled onto these pages. The origin of illness has been found, and science will back that up. So will the most advanced artificial intelligence.

I've made this free to read on Substack (davebexfield.substack.com) and a convenient PDF for a reason. It's easy to share with friends, family, colleagues, and online communities. It's easy to evaluate with clickable links for scientists and researchers. And whether you are a medical expert, a concerned parent, a frustrated patient, or an inquisitive fifth grader, it's also easy to vet my discoveries with AI. (To do so effectively, I've published a separate companion document—How to Evaluate *On the Origin of Illness*.)

But it won't just be AI and researchers grappling with this information. *On the Origin of Illness* will get into the hands of countless businesses in position to lose billions of dollars because of this stealthy infection, industries desperate to guard assets, and they will not wait for science to slowly self-correct. The food and beverage industries shouldering excessive blame. Sports teams losing star players to injuries, from back spasms to wonky knees, that may not be strictly physical. Vaccine manufacturers wrongly held responsible for adverse reactions that are, in most cases, the fingerprints of a dormant bacterial infection reawakened by immune disruption—not the vaccines themselves. This will also find its way to law firms defending clients who may have committed offenses due to altered thinking beyond their control. To advocates trying to protect the most vulnerable. To families seeking answers for intractable health problems.

Society may not be ready for what this means. The scientists who received this are still processing it. That processing will take time—as well as unprecedented coordination, intense research, and copious clinical trials. But here is what gives me confidence: we've done this before. Covid proved that when scientists finally know what they're fighting, they move with extraordinary speed. That is, if they can accept what the evidence has been pointing to all along. That it has been Lyme disease.

Right now, I expect two emotions are colliding with each other simultaneously—joy and horror. The feeling of being overjoyed at finally realizing what has been behind the unsolved health issues of humankind. And the sense of being horrified at the tragic consequences of not realizing this sooner. Every misdiagnosis. Every failed treatment. Every patient told it was in their head. Every researcher who spent a career looking in the wrong direction.

Like the German word *schadenfreude*, there isn't a word in English for what so many of us must now be feeling, so I created one: joyrified.

As hard as this is to accept, virtually all of us are infected with Borrelia. The bacteria are either dormant—for now—or active. Either way, the following interventions hold the most promise. They

might not fully eradicate the infection, but they are the best-available treatments for keeping our Lyme disease at bay and, critically, they buy us valuable time. Please discuss these interventions with your medical provider.

- **GLP-1s**. These drugs appear to be the best available medications[417] (even in patients deemed "non-responders"[418] in terms of weight loss) to effectively tamp down Lyme, but they are expensive, supply is limited, and you likely will need to stay on them or the benefits can vanish quickly.[419] Even so, the evidence is overwhelming and growing. This treatment prevents dormant Lyme disease from getting a foothold and will extend lifespans and broadly benefit quality of life. For those with active disease, it quiets Lyme better than any existing option, often reversing symptoms. Any weight loss is a bonus. Financial considerations aside, the case for GLP-1s extends well beyond the visibly sick. If these drugs dramatically reduce the risk of autoimmune disease, cancer and myriad other chronic illnesses, the question isn't why a person would take one—it's why they wouldn't. (Clinicians are already asking that question: a June 2026 CBC investigation[420] posed it directly, even as mainstream medicine hasn't yet caught up.)
- **Metformin**. For many, the diabetes drug metformin,[421] first synthesized over a century ago and widely available for the past 30 years, is a compelling alternative. It's considered generally safe, extremely inexpensive (governments should consider an over-the-counter option), and has a wealth of unexpected health benefits,[422] including slowing brain aging,[423] improving lifespan, aiding cancer recovery,[424] and even reducing the risk of long Covid. Recent research suggests that combining it with GLP-1s might supercharge their benefits[425] without increasing adverse reactions.
- **Antibiotics**. Tetracyclines, particularly doxycycline, are standard treatments for Lyme, but there are many caveats. They have questionable success with entrenched disease, can trigger side effects (particularly Herxheimer reactions), and could contribute to antibiotic resistance. Until more data is available on optimal protocols for entrenched disease, these are most reliably effective in acute cases.
- **Carbohydrate-limiting diets**. While a true medical ketogenic diet holds the most promise for health gains,[426] it is hard to do properly without professional assistance and is onerous to maintain. That said, any diet that limits carbohydrates will help, from Atkins to anti-inflammatory; a June 2026 study out of Sweden[427] found that anti-inflammatory diets were particularly associated with lower dementia risk. So will reducing or eliminating alcohol, sugars, and high-carb ultra-processed foods. The case for cutting sugars specifically keeps growing: a July 2026 study[428] found that fructose signals ovarian cancer cells to spread, with researchers suggesting the effect likely extends to pancreatic, colon, and liver cancers as well.
- **Manipulating manganese**. Denying Borrelia its manganese through food and beverages is a sound, scientifically backed approach, and forms the backbone of the Mediterranean diet. A number of common substances effectively block the absorption of the trace mineral: iron (specifically non-heme iron), fiber (vegetables, legumes, seeds and nuts, grains, and berries), calcium (dairy products especially—a randomized trial found that three daily

servings of full-fat dairy raised neither body weight nor cholesterol[429]), and tannins. Coffee is most reliable in that area. The American Heart Association's July 2026 scientific statement[430] concluded that 3–5 cups daily is not only safe, but also lowers the risk of heart disease, stroke, heart failure, hypertension, and type 2 diabetes. Its benefits extend further still, from boosting liver health to lowering the risk of Parkinson's.[431] Meanwhile, trying to overload with manganese is best left to researchers.

