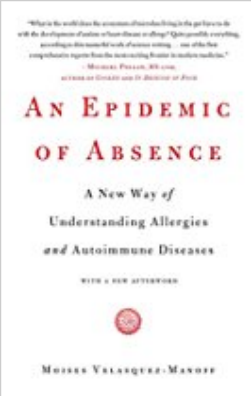

An Epidemic of Absence: A New Way of Understanding Allergies and Autoimmune Diseases

By Moises Velasquez-Manoff

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A brilliant, groundbreaking report on the dramatic rise of allergic and autoimmune disease, and the controversial therapies scientists are developing to correct these disorders.

From asthma to Crohn's disease, everyone knows someone who suffers from an allergic or autoimmune disorder. And if it appears that the prevalence of these maladies has increased recently, that's because it has—to levels never before seen in human history. These days no fewer than one in five—and likely more—Americans suffers from one of these ailments. We seem newly, and bafflingly, vulnerable to immune system malfunction. Why? One possibility is that we have systematically cleaned ourselves to illness; this belief challenges deeply entrenched notions about the value of societal hygiene and the harmful nature of microbes. Yet scientists investigating the rampant immune dysfunction in the developed world have inevitably arrived at this conclusion. To address this global "epidemic of absence," they must restore the human ecosystem.

This groundbreaking book explores the promising but controversial "worm therapy"—deliberate infection with parasitic worms—in development to treat autoimmune disease. It explains why farmers' children so rarely get hay fever, why allergy is less prevalent in former Eastern Bloc countries, and how one cancer-causing bacterium may be good for us. It probes the link between autism and a dysfunctional immune system. It investigates the newly apparent fetal origins of allergic disease—that a mother's inflammatory response imprints on her unborn child, tipping the scales toward allergy.

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Review
“Remarkable…Moises Velasquez-Manoff draws together hundreds of studies to craft a powerful narrative carrying a fascinating argument.” (*Wall Street Journal*)

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About the Author
Moises Velasquez-Manoff covered science and the environment for *The Christian Science Monitor*, and his work has appeared in *The New York Times Magazine*, *The Chicago Tribune*, and *Slate*, among other publications. He graduated from the Columbia Graduate School of Journalism’s Master of Arts program, with a concentration in science writing.

An Epidemic of Absence

CHAPTER 1

Meet Your Parasites

Mother, it is no gain, thy bondage of finery, if it keep one shut off from the healthful dust of the earth, if it rob one of the right of entrance to the great fair of common human life.1

—Rabindranath Tagore,
Bengali poet and Nobel laureate

One chilly November morning, I head south from San Diego in a bottom-tier rental car. The standard journalistic paraphernalia—a digital recorder, camera, notepad, and pencils—accompany me in the passenger seat, as well as directions to my meeting point: the last exit before Mexico. I also have a printout of my recent blood work, proof that I’m not anemic, not infected with hepatitis or HIV—that I’m healthy enough for the coming experiment.

As I drive, the radio announcer conducts a gruesome tally of the most recent violence in Tijuana, where I’m headed: two bodies hung from a bridge, a third decapitated, a fourth shot. More than this terrible, ongoing brutality, however, parasites occupy my mind—worms that migrate through flesh, burst into lungs, crawl down throats, and latch on to tender insides. Any traveler might fret over acquiring such hangers-on while abroad. But I’m heading to Mexico precisely to obtain not just one, but a colony. Today in Tijuana I’ll deliberately introduce the hookworm Necator americanus—the American murderer—into my body.

And for this dubious honor, I’ll pay handsomely—a onetime fee of $2,300. If I receive twenty of the microscopic larvae, that’s $115 apiece for a parasite that, in the early decades of the twentieth century, was considered a scourge on the American south. Some worried—without condescension, I should add—that hookworm was making southerners dim-witted and lazy, that it was socially and economically retarding half

the country. And photos of poor, worm-ridden country folk from the time—followed by their robust health after deworming—clearly show the dire costs of necatoriasis, or hookworm disease: jutting collarbones, dull eyes, and listless expressions on wan faces. They appear as if consumed from the inside.

