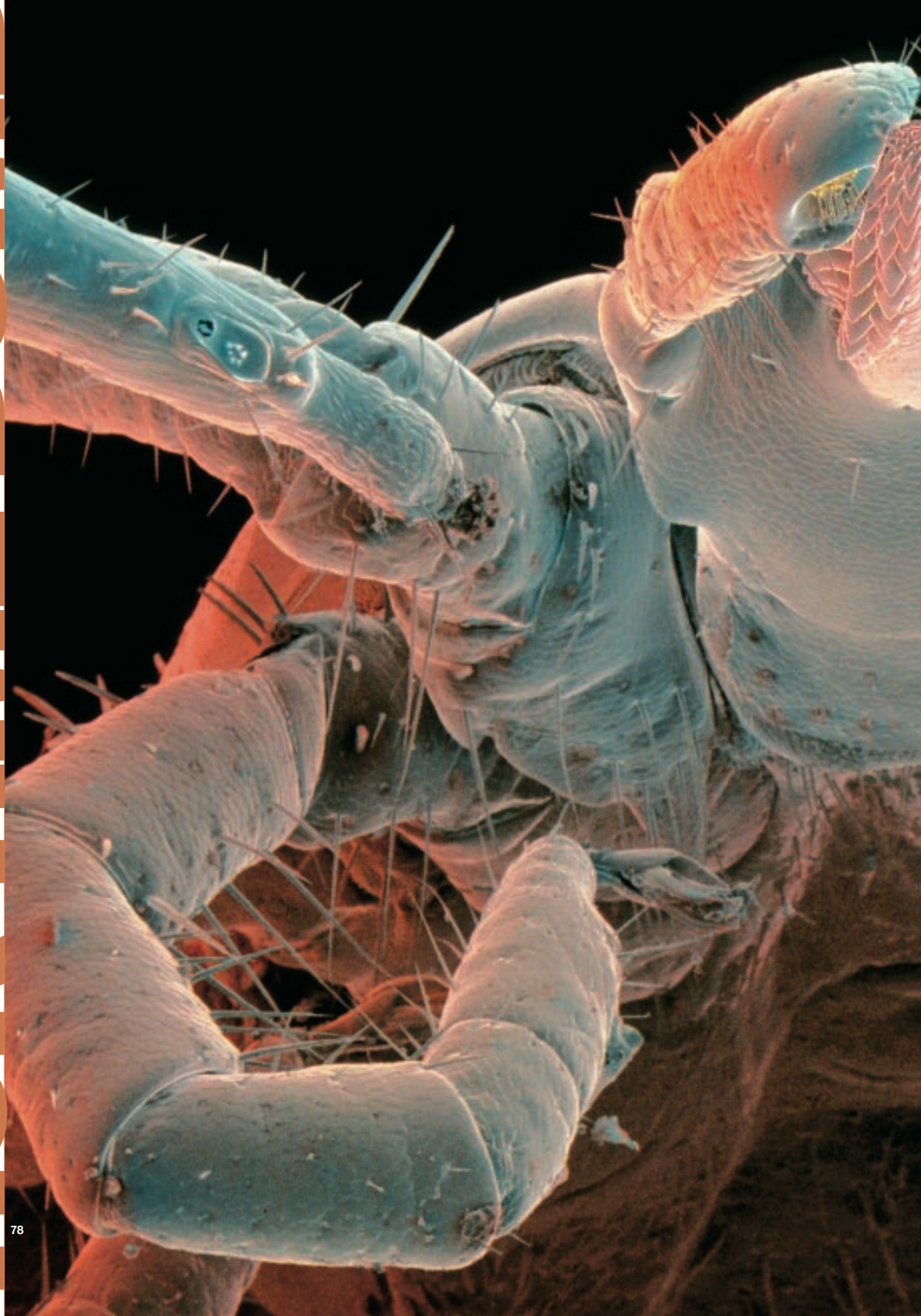