- **Antihistamines.** Over-the-counter antihistamines—particularly second-generation options like loratadine (Claritin) and desloratadine (Clarinex)—have shown unexpected benefits across a range of conditions[432] far beyond allergies, including improved survival across ten immune-related cancer types. Famotidine (Pepcid), an antihistamine available for under $10, has shown measurable benefit in IBS patients and was found to blunt cytokine storm in severe Covid.[433] These are among the safest, most accessible, and least expensive interventions available—and their reach should span across conditions.
- **Exercise.** Exercise can lower cancer risk, reduce inflammation and improve immune system function[434] as well as lower blood sugar.[435] That last point is key—it's doing the same work as GLP-1s, diabetes drugs, and carb-reducing diets: robbing Borrelia of its fuel. Resistance training in particular appears to starve neurological disease at the source—a 30-year Harvard study of nearly 150,000 adults found that 90 to 119 minutes of weekly weight training was linked to a 27 percent reduction in dying from neurological conditions[436] like Alzheimer's, the largest mortality benefit of any disease category studied.
- **Lifestyle**. Pay attention to the biggies. Lowering stress and avoiding illnesses reduce the risk of disease reactivation. And getting ample sleep is important.[437]

Laura and I have been lucky enough to crisscross the world, exploring the breadth of human culture. In each of the more than 50 countries we have visited, our experiences have been strikingly similar. From Vietnam to Bhutan, from South Africa to Venezuela, from New Zealand to India, we are all connected, our futures intertwined. I made a promise to my wife that I would try my best to recover, that one day I would be back as her husband, whole. For those unfamiliar with my history, I keep my promises.

I've reached the apex of what I can do. I've laid out everything I can—the unlikely connectedness of it all.

Da Vinci was right all along.

Appendix A — Additional Hallelujah Booyahs

While writing *On the Origin of Illness*, I've discovered more patterns that strongly, overwhelmingly, suggest Borrelia's involvement. I call these revelations, when all the dots connect, hallelujah booyahs. Here are four additional HBs with expanded breakdowns and discussion.

Lower Back Pain

When *The New York Times* published a piece in March 2025[266] on the challenges of treating acute and chronic back pain, I perked up. Lyme disease is synonymous with lower back pain (LBP). Could the disease be contributing to caseloads?

I had long assumed back pain was the result of a physical injury, of not using proper technique when lifting heavy items. But as I replayed my own experience with these injuries, and listening to stories of other people who had inadvertently tossed out their backs, forgetting to "lift with your legs" was not a thing. People weren't throwing out their backs improperly moving pianos on the weekend, they were picking up dropped pencils or reaching for toilet paper. I started looking for patterns that might suggest Lyme. It didn't take long.

The roster of people who have suffered from chronic, debilitating back pain alongside a constellation of other unexplained health conditions is not a short one. JFK was awash with debilitating health problems since his youth[438]—Addison's disease, colitis, osteoporosis, chronic infections, and agonizing lower back pain—his family joking that "if a mosquito bit Jack Kennedy, the mosquito would die, because apparently there was something in poor Jack Kennedy's bloodstream." Mario Lemieux went in for a routine checkup alongside his chronic back problems[439] and walked out with a cancer diagnosis—Hodgkin's lymphoma at 27, later followed by cardiac arrhythmia that ended his career, all health issues with Lyme's signature involvement. FDR's paralysis—long attributed to polio, then to Guillain-Barré syndrome—began with lower back pain and fever,[440] symptoms that neither diagnosis fully explains, but that fit Lyme disease with uncomfortable precision. The pattern repeats across history with a reliability that is difficult to dismiss as coincidence.

My best friend Scott, whose sharp academic mind has been forced to absorb many of my theories throughout this journey, survived bladder cancer a few years ago, a cancer I've connected to Lyme. Recently he has been beset with lower back pain, family members have fallen ill. Sometimes his own intelligence gets in the way—logically, it couldn't possibly all be connected, his brain reasons—so even he remains skeptical.

Now for the heavy lifting.

Is lower back pain widespread? Yes, as reported in *The Lancet* in 2023,[441] LBP is considered a global epidemic, with 619 million suffering from the condition in 2020, a rate that's predicted to near 850 million, nearly 10% of the global population, by 2050.

Have cases been steadily increasing inexplicably? Yes, says the International Association for the Study of Pain.[442] "There have been increases in both the number of people living with LBP and the prevalence of LBP in all age groups from 1990 to 2017." Today it is the leading cause of disability worldwide for all ages.

Is it largely unexplained? Yes, confirms the WHO,[267] 90% of cases have no explanation, "when it isn't possible to identify a specific disease or structural reason to explain the pain."

Are women more affected? Yes, found a 2020 study,[443] and the split is large: 61% to 39%. Another study confirmed[444] that "globally, women bear a higher burden of LBP than men, with middle-aged populations experiencing the heaviest burden."

Is LBP frustratingly resistant to treatment? Yes, the *Times* highlighted a study that looked at 56 non-surgical interventions. With the exception of anti-inflammatories, which helped modestly with pain, all of them generally failed to provide much relief. Indeed, LBP is so widespread and hard to treat that it has predictably spawned an entire new field of medical practitioners: chiropractors. It's no surprise that they share with Lyme doctors unconventional, often controversial methods to treat their patients.

But do antibiotics, GLP-1s, or diets help? Yes (with an asterisk[445]). While "anecdotal reports and clinical experience from obesity specialists often include notable back pain relief as one of the first changes patients notice within the first few months of GLP-1 therapy" and Reddit is awash[446] in the success of ketogenic diets stunningly relieving years-old back pain, the clinical evidence is catching up. Although not an LBP study, a randomized feeding trial in knee osteoarthritis patients[447] found pain severity dropped significantly on a Mediterranean diet compared with both low-fat and regular diets—with no significant difference in weight loss between groups, suggesting the diet's anti-inflammatory components, not the pounds shed, drove the relief. Nor is it alone: the randomized STEP-9 trial[448] found that semaglutide reduced joint pain scores by 41.7 points versus 27.5 for placebo—a difference researchers noted was partly independent of weight loss itself, again suggesting a direct anti-inflammatory mechanism at work.

Even so, formal investigations into GLP-1s, antibiotics and diets specifically for LBP remain conspicuously absent. Why the oversight? If LBP is believed to be caused by an injury, not a bacterial infection, such interventions shouldn't influence LBP. So why in the world would one research them? A few well-crafted studies will right that wrong.