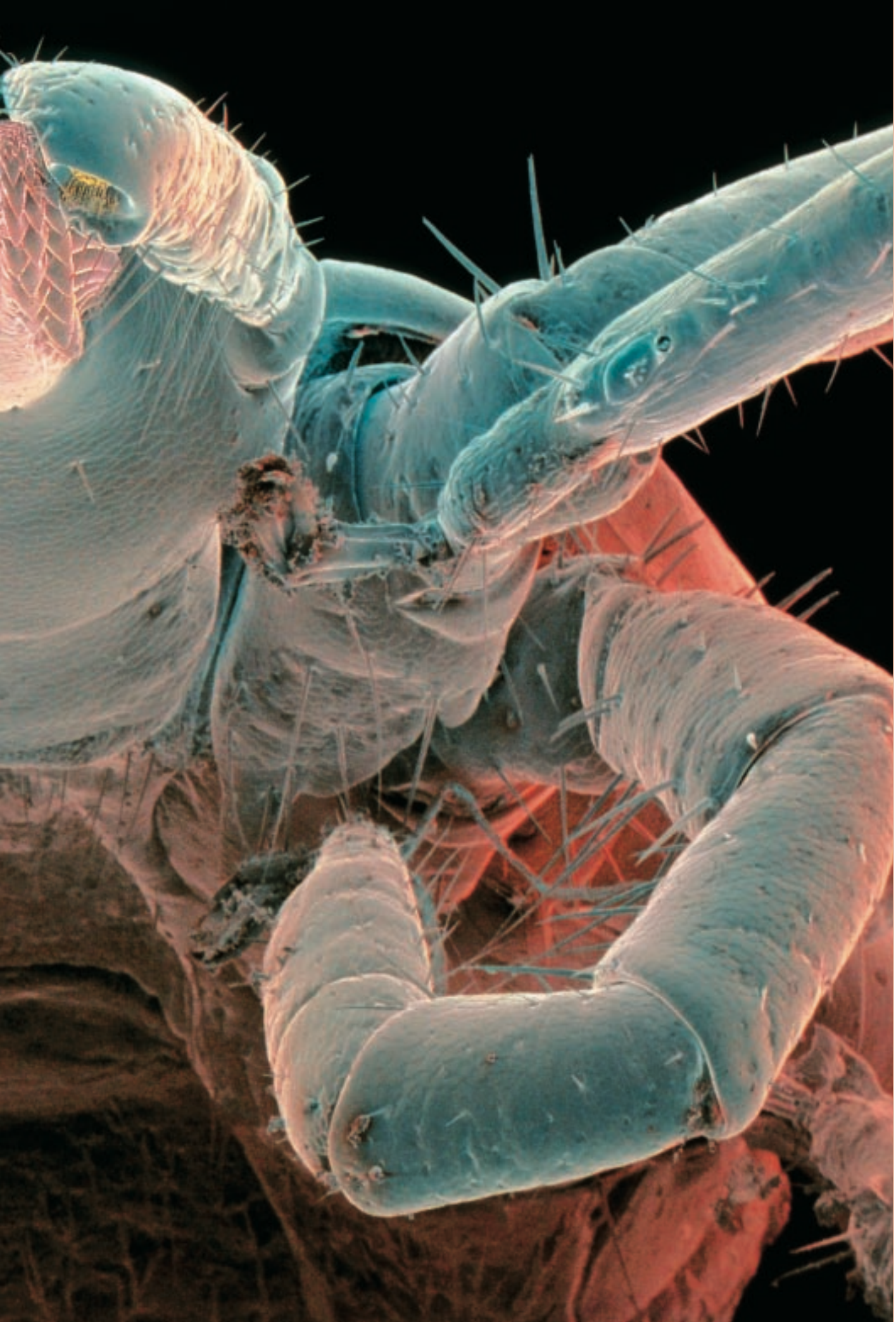
TICK TIME BOMB