And Lyme disease isn't just driving pain in the lower back; its fingerprints are all over the chronic pain epidemic disproportionately affecting women. My first symptom of Lyme disease interrupted a 2005 adventure in Cambodia's Angkor Wat, my right wrist howling in unexplained pain, eventually leading to an MS diagnosis. Until I started treating the infection, every few years my wrist pain would flair (or my toe, or my knee, or my back, or my shoulder). I suspect the same is true for many of the 1.71 billion people worldwide living with musculoskeletal conditions—the single largest contributor to years lived with disability on the planet.[449] In 2019 alone in the U.S., 39% of adults reported back pain, 36.5% lower limb pain, and 30.7% upper limb pain.[450] These are

not rare conditions. And they are persistent. Only 60% percent of shoulder pain resolves after a year[451]—frozen shoulder, anyone?—and "surgery is not superior to conservative treatment options in the long-term."[452] These "injuries" are the background noise of modern human existence, so ubiquitous that we have stopped asking why, our brains conjuring plausible reasons. An old sports mishap, too much time at the keyboard, awkwardly reaching for a shot playing pickleball or the TV remote control—always tweaking something doing something physical.

Just look closer at carpal tunnel syndrome. Wrist pain broadly affects around 10% of the general population,[453] and the strongest predictor of disabling wrist pain, researchers found, isn't what you do with your wrists—it's how many other places in your body hurt.[454] That means it's systemic, not mechanical. CTS specifically constitutes 90% of all neuropathy cases[455] and affects 3 to 6% of adults. The gender gap is not subtle: when examining prevalence specifically, women come in at 7–9% compared to just 0.6% in men,[456] a gap of more than ten to one. Medicine has already catalogued what causes CTS, and the list reads like a Lyme disease index. Its official list of etiologies[455]—from most to least common—includes obesity, arthritis, hypothyroidism, diabetes, amyloidosis, sarcoidosis, multiple myeloma, and leukemia. For a wrist "injury" usually blamed on overuse. (Often plagued with wrist issues,[457] professional tennis players—including Carlos Alcaraz, 23, one of the world's greatest and currently sidelined with wrist inflammation—should be taking notes.)

1.71 billion people. Rising with every generation. A gender gap that never closes. An autoimmune thread that keeps appearing in the research whether or not anyone is looking for it. This is not wear and tear. Wear and tear doesn't respond to diabetes drugs. There's a reason GLP-1s are reducing pain across the board—knees, hips, backs, joints of all kinds. Weight loss doesn't explain it. The pain relief arrives faster, broader, and deeper than shedding pounds alone would predict. This is a disease—and it is the same disease.

Allergies

Meanwhile, four months after the *NYT* piece published on lower back pain, a headline in *The Washington Post*[269] made me do a double take. "Allergies seem nearly impossible to avoid—unless you're Amish." Amish communities check the requisite boxes of Blue Zones: isolated and insular. These attributes would severely limit the sexual spread of Lyme, at least for a time. The Amish studied in this article hailed from northeastern Indiana, not known for being overly tick infested, although it is a growing concern in the state. Did I just trip onto an HB?

When I was younger, watery eyes and a runny nose led my mom to accuse me of doing drugs in high school. Like throwing out your back, I had always assumed that allergies were just a normal part of life. Researchers, meanwhile, hypothesize that this type of immune hypersensitivity, an overreaction to harmless substances,[458] evolved from the body's struggle to fight off parasitic worms and other pathogens of the past.

Recall the Tsimane, the Indigenous group in Bolivia. They don't get autoimmune diseases, and they don't have any allergies[459] (and good luck trying to find any autoantibodies). Makes sense. Autoimmunity, a telltale sign of Lyme's involvement, and allergies are so closely tied together that research suggests the relationship to be bidirectional. If you have allergies, the risk substantially rises that you'll develop an autoimmune disease and vice versa. "Their incidence has constantly increased in the last decades, and their co-occurrence defies current standards in patient care," found a study published in February 2025.[460] Researchers excitedly reported that "growing evidence suggests that these conditions not only share some common inciting triggers but also are subtended by overlapping pathogenic pathways."

Nearly a third of U.S. adults and children suffer from at least one allergy, according to the CDC.[461] It's bad and getting worse. A study published in January of 2025[462] had already sounded the alarm, noting dramatic increases in rhinitis, asthma, and allergies to food and drugs. "Globally, the prevalence of allergies is rising… The precise mechanisms underlying this rapid increase in prevalence are unknown."

Also unknown to researchers, an explanation for the confounding gender ratio. A 2010 study[463] found that "only in patients below the age of 15 years, allergies are more frequently diagnosed in males. … At a later stage in life, female adolescents clearly suffer more often from respiratory allergies and asthma. Moreover, there has been observed a 60:40 ratio for female to male patients of severe food allergy." This is the exact pattern exhibited by Lyme disease. If the gender disparity were to be revisited today, the gulf between women and men should be even larger, which 2024 research, a study of patients seeking allergy testing, supports:[464] 72% female to 28% male. Finally, as expected, our now familiar trio of treatment interventions has shown significant promise in limited studies, from the "multiple anti-allergy effects"[465] of doxycycline to GLP-1s' potential to treat "chronic airway inflammation in asthma."[466]

The implications extend further still. The allergy medication sold as Clarinex (desloratadine) has been associated with improved survival[432] across ten immune-related cancer types in a nationwide Swedish study of nearly 430,000 patients, including melanoma, breast, gastric, colorectal, pancreatic, lung, prostate, kidney, bladder cancer, and Hodgkin lymphoma—but not in non-immunogenic tumors. A separate study drilling into the lung cancer finding discovered why: loratadine, the active ingredient in Claritin, triggers cancer cell death[467] by deactivating key inflammatory signaling pathways, an effect entirely unrelated to its original purpose of blocking histamine. A 2025 Hong Kong study of cancer patients receiving immunotherapy found the same pattern: H1 antihistamines were associated with improved overall survival[468] in lung and skin cancers.

On the surface, none of this makes sense. A drug for allergies has no business showing up in cancer survival data. Under this theory, it makes perfect sense. Targeted anti-inflammatory drugs—antihistamines fall into that class—keep showing up across every condition Lyme drives because they're all fighting the same fire.