The explosion of symptoms—muddled thinking, fatigue, a buzzing numbness in her limbs—

set this writer on a quest for an answer. Stress, the doctor said. But the culprit lurked not in her 24/7 schedule but in the woods surrounding her home

By Pamela Weintraub

By age 16, Jason couldn't tolerate sound or light or, many days, even get out of bed. Specialists suggested diagnoses from "migraine aura" to "parvovirus" to depression, but no treatment ever helped him get well

In the mid-1990s, after moving to the upscale, lushly forested suburb of Chappaqua in Westchester County, New York, I began to slide. The flickering headaches, dully aching joints, and intermittent fatigue were so subtle they seemed almost normal in the life of a working mom. Yet by 2000 the odd mix of symptoms had intensified into signs of disease. My knees swelled, sometimes hurting so much I descended the steps of my house while sitting. Whenever I ate, I choked. My arms and legs buzzed, at first gently but then so palpably it felt like an electric current was coursing through them. My vision blurred and the fatigue became withering, laying me low for hours every day. The headache (now tinged with nausea) occurred more frequently, and eventually it never left. Most disturbing was a strange inability to think. I was under a publishing contract to write a book about the brain, but my own brain was shrouded in fog. Despite two decades as a science journalist, I couldn't follow the neuroscience I'd once read with ease.

Seeking treatment, I was told the problem might be genetic. "Migraines run in families," a local internist blithely said. She prescribed a number of the latest migraine medicines in quick succession, but although they were considered effective for other people, none worked for me. My issues amounted to aging combined with stress, the doctor explained. In my forties I could expect some decline. And I certainly was stressed out: My oldest son, Jason, suffered a mystery illness far more disturbing than my own.

Starting the year we took up residence in our fairy-tale house by the woods, at the age of 9, Jason had become exhausted and then suffered shooting, stabbing pains in his legs. The doctors called these "growing pains" normal, but by 2000, at age 16, he couldn't tolerate sound or light, follow the text in his schoolbooks, or, many days, even get out of bed. As his condition worsened and all sorts of lab tests came back negative, specialists at New York City's top teaching hospitals suggested diagnoses from "migraine aura" to "parvovirus" to depression, but no treatment prescribed ever helped him get well. Getting to the bottom of Jason's illness took most of my time.

Hitting Lyme Bingo

"Could this be Lyme disease?" I asked the nearby Mount Kisco Medical Group and other doctors we consulted again and again. After all, Jason had spent summers in the woods in a toy-stocked fort behind our house. Chappaqua was overrun with deer that carried ticks, and it took only a single tick bite to cause Lyme disease.

"No, absolutely not," I was told by expert after expert, as tests came back negative or equivocal for *Borrelia burgdorferi,* the tick-borne spirochete that causes Lyme.

When Jason developed a huge red rash with areas of white across his torso in 1998, I called the Mount Kisco Medical Group again, describing it in detail over the phone. "Shouldn't I bring him in? Maybe it's Lyme disease," I said.

"Don't bother coming in," the nurse responded with great authority. A Lyme rash, she informed me, was always literally a bull's-eye. As a medical journalist, I should have known better; I could have turned to the world's top medical journals to see hundreds of Lyme rashes just like Jason's. Incredibly—perhaps because my own brain was already so compromised—I did not.

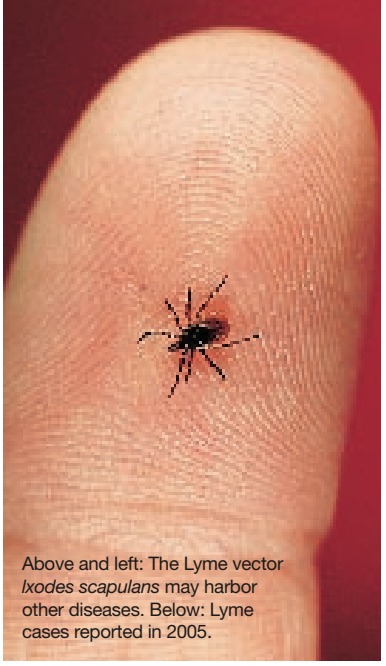
I didn't understand back then how controversial Lyme disease had become: While academic medicine insisted Lyme was easy to treat, a group of persistently sick patients and their doctors, mostly from the suburbs, declared that sometimes the infection couldn't be cured. Incurable, chronic Lyme, these heretics said, required long-term or even lifelong antibiotic treatment, often at very high doses. Patients like Jason, with acute or long-standing untreated Lyme disease, could be swept up in the fight and summarily dismissed as well.

It was in 2000 that Jason hit Lyme bingo. He finally tipped the scales for Lyme disease on a Western blot, a test for detecting particular antibodies in blood. To validate a case of late Lyme disease beyond any doubt, the Centers for Disease Control and Prevention (CDC) require that the blot detect 5 of 10 specific antibodies produced to fight the spirochete. Jason's test from the standard commercial lab, LabCorp, had 8.

The author with her son. Previous pages: The tick that carries Lyme.

Later, as I looked over Jason's old blood tests and interviewed experts at teaching hospitals, I came to understand the magnitude of a medical error that should never have occurred: Jason had harbored evidence of infection for years in the form of increasing numbers of antibody bands—first 2, then 4, until he reached the unequivocal 8. Combined with the expanding rash and the swollen knees, his turned out to be such a classic case of Lyme that it was reported to the CDC as a surveillance case, one considered so definitive it is used to help scientists track the disease. Doctors had missed the correct diagnosis for a year or two at least, according to one specialist. I suspected it was much longer. Jason would have a long road ahead of him on the path to wellness, but at least now we knew what was wrong.

With Jason's diagnosis set in stone and his treatment under way, I turned back to myself: Could I have Lyme disease, too? I lived alongside the same forest dense with deer, in a town identified by Yale University as a Lyme disease hot spot. Often I gardened, plunging my fingers into the soft brown soil without gloves or any other protection.



Above and left: The Lyme vector *Ixodes scapulans* may harbor other diseases. Below: Lyme cases reported in 2005.

Yet in my quest for a Lyme diagnosis, I was at a terrible disadvantage, compared with Jason. While he'd finally tested positive on the gold-standard Western blot and had developed the rash, I had no such absolute proof. Instead, my tests, conducted at the well-regarded Laboratory for the Diagnosis of Tick-Borne Diseases at Stony Brook University School of Medicine, were equivocal.

First my blood was tested on a Lyme ELISA (Enzyme-Linked Immunosorbent Assay). This test employs a plate coated with ruptured spirochetes. When an infected patient's blood is washed over the plate, the antibodies generated against the spirochete will latch on and stick to the plate; the "stickier" the blood, presumably the worse the Lyme. As it happened, my Lyme ELISA was highly reactive, four times the level required for a positive. But this was only a first step, according to the doctor and the CDC. A positive ELISA would have to be confirmed by a Western blot, and there I was out of luck. Like Jason before me, I needed 5 of 10 bands for a positive, but I had only 4. Four specific bands combined with signs of disease in an area as infested as Chappaqua should have warranted treatment, some experts later told me. But with Lyme disease so controversial and doctors so cautious they stuck to cookie-cutter guidelines, I landed outside the curve.

In the end, I sought treatment from a nurse practitioner with an office across the street from Chappaqua's favorite watering hole, Starbucks, right on the main road. Considered skilled by the Lyme patient community, she enjoyed a small clientele. These patients came from a handful of Westchester physicians who rejected restrictive views on Lyme disease but didn't want to deal personally with the controversy. She was as natural to Chappaqua as its medical massage parlors, as native as Lyme disease itself.

I was lucky she took me on. Despite my equivocal test results, she understood that I was sick. It wasn't just my residence at the heart of the epidemic in Chappaqua and my constant exposure to ticks that informed her but also my signs and symptoms. The Mount Kisco doctors had said my fatigue and migraine, my numbness and confusion had nothing to do with Lyme disease, but she vehemently disagreed.

At first she treated me with the common antibiotic, amoxicillin. Since the buzzing, confusion, and headache indicated the infection was neurological, she prescribed a dose high enough to reach my brain. Initially, I got worse. It was the die-off of the spirochete flooding my system and causing what experts called a Herxheimer reaction, "an intensification of the symptoms," the nurse practitioner said. My "Herx" was so severe that after a few days of the antibiotic I took to bed. Every joint ached, my left hand formed a claw that couldn't unclench, and every nerve felt electrified to the root. The sensation of cotton seemed to push out of my extremities, especially my hands and my head. My migraine flared so much it felt like a drill in my brain, revving a tidal wave of nausea in my gut. I tried to stand up, but my muscles were too weak. It lasted days.