The pattern even reaches the medicine cabinet's humblest residents. Another close friend of mine—someone whip smart and not easily swayed by this amateur scientist—has been struggling recently with health issues; her doctor's best guess was IBS and had no suggestions for treatment. Frustrated, she took it upon herself to track down something that might help and landed on allergy medications. She had stumbled onto a rare over-the-counter treatment for Lyme.

Clinical trials have found[469] that antihistamines, including Pepcid (famotidine), show measurable benefit in IBS patients. The same $10 heartburn drug was found in a randomized controlled trial to blunt the cytokine storm[433] seen in severe Covid and a separate RCT confirmed[470] it also reduced inflammation and improved recovery in outpatient Covid,[470] broadening the claim beyond severe disease. A drug for acid reflux, helping IBS, reducing Covid severity, and sitting next to Claritin on the pharmacy shelf. If these conditions had nothing to do with each other, none of this would make any sense. They do, and it does.

The reach extends further still. Antihistamines are a first-line treatment for acute vertigo[471]—outperforming benzodiazepines (standard-of-care sedatives like Valium and Xanax) in a meta-analysis of 17 trials—because histamine directly regulates the vestibular system, the same system Lyme disease is known to disrupt. Hydroxyzine, an antihistamine, is FDA-approved for anxiety[472] and used as an alternative to benzodiazepines for panic attacks and insomnia. And here is perhaps the most revealing detail of all: the first antidepressants grew directly out of antihistamine research,[473] making this drug class the accidental founding father of modern psychiatry. Accidental, that is, under the prevailing model. Under this one, it makes perfect sense.

A drug class designed to reduce drippy noses turns out to touch the inner ear, the brain, the gut, the immune system, and cancer survival. The more conditions you look at, the more antihistamines keep showing up—for the same reason everything else in this document keeps showing up.

PMS and Menopause

Other conditions have also grabbed my attention. Of those, premenstrual syndrome (PMS) is particularly intriguing. The typically transient emotional and physical symptoms—from depression and cognitive issues to fatigue and joint pain—are unmistakable characteristics of Lyme. The act of losing blood is not an immune system disruptor in and of itself, but the wild hormone swings that occur before menstruation are. Progesterone levels in the luteal phase rise sharply after ovulation, before plummeting if there is no pregnancy. This coincides with the timing of health risks in adolescent athletes,[474] who experience a wide range of medical issues during the late luteal timeframe, including physical injuries. It's gotten so bad that female professional athletes are suffering debilitating injuries and muscle tears at an unprecedented rate,[475] puzzling doctors. But there is something else occurring.

During this luteal phase, manganese plays a crucial role in the corpus luteum,[476] the temporary endocrine structure that produces progesterone. Sure enough, manganese superoxide dismutase

(MnSOD), which regulates oxidative stress, expression is low in the mid-luteal phase before increasing significantly[477] during the late luteal phase and luteal regression. This is key, as our holy grail researchers (Aguirre JD, et al., 2013[33]) found, critically, that "high manganese is necessary to activate the SodA superoxide dismutase (SOD) essential for virulence." Both deserve further investigation. I am suspicious of the hormone spikes and valleys, but the striking MnSOD connection is hard to ignore. Is PMS merely a mini Lyme relapse? Signs point to yes.

Women taking birth control pills bleed less and their progesterone levels remain mostly stable. That means they are absorbing less manganese than their peers, with one less immune-upending monthly trigger to awaken dormant Lyme. That also means that being on the pill should offer some modest health protection. True of every test I've thrown at my manganese theory, it does, lowering lifetime risk of ovarian and endometrial cancer by roughly 50%[478]—protection that persists for up to 35 years after women stop taking the pill. What else helps? In one randomized, placebo-controlled trial,[479] the doxycycline group "showed a highly significant reduction of symptoms," while "subsequent antibiotic treatment of the original placebo group similarly diminished" symptoms. GLP-1s and diets boast benefits in PMS as well. A 2024 systematic review of randomized trials[480] found that low adherence to a Mediterranean diet was linked to greater PMS risk, though the review itself notes formal trials in this space remain scarce, the same gap that dogs LBP research.

Interestingly, the stifling symptoms of menopause,[481] from joint pain to migraines, also follow a strikingly similar pattern that strongly suggests Lyme is goosing this natural aging process. Just as in PMS, symptoms have been steadily increasing with each generation. A 2022 Swedish study[482] tracked menopause across four generations and came away thoroughly flummoxed. "In this prospective longitudinal study of 50-year-old women, we found nearly twice as high odds of reporting daily hot flashes in the later-born women compared with earlier-born. When controlling for potential predictors, there was still an obvious difference, which cannot be explained in our study."

Weight gain during menopause follows the same generational escalation. At least 50% of women report significant weight gain at or near menopause,[483] an average of 12 pounds within eight years of onset.[484] But it's getting worse: 70% of perimenopausal women now meet criteria for overweight or obesity,[485] compared to 56% of all adults in 1988. Most striking is what researchers call weight loss resistance[486]—the inability to lose weight despite caloric deficits and exercise, peaking in postmenopausal women—as if something is actively working against the body's attempts to shed the excess. Under the prevailing model, that something is hormones and aging. Under this one, it is the infection using the hormonal disruption of menopause as cover to accelerate its fuel consumption, and GLP-1s, by starving that fuel source, are already showing results in menopausal weight management[487] that lifestyle interventions alone cannot match.

Other reliable markers line up seamlessly, from the rise of mental illness[488] issues to GLP-1s tamping down symptoms,[489] and doxycycline as well. The mystery of why allergy medications seem

to help with menopause symptoms as well as premenstrual ones, reported in May 2026 in *The New York Times*,[490] might not be such a mystery after all. They all, predictably, appear to be connected.

Sexual Orientation

The pattern had been building for years, but I hadn't paid much attention until it started showing up regularly in Christmas newsletters and at catch-up lunches with friends. Sons and daughters, most now young adults, were coming out as gay or lesbian. Some were transitioning or announcing their bisexuality, their names and pronouns often changing. At first, I was inspired by their courage and conviction to follow their hearts, but soon the sheer volume raised questions. As a Gen Xer, when I was younger, people weren't remotely this gender fluid. Is it just changing times and rising acceptance, or is it something else? I discovered it's something else.