Then the illness subsided, and I floated back up. Slowly, the "cotton" thinned, and the drill in my brain quieted. The claw unclenched and the buzzing subsided. I could get out of bed and walk. Several weeks later I awoke with a lightness of being I could not at first pinpoint. One of my symptoms was missing: the headache. Along with the nausea, it was gone for the first time in years.

A week or so afterward, my 20/20 eyesight returned, allowing me to see into the distance and read the fine print in *The New York Times.* One day I picked

I lived alongside a forest dense with deer, in a town identified as a hot spot. Often I gardened, plunging my fingers into the soft brown soil without protection

P. 78–79: DAVID SCHARF/PHOTO RESEARCHERS; KENT WOOD/ PHOTO RESEARCHERS. P. 80: COURTESY PAMELA WEINTRAUB. P. 81, CLOCKWISE FROM LEFT: GARY GAUGLER/PHOTO RESEARCHERS; SCOTT CAMAZINE/PHOTO RESEARCHERS; U.S. CENTERS FOR DISEASE CONTROL AND PREVENTION.

up the neuroscience I'd been unable to comprehend and read it to the end. To be sure, I wasn't entirely well. The headache would return. I was exhausted, and intermittently I still buzzed. But a layer of the illness was gone.

On the Trail of Nantucket Fever

Sometime after the New Year, I drove into town for a latte at Starbucks and an appointment with the nurse. She greeted me with a smile, waving lab results at me. A couple of weeks before, dissatisfied with a plateau in my recovery, she'd drawn blood and sent it to Quest Lab to test for the presence of *Babesia,* a genus of tick-borne sporozoa known for causing fevers and sweats, as well as headache and malaise. "When Lyme disease patients don't get well," she told me, "coinfection with babesiosis can be the cause." Now the results had come back. As with my Lyme ELISA, antibodies were sky-high, four times the cutoff for positive.

Few Lyme patients had even heard of babesiosis, though the two epidemics had been spawned in tandem, could be equally debilitating, and were spreading at proportional rates

The pieces were falling into place; the babesiosis diagnosis certainly made sense for me. A decade earlier, in 1990, I'd spent a month as a science-writing fellow at the Marine Biological Laboratory at Woods Hole, Massachusetts, right across the water from Nantucket Island, where tropical medicine expert Andrew Spielman of the Harvard School of Public Health had charted human babesiosis more than 15 years before.

It was during the early seventies, before Lyme was even recognized, that Spielman was asked to investigate what locals called Nantucket fever; at the time, only two patients were known. The first was a wealthy Nantucket woman who came down with a disabling mystery illness marked by extreme anemia, fatigue, and fever that local doctors could not explain. So she chartered a plane to Rutgers in New Jersey. Rutgers doctors examined her blood under a microscope, diagnosed her with malaria, and placed her on the standard treatment, chloroquine. When the treatment didn't work, they grew alarmed because treatment-resistant malaria is, after all, a threat to public health.

A slide of blood was shipped off to the CDC, where experts identified not malaria but another similar agent that also inhabits red blood cells—*Babesia microti,* a cousin of *Babesia spp.,* known to cause cattle epidemics that wiped out entire herds. With the identification of her infection, the woman was finally treated correctly and got well.

When a second case of babesiosis appeared on Nantucket Island a few years later, physicians again were stymied. But the second patient happened to be friends with the first, and finally, with doctors throwing up their hands, it fell to the first patient to diagnose the disease in the second. Her lay diagnosis was correct, and the second patient was treated and recovered as well.