In February of 2025, *The New York Times*[274] reported that nearly 10% of U.S. adults now identify as LGBTQ, a rate that defies the traditional trappings of evolution, tripling since 2012. And it is clearly generational, the identical pattern of Lyme: Generation Z 23%, Millennials 14%, Generation X 5%, Baby Boomers 3%, Silent Generation 2%. If it was merely people coming out more due to societal acceptance, the leading theory, all ages and both sexes should have seen largely equal increases in the Gallup survey, but no.

"The increases have been driven by young people," reports the article. "Nearly one-quarter of adults in Generation Z, defined by Gallup as those 18 to 27, identify as L.G.B.T.Q." with more than half of this group identifying as bisexual. Then the other shoe dropped: Women are driving these increases at a revealing 3-to-1 clip, most noticeably in bisexuality. This gulf between sexes is consistent with a Borrelia infection; spirochetes appear to be affecting the area of the body that determines gender. But medicine's holy grail—the ultimate box out, the final arbiter—leads us to so much more.

Let's start by examining the stark gender difference in bisexuals. While men and women identify as gay or lesbian (or transgender) at similar rates, suggesting sexual preferences in these populations were determined before birth, women are significantly more likely to identify as bisexual than men. This means that flipping this particular gender switch is occurring after girls begin menstruating, a revealing sign of Lyme's involvement. Most in the LGBTQ+ community report first realizing their gender identity in their mid-teens, with 72% knowing by age 18. A full quarter, however, are in the dark until adulthood, many in their 30s and 40s. A majority are women.

If Lyme disease is upending gender identity, it stands to reason that it must also be upending health. If everyone in the LGBTQ+ community is afflicted with at least a somewhat active form of Lyme, they are going to be noticeably sicker than heterosexuals. And they are, by every metric. Cancer: "LGBTQ+ people face a disproportionate burden of cancer,[491] with both a higher incidence of cancer and later-stage diagnoses." Heart problems: "Lesbian and bisexual women have lower cardiovascular health scores[492] than their heterosexual counterparts." Long Covid:[493] "Nearly 24% of all [bisexual and] transgender adults report that they have experienced long Covid … the highest

percentages in the country." Mental health issues:[494] "Those who are LGBTQ are nearly three times more likely [than heterosexuals] to develop a mental health disorder [and] they are also significantly more likely to attempt suicide and abuse substances." And death.

A comprehensive 2024 study published in *JAMA*[495] strongly suggests the devastation Borrelia can rain down. "Lesbian, gay, and bisexual women died 26% earlier than heterosexual women in a longitudinal cohort of nurses followed up for 3 decades." Curiously, though, researchers hypothesized that the egregious mortality in this community was caused chiefly by discrimination, which causes stress, which leads to making poor health choices. I don't buy it, and neither does the science.

One notable cohort study found that, "lesbian and bisexual women reported higher diet quality[496] than heterosexuals." They care about their health. The death gap is big, far too big, to pin entirely on discrimination, which doesn't typically lead to higher incidences of breast cancer, heart disease, chronic illnesses, long Covid and mental disorders. A study out of the University of Washington[497] confirms the health risk women face in these communities, and adds a twist. "In general, lesbian, gay and bisexual (LGB) older adults were found to be in poorer health than heterosexuals, specifically in terms of higher rates of cardiovascular disease, weakened immune system and low back or neck pain."

It all tracks, and at least now we can remove the mystery of lower back pain, but neck pain? That's perhaps most revealing. The neck harbors the thyroid, which is of significant importance in this population. For years, researchers have detected a link between thyroid dysfunction and same sex attraction. "There is evidence that thyroid gland plays a crucial and decisive role in determining sexual orientation in people," reported a 2015 study.[498] A large examination of 4.4 million Danes in other research found that "the risk of autoimmune thyroid dysfunction was increased,[499] notably Hashimoto's thyroiditis and Graves' disease." This bump in thyroid dysfunction has been consistent and widespread in the LGBTQ+ community.

Is manganese involved? Of course it is. A 2007 study[500] warned that manganese "may alter the regulatory function of thyroid hormone, [and that it] may affect a plethora of other neurotransmitters, in turn, causing dysregulation of thyroid hormone homeostasis." Further study was urged. Researchers aimed to plug that gap with a study published in July of 2025: Critical review of the association between environmental manganese and thyroid function, with implications for potential neurodevelopmental effects.[501] "Toxicokinetic data demonstrated that Mn accumulates in thyroid tissue and might interfere with thyroid function," they discovered, but what did that mean? A total of 31 studies were examined leading to no more clarity, lamented researchers. "Currently, there are limited data on biological mechanisms of action for the effects of Mn on the thyroid."

Suggestive evidence of Borrelia's influence on sexual orientation even bleeds from research on the communities least likely to be overrun by the infection: Blue Zones and Indigenous populations like the Tsimane. In stark contrast to the rest of the world, people identifying as LGBTQ in these areas are virtually nonexistent. That extends to supercentenarians. Scientists reflexively point to

generational norms or societal taboos, but what if it's not that at all? Although homosexuality has been called a Darwinian paradox,[502] that's not entirely clear. And despite new research that suggests there may be an "evolutionary upside"[503] to same-sex relationships, it in no way accounts for the dramatic increase in recent generations or the striking gender gulf in bisexuals.

Gay or straight, we are all sick with a bacterial infection, the only difference being whether the infection is active or dormant. Bisexuals, who typically develop feelings for the same sex after puberty, would then be at higher risk of having an active infection compared to others in the community whose sexuality is determined much earlier. And they are, in a huge way. Bisexual women die 37% sooner than heterosexual women,[275] a staggering statistic.

A disturbing question in the LGBTQ+ community now looms. How will treatment affect their sexuality?