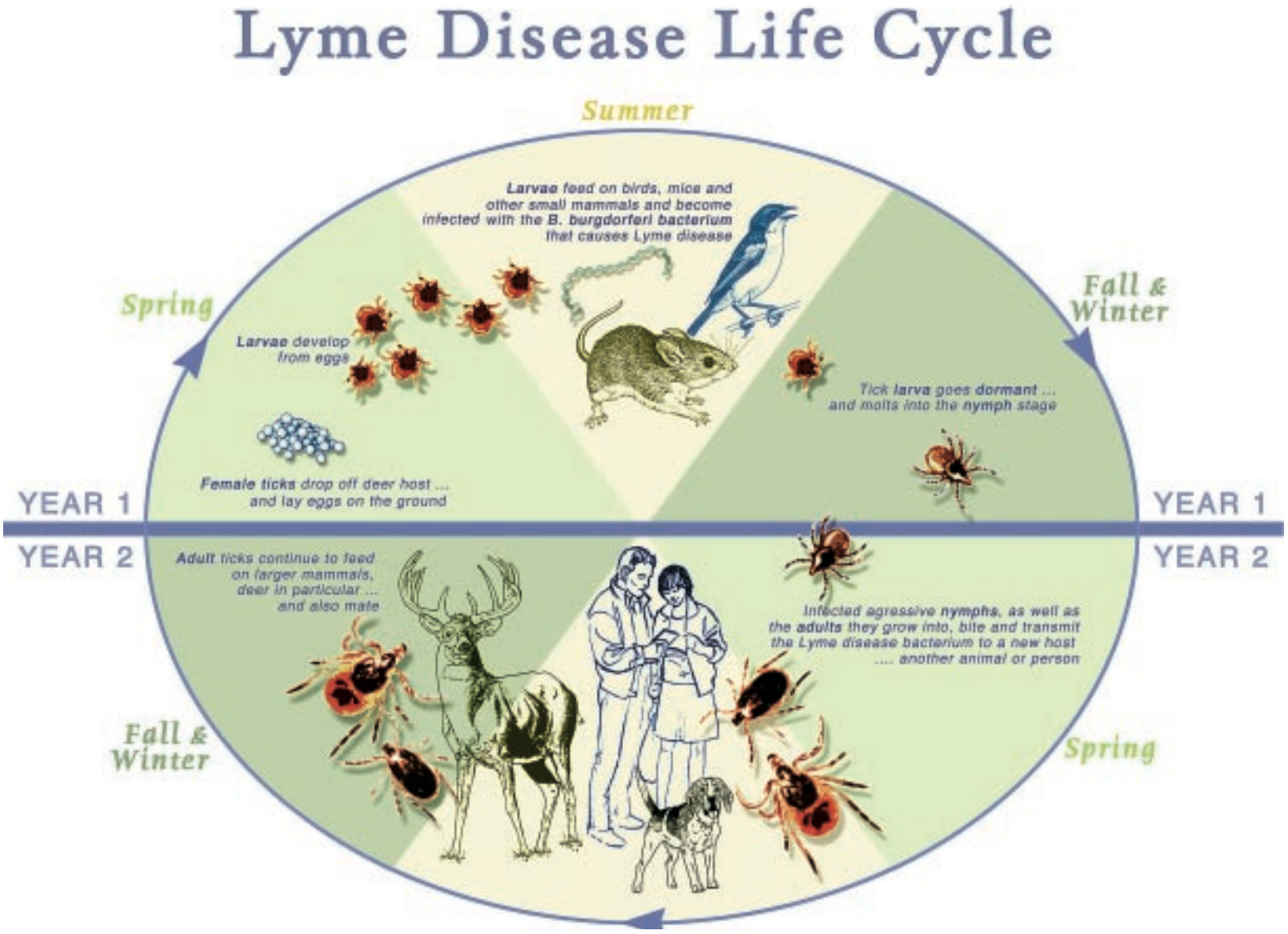
That's when Spielman entered the fray. Would he care to find the cause of these cases in the environment? Observing the cycles of infection year after year, he finally tracked *Babesia* through the ecosystem, discovering that it lived in the blood of mice and spread from one mammal to the next through the bite of an *Ixodes* tick. Larva and nymphal (baby and adolescent) ticks fed off the mice, but adult ticks reproduced en masse only after taking a blood meal from large mammals like deer, ensuring the cycle would continue. The spirochete *Borrelia burgdorferi* was identified as the cause of Lyme disease in 1975, and Spielman proved shortly afterward that this newly discovered illness involved the same tick and the same natural cycle.

But few Lyme disease patients had even heard of babesiosis; though the two epidemics had been spawned in tandem, could be equally debilitating, and were spreading at proportional rates along with migrating deer, few primary care doctors in endemic areas like Chappaqua ran the babesiosis test. "We don't test for that," our Mount Kisco pediatrician explained at the time. The internist who tried to treat my headaches—classic for babesiosis—never mentioned the possibility that infection, either Lyme or babesiosis, might be a cause.

Yet in retrospect I believe the babesiosis diagnosis was my missing link. Most science-writing fellows at Woods Hole had stayed in residence halls near the lab, but with a family in tow, I was given a spacious rustic cabin in the woods. Way before my arrival, *Babesia microti* had begun its migration, first over Cape Cod and then down the Long Island Sound, fast on the heels of Lyme disease, toward Connecticut, Westchester, and points beyond. In 1990, still traveling incognito toward New York State, *B. microti* was already rife in the forested enclaves of Woods Hole.

The author's house in leafy Chappaqua. The neighborhood looks benign, but *Ixodes* ticks in these backyards harbor diseases that may not be picked up by routine medical screenings.

It wasn't just my exposure that fit with the Quest Lab results but also the mystery illness I'd suffered after returning from Cape Cod. I never understood the strange spikes of fever up to 105 degrees Fahrenheit that hit me in hallucinogenic waves for more than a week that August, or the gullies of sleep so black that, except for the nightmares, I thought I might be dead. When the fe-



ver broke and I noticed the sweating, it seemed just a consequence of summer—perhaps the air conditioner was on the fritz, but only I could tell. After the sweat leveled off, I felt the start of what would become, a decade hence, the headache without end.

This was classic acute babesiosis. Without treatment, an acute infection may have flared and then smoldered. Later it may have acted synergistically with the Lyme.

My Chappaqua nurse had a treatment to push the babesiosis back: I now added Mepron (atovaquone) to my arsenal of antibiotics. The thick gold sludge, known for treating malaria, made me want to vomit. But I held it down. Some six weeks later the drill in my head stopped whirring, and the nausea and dizziness I'd lived with for years receded like a tide pulled back to sea. I still wasn't well, not entirely, but the treatment had extinguished another set of disease symptoms.

The archaeologist of my own illness, I sifted through the years: Perhaps I'd first gotten sick at Woods Hole with a mix of infections that make up the Lyme soup. With infections smoldering but symptoms under the radar, I could have been bitten repeatedly during my travels to the Hamptons, to a campground in Dutchess County, to a bed-and-breakfast in Bucks County, to Martha's Vineyard and a dozen towns on Cape Cod, to beaches and parks up and down the Jersey shore—all areas known for Lyme and babesiosis. Then there was the disastrous move to Chappaqua and our permanent residence in the woods.

It had taken years to get to the bottom of my strange illness, but I was lucky to be diagnosed at all—especially because the Lyme-babesiosis combination remains a confounding and toxic brew. One of the first to understand the implications was University of Connecticut physician Peter Krause, an expert in pediatric infectious disease. Patients infected with Lyme and babesiosis together often feel sicker, and stay sick longer, than those with just one disease. In one telling study, Krause found some 3 percent of patients treated for early Lyme were still fatigued six months later, compared with more than a third of those coinfected with babesiosis and Lyme. No wonder I felt so ill.