I don't know. Over time, it would not be surprising if observational GLP-1 studies show a notable decrease in those who will later identify as bisexual. But until these drugs are deemed safe for pregnant women, those identifying as gay, lesbian, or transgender should remain largely unchanged. Critically, I *don't* see clear evidence of these drugs changing existing sexual persuasion. Regardless, this is a deeply complex issue with profound ramifications for relationships, both current and future.

One thing, though, is certain. People identifying as LGBTQ+ are just ordinary people who struggle with life's challenges—and then some—just like the rest of us, whose sexuality appears to have been shaped, at least in part, by this disease. That is no different from a breast cancer diagnosis, an autoimmune condition, a mental disorder, or seasonal allergies. It is simply biology.

Lastly, "ordinary" might be underselling this community. Greatness often follows. Da Vinci, Michelangelo, Shakespeare, Socrates, Whitman, Dickinson, Woolf, Eleanor Roosevelt and so many others were gay (or believed to have been gay). It's no surprise that today's world is bursting with LGBTQ+ talent in all industries. As much as Lyme disease has taken from us, on rare occasions it deserves a thank you.

Appendix B — Summary of the Treatise in 9 Facts

The following is a summary of the major findings in On the Origin of Illness by Means of a Hidden Infection*, presented in nine facts.*

Scientists have been trying to solve the towering medical mysteries of today by piecemeal, using ever-advancing technology to analyze and parse illness, seeking out the smallest of clues to accelerate diagnosis, improve care, and better direct the treatments of tomorrow. This evidence-based approach appears reasonable and sound. But appearances can deceive. It is doomed to fail, and artificial intelligence will be powerless to help.

The evidence scientists are seeking is instead buried in over a century of research, a treasure trove of data that, once exposed and reinterpreted, will reveal the missteps, the miscalculations, and the grave mistakes made over the years. This realization will simultaneously be both glorious and horrifying. Nine facts, when taken together, fully collapse the foundational pillars of modern medicine, rewriting everything we understand about human health.

FACT #1: LYME DISEASE MUST BE SPREADING WITHOUT THE AID OF TICKS

The unraveling of modern medicine began innocently. A 2022 meta-analysis in BMJ Global Health—spanning 28 countries, six continents, and 158,287 subjects—found that 14.5% of the global population carries Borrelia burgdorferi antibodies, implying over one billion people have evidence of active or recent infection. For perspective, the CDC estimates that approximately 476,000 people are diagnosed and treated for Lyme disease each year in the United States. There's a gobsmacking 2,311-fold difference between the two figures. That gap is not a rounding error. It is a diagnostic catastrophe.

The prevailing explanation—that Lyme is confined to tick habitat—fails under scrutiny. The study documented antibody-positive individuals with no plausible tick exposure. Even if testing for Lyme is unreliable on a massive scale, the delta between estimated cases and positive seroprevalence is too vast—the disease must be spreading without the aid of ticks. But if it's spreading, where are all the cases? Lyme disease is one of the most destructive infections in human history; it cannot be hiding.

That question was inadvertently addressed 70 years ago.

The cascade of symptoms caused by the bacterial infection tellingly resemble the symptoms found in every autoimmune disease—the fatigue, the pain, the mental challenges, the neurological issues, all of it. The very concept of the body attacking itself is a cascade of contradictions, defying Darwin. Instead of rates falling, they've been increasing steadily with every recent generation. Instead of symptoms fading, inflammation and disability rage. Mental illness, metabolic disorders, and cancer are frequent partners. Autoimmunity had appeared to be an unsolvable mystery of the ages, destined to vex scientists for eternity. Except. This leads us to our second uncomfortable fact.

FACT #2: THE AUTOIMMUNE HYPOTHESIS DEFIES DARWIN AND LOGIC

In the 1950s, when frustrated scientists kept finding unusual antibodies accompanying a swath of diseases—yet never finding an attacker—they came to the fateful conclusion that the immune system must be turning on itself. Fateful, because there is a telling maxim in science: Absence of evidence is not evidence of absence. Even though researchers had found spirochetes in autoimmune diseases like MS, those discoveries were dismissed as laboratory errors, "scratches in the glass" on microscope slides.

Autoimmunity, indeed all chronic diseases, share a curiously consistent pattern. When it comes to suspected causes, it's always a mysterious combination of genetics, the environment, lifestyle, and, when all else fails to explain it, bad luck. When siblings fall ill, that's genetic. When both partners experience illnesses, that's environment. If weight gain is involved, that's lifestyle. And if nothing else fits, someone just drew the short straw. There's a far cleaner, more rational, and eminently more coherent reason.

Epidemiological evidence, when viewed from the perspective of an infection, is robust and clear, present in countless studies. The reason why spousal concordance in chronic disease rivals sibling concordance? That's the distinctive pattern of a disease spreading sexually and vertically (mother to unborn child). Although thought to be disseminated solely by blacklegged ticks, that's merely how Lyme was discovered. Now we are in an unexpected perfect storm.

The ancient parasitic disease had remained modestly contained for millennia until the 1950s, when its spread accelerated dramatically due to three major factors: human migration after WWII, the Baby Boom, and the rising popularity of international air travel. This trifecta of events erased any possible chance of containing the disease, ensuring that every subsequent generation is met with a stark, predictable rise of chronic illness, which is exactly what is unfolding. Today's primary suspects—ultra-processed food, environmental toxins, and poor lifestyle choices—are all red herrings. There are not dozens and dozens of causes for these diseases, which brings us to our third incontrovertible fact.

FACT #3: THE REMARKABLE CONCORDANCE IN ILLNESSES CAN ONLY BE EXPLAINED BY A SINGLE COMMON CAUSE

Autoimmunity is one of the foundational pillars of modern medicine, supporting countless other theories. If it collapses, it would immediately throw into question the origin of every health condition it is closely tied to. The published scientific studies, hundreds of thousands of studies over the past 50 years, leave no doubt as to the enormous ramifications.

Virtually all major illnesses have an unmistakable bidirectional relationship with autoimmunity—mental disorders, cancers, metabolic disorders, birth defects, addictions, obesity, chronic diseases,

even long Covid. Coincidentally, autoantibodies are frequently found in each of those conditions, and being diagnosed with one significantly increases the odds of being diagnosed with another.

This is not normal. They are all interconnected in a way only a single common denominator can explain. A hidden infection. Antibodies the body is producing to fight the hidden bacteria—predictably triggering widespread inflammation—have been mistakenly labelled "autoantibodies," a catastrophic epidemic of mistaken identity.

On the surface it sounds outrageous that a lone type of bacteria could cause that much carnage. Scientists have long believed it's a complicated confluence of factors that shape our wellbeing. But what if it's not that at all? To see what life might look like without Lyme disease, we need to remove it from the equation. That introduces our fourth unassailable fact.

FACT #4: LYME-FREE COMMUNITIES MUST BE LARGELY DISEASE FREE

If Lyme is driving the majority of illnesses found in society today, some communities must still exist somewhere on the planet where the disease has not yet spread widely. And those communities should be uncommonly healthy, with few of the same diseases plaguing the world at large.

Unexplained pockets of wellness do indeed exist across the globe, and they all have in common characteristics that would limit Lyme's sexual spread—communities that are both insular and isolated, so-called "Blue Zones." If intimate relations with outsiders are discouraged, particularly common with some religious groups, it makes it more challenging for the disease to get a foothold. So it's no surprise that these areas frequently have remarkably lower rates of chronic disease, cancer, metabolic diseases, and mental health issues. But for proof of Lyme's penetrative power, we need to visit the world's most isolated: those rare Indigenous communities not yet assimilated.

A 2025 study by researchers at Columbia did just that, examining Indigenous peoples from the deep jungles of Bolivia and Malaysia. And they found exactly what one would expect if Lyme was at the root of most illnesses in the world today: zero sign of chronic diseases. No diabetes, no heart disease, no Alzheimer's, nothing other than inflammation from jungle parasites. In fact, they had no age-related inflammation whatsoever, shocking scientists, completely upending the idea that aging and inflammation go hand-in-hand. But it makes sense if that inflammation is being caused by a simmering infection.

Perhaps the most illustrative example of the carnage Lyme delivers is the history of the Inuit, for a time famously isolated in the Arctic. Again, despite high-fat diets devoid of most vegetables, the Inuit enjoyed a health utopia. Other than lung cancer in women due to tending cooking fires and occasional EBV-related throat cancers, they largely lived disease-free lives. (The EBV link is especially revealing. If it, and not Lyme, had been the hidden cause of many diseases, those diseases would have shown up in this population as the virus was already widely circulating. They didn't.) But by the 1960s, assimilation with the world gradually eroded the Inuit's protective shield of isolation. Borrelia had arrived, and was never leaving, leading us to our fifth sobering fact.

FACT #5: MOST PEOPLE ARE BORN WITH AT LEAST ONE STRAIN OF LYME

Today, before leaving the womb, most children are infected with a variant of Lyme, passed down from one or both parents. Occasionally the disease will surface in childhood—often as juvenile forms of arthritis or blood cancers—but the bacteria routinely remain dormant in its human host for years, often decades, until a major immune system disturbance rousts it from its slumber. There are three primary triggers that research has identified: Illness (e.g., the flu, Covid), trauma (e.g., childbirth, car accident), and stress (e.g., death of a loved one, a breakup). The pattern is unmistakable, repeating across studies.

This also explains the odd outliers, even as doctors reflexively wave away any association. Infrequently, minor immune disruptions can awaken Lyme, which can include vaccinations, tattoos, innocuous scrapes, piercings, bee stings, etc. You're not crazy for making the connection.

This extends to the range of illnesses that tend to surface after a specific event. Scientists have documented this phenomenon for years and years, applying specific labels to each instead of identifying the universal pattern. You know them well: post-partum depression, post-traumatic stress disorder, long Covid, any number of post infectious syndromes (IBS is a dreadfully common one), including the occasional diseases that surface after routine vaccinations. It's all explainable, vindication for everyone who has been gaslit and disbelieved.

Because there are many different strains—more than 20, again, identified by research—and people can be infected by more than one, the disease presents in wildly different ways. It masquerades as autoimmune diseases, mental illnesses, myriad chronic conditions, birth defects, post infectious syndromes, addictions, and causes other medical issues including cancers, obesity, and metabolic problems, making it all but impossible to reliably and accurately identify. These misdirections have resulted in the near universal failure to develop diagnostic tests and effective treatments for countless health conditions. But not total failure. And that takes us to our stunning sixth fact.

FACT #6: ANY SUCCESSFUL THERAPY FOR LYME MUST TREAT A VAST ARRAY OF ILLNESSES.

Look at that extensive list of health issues that I've connected to Lyme again. An effective therapy for the disease should positively influence all of them. If it were a drug, no doubt it would be a blockbuster of blockbusters, run into shortages due to overwhelming demand, and its success over such a broad range of conditions would confound scientists to no end. Sound familiar?

The newest weight loss drugs—GLP-1s, more commonly known by their brand names like Ozempic and Wegovy—are doing far, far more than helping people drop pounds. To understand what's happening, it helps to know more about how Borrelia operates.

The bacteria derive energy from glucose, essentially carbohydrates. Every intervention that works across an inexplicable range of conditions—from GLP-1s to carb-limiting diets to exercise—works because it restricts the bacteria's food supply. That's why exercise helps prevent the recurrence of

cancer and why ketogenic diets aid mental health conditions. The bewildering success of the newest weight-loss drugs—improving 175 health conditions and counting—isn't bewildering at all. It's starving out the bacterial infection.

We've unknowingly been treating Lyme for ages. Once you realize that virtually everything we have been doing has been to control Lyme disease—our drugs, our diets, our exercise routines, our lifestyle changes—everything crystallizes.

The evidence is clear, overwhelming, indisputable. A deviously hidden invader lurks within most of us. But blink, and you'd miss it. Everyone blinked. Everyone missed it.

There is a familiar aphorism in science: "Extraordinary claims require extraordinary evidence." To say that Lyme is at the root of most illnesses plaguing our planet, an extraordinary claim to be sure, it needs to resolve every significant question. One stands out as the most pressing. The most vexing mystery scientists have struggled with for more than half a century: Why these illnesses disproportionately affect women. If Lyme resolves that cleanly, we have our answer.