Yet babesiosis can be devastating

The nausea and dizziness I'd lived with for years receded

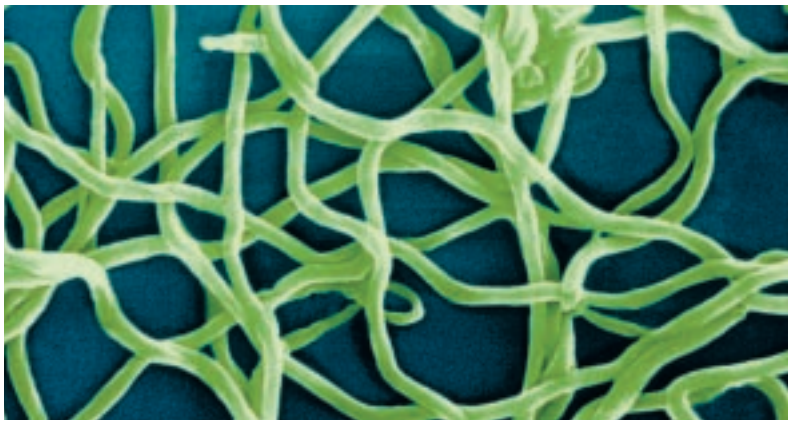
P. 82: COURTESY PAMELA WEINTRAUB. P. 83: KEVIN A. SOMERVILLE/PHOTOTAKE.

A host of alternate tick-borne spirochetes will never register on a Lyme test but may cause illness. As long as we live in suburbs carved into woods, we'll be in the path of the tick tornado

on its own. It is fatal in 5 percent of patients, especially the elderly or those with immune problems. As apparently had happened to me, untreated babesiosis can smolder, persisting for months or even years. Unmanifested *B. microti* creates a worst-case scenario, leaving patients with such a high parasitic load that the illness, when it arrives—and it generally does—makes them especially sick.

As I learned while navigating my way back to health, the disease we colloquially call Lyme may consist of other tick-borne infections—and may or may not involve the spirochete associated with the disease discovered in Lyme, Connecticut, in 1975. A third category of infection, referred to as ehrlichiosis, is caused by the microbes *Ehrlichia chaffeensis* in the South and *Anaplasma phagocytophilum* in the North. Infecting the white blood cells, these microbes cause fever like babesiosis, and a deep, painful malaise, as is often reported in Lyme. When Lyme disease patients are treated with the antibiotic doxycycline, ehrlichiosis is treated as well. But when doctors treat Lyme disease with amoxicillin or a variety of other antibiotics, ehrlichiosis persists. Many an "incurable" Lyme patient has discovered the existence of a second, lurking disease—ehrlichiosis—only to be treated with doxycycline and finally get well.

Added to the triad of Lyme-babesiosis-ehrlichiosis are other suspect pathogens inhabiting the same ticks, among them the rod-shaped bacterium *Bartonella henselae* and *Mycoplasma fermentans*, both sometimes invoked as causes of neuropsychiatric symptoms and chronic fatigue. A host of alternate tick-borne spirochetes still under investigation will never register positive on a Lyme disease test but may cause illness as well. As long as we live in suburbs carved into woods, we'll be in the path of the tick tornado. No one would think of trekking through Africa without prophylaxis for malaria, yet we take that risk every day by venturing, without worry, through our own backyards.



Top: In the spring, tick nymphs linger near the ground, lurking in grass and vegetation as they wait for a passing bird or mammal. Bottom: The Lyme disease spirochete *Borrelia burgdorferi*.

Our personal risk stayed high until we left Chappaqua in spring 2004. Though we never got as sick as we'd been in 2000 and 2001, we continued to relapse as long as we lived in the ecosystem, requiring new rounds of antibiotics that pushed the illness back, only to relapse again. I was meticulous about avoiding exposure, yet I could never be sure whether I relapsed because I suffered the controversial chronic form of the illness or had simply been bitten again. It was only after I moved to an urban high-rise surrounded by concrete that a final round of antibiotics extinguished my illness for good in 2005. As for Jason, he regained his hard-driving intellect after aggressive treatment for Lyme disease, but for a very long time, his pain and fatigue remained. He is currently pursuing his dream as a film student at Brown University, but he paid a hefty price, sacrificing his childhood for an infection that could have been cured swiftly if it had been treated early on. Ω

Pamela Weintraub is a consulting editor at *Psychology Today* and executive editor at *MAMM*. She is also working on a book about tick-borne disease.

FROM TOP: PANORAMIC IMAGES/GETTY IMAGES; STEM JEMS/PHOTO RESEARCHERS.