It does, and we do, our seventh fact, the extraordinary evidence required to support extraordinary claims.

FACT #7: BORRELIA MUST EXPLAIN WHY WOMEN DISPROPORTIONATELY ARE AFFECTED BY CHRONIC DISEASE

There is only one organism on the planet that can explain why women have clear targets on their backs—and, critically, why it affects them most often in the prime of their lives. Borrelia burgdorferi, the bacterial infection more commonly known as Lyme disease. The evidence lies in a trace mineral required for the spirochete to survive and cause disease. Unlike virtually every living organism, Borrelia has no need for iron. Instead, it requires manganese, a unique, signature characteristic of the bacteria.

The damning clue is in the distinctive interplay between iron and manganese. As manganese levels rise, iron levels fall and vice versa. In childhood, boys and girls have similar levels of manganese. But once young women begin their monthly period, and start routinely losing blood, iron levels dip and manganese levels spike. The enduring mystery—why women, why in their prime—cleanly answered.

That's the reason women are far more likely to be afflicted with chronic conditions, from autoimmune diseases to long Covid. Now the dots all connect. Recall from Fact #5 that Lyme tends to remain dormant until a major immune system disturbance rousts it from its slumber. Covid does just that—a landmark August 2026 *Nature* study of more than 1,100 hospitalized patients found that Covid reactivated dormant pathogens in nearly half,[504] introducing the concept of "dysvirosis," a disruption of the body's internal ecosystem of latent infections. Researchers attributed the damage to reactivated viruses. But with women dominating long Covid by a ratio of nearly two to one, only Borrelia can explain that striking gap.

Borrelia's appetite for manganese is the elusive missing puzzle piece scientists have desperately been searching for. The body absorbs manganese from food, but several common substances block that absorption: iron (specifically non-heme iron), fiber (vegetables, legumes, seeds and nuts, grains, and berries), calcium (dairy products especially), tetracycline antibiotics (the type most recommended to treat Lyme), and, revealingly, tannins.

What's rich in tannins? Coffee, dark chocolate, and red wine—their inexplicable health benefits now fully explainable. Their tannins, when consumed in quantity, prevent manganese from being absorbed, denying Borrelia its lifeblood. (Now it makes sense why cranberry juice, chock-full of tannins, is often recommended for preventing UTIs.) And if you look more closely at the foods that manipulate manganese, you'll see the core elements of what is considered the world's healthiest diet backed by science: the Mediterranean diet. It works by denying Borrelia access to its required manganese.

For confirmation that both isolation and manganese are indeed shielding us from the devastating effects of Lyme, our eighth fact must be true.

FACT #8: THE PROTECTIVE MECHANISMS USED BY HUMANS TO EVADE OR BLUNT THE EFFECTS OF LYME MUST ALSO APPLY TO ALL MAMMALS.

Although humans sit atop the food chain, ticks don't particularly fancy where they get their next blood meal. That means all land-dwelling mammals are at risk of Lyme, the disease not only spreading by tick, but also by sex and vertically. By extension, the same factors that protect us should also apply to the animal kingdom. Two huge populations, humans and animals, with identical defenses would be devastating evidence. But medical specialists and biologists don't typically swap notes.

They should have.

The power of tannins blocking manganese absorption absolutely extends to the animal kingdom. Unlike carnivores who consume heme iron, which has little effect on manganese, plant-eating herbivores ingest the trace element, which provides prodigious protection against cancers. Size doesn't predict cancer in mammals—a long-held belief by scientists despite numerous paradoxes—diet and isolation from ticks do. That's why squirrels, beavers, and elephants have such long lifespans; acorns, bark, and many types of vegetation are loaded with tannins. Isolation is also in play, the reason whales, walruses, and subterranean naked mole rats live such long, disease-free lives. There are no ticks in their natural habitats to spread disease.

Now for our final, inescapable fact.

FACT #9: ALL OF US HAVE LYME DISEASE. THAT INCLUDES YOU.

Now, perhaps the biggest immediate challenge rests within us as humans. How can we wrap our brains around the fact that so much of modern medicine is wrong, that most of what we've been taught is incorrect? That cancer doesn't "run in the family" or that those aches and pains might not be from nagging injuries? That obesity isn't a lack of will or that mental illness may not remotely be permanent? That obesity isn't a lack of will or that mental illness may not remotely be permanent? That chronic inflammation is not a normal part of aging or the cause of disease. A landmark Novo Nordisk trial published August 6, 2026, confirmed as much[505]—a drug that successfully eliminated inflammation markers still failed to prevent cardiovascular death, leaving cardiologists "in a state of shock."

But the one hurdle hardest to overcome is ourselves—admitting that each of us is infected. History has demonstrated that evidence, even overwhelming, indisputable evidence, struggles to gain traction when minds are convinced otherwise. Which is why this solution, a paradigm shift of the highest order, faces daunting odds.

Will science and the data supporting it ultimately win out? Yes, it always does. The good thing about science is that it's true whether or not you believe in it. There is only one question that remains.

When?

Regardless of when humanity accepts its fate, there is no time to waste. Globally, an estimated 110,000 people die every day from chronic disease, most of which are directly connected to Borrelia. In the United States alone, heart disease, cancer, stroke, and Alzheimer's—all conditions driven by this infection—kill more than 1.5 million Americans annually. The financial toll is equally staggering: 90% of the nation's $5.3 trillion in annual healthcare expenditure goes toward managing chronic disease and mental health conditions. That is not a healthcare system. That is a civilization paying an annual ransom to a bacterium it doesn't know it has.

Until a cure is found, these are the interventions that can address Lyme immediately: GLP-1s, metformin, tetracycline antibiotics, carbohydrate-limiting diets, manganese manipulation through diet, antihistamines, exercise, and lifestyle changes. Most are already widely available. None require a confirmed diagnosis. All buy time.

The path to a cure exists. The research to find it is already underway. The question is whether the scientific community will engage with the evidence before more lives are lost.

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