Hookworm has mostly disappeared from the U.S., the result of protracted eradication efforts in the early twentieth century. But in the usually poor, tropical countries where it's still endemic, it can cause anemia, stunt growth, halt menstruation, and even retard mental development in growing children. Between 576 million and 740 million people carry the parasite. And for all the aforementioned reasons, public-health types consider worm infections a "neglected tropical disease." Helminths, as they're called, are not as obviously fatal as malaria, say, but their constant drag on vitality is insidious. The parasites keep children from learning in school. They prevent parents from working. Some argue that they contribute to the self-reinforcing cycles of poor health and poverty that plague entire nations.

So why am I considering acquiring this terrible creature? Scientists have two minds about parasites these days. Some consider them evil incarnate, but others note that while the above-mentioned horrors are sometimes true, the majority of humans infected with parasites today—upward of 1.2 billion people, or somewhere between one-fifth and one-sixth of humanity—host worms with few apparent symptoms. This camp has begun to suspect that worms may, in fact, confer some benefits on their human hosts.

As early as the 1960s, by which time hookworm had been largely eradicated in the U.S., scientists puzzled over the lack of symptoms in some. "Well-nourished persons often harbor helminths without apparent damage," remarked one physician in 1969.2 "One may question the wisdom of treating such infections, especially with chemotherapeutic agents with toxic qualities."

Decades of plumbing the mechanisms that allow one creature to persist within another, a clear violation of the self-versus-nonself rules thought to govern immune functioning, has taught immunologists much not only about how wily worms really are, but also about how the human immune system actually works. Parasites like hookworm were ubiquitous during our evolution. Might our bodies anticipate their presence in some respects, require it even? And might some of the more curious ailments of modernity result partly from their absence?

That brings me to my motive: A large and growing body of science indicates that parasites may prevent allergic and autoimmune diseases. And I've got both.

* * *

When I was eleven, my hair began falling out. My grandmother first noticed it. I was visiting my grandparents at their beach house that summer when, one afternoon, she called me over, examined the back of my head, and proclaimed that I had a nickel-sized bald spot. Then we all promptly forgot about it. With the sand, waves, and sun beckoning, it just didn't seem that important.

But by the time school started a few months later, the bald patch had grown. A dermatologist diagnosed alopecia areata, an autoimmune disorder. My immune system, normally tasked with protecting against invaders, had inexplicably mistaken friend for foe, and attacked my hair follicles. Scientists didn't know what, exactly, triggered alopecia, but stress was thought to play a role. And at first glance, that made sense. My parents were in the middle of a messy, drawn-out divorce. I was also beginning at a new junior high school that fall; I had, it seemed, much to worry about.

I also had other, better-known immune-mediated problems. I suffered from fairly severe asthma as a child,

and food allergies to peanuts, sesame, and eggs. (Only the egg allergy eventually disappeared.) At least once yearly, usually during seasons of high pollen count, my wheezing became so severe that my lips and fingernails turned blue, and my parents had to rush me to the emergency room. There, doctors misted me with bronchodilators, or, during severe attacks, pumped me full of immune-suppressing steroids.

“Aha!” said the dermatologist when he learned of these other conditions. There was a correlation among allergies, asthma, and alopecia, he explained. No one was sure why or what it meant, but having an allergic disease like asthma increased one’s chances of developing alopecia.

Years later, I would learn that the co-occurrence of these two disorders was likely evidence of a single, root malfunction. But at age eleven, I accepted on faith that where one problem arose, so, probably, would others. So what to do? Given my age and the relatively small size of the bald spot, the doctor recommended watching and waiting. Alopecia usually corrected itself in time, he said. So we waited.

In a month, another bald spot appeared, on the right side of my head. Then one on the left. Seemingly overnight, a large one opened up just above the middle of my forehead. As more hairless patches appeared, the pace at which new ones emerged accelerated. Every morning, my mother combed and gelled my hair into place to hide the growing expanse of denuded skin; but soon, concealing my bare scalp became nearly impossible. The spots began to converge. I was going bald.

We returned to the dermatologist. This time, he had a less upbeat assessment. The more the disease progressed, he noted, the less likely recovery. The odds worked like this: Only 1 to 2 percent of the population got alopecia areata at all, a bald spot or two that, after a time, usually filled in again.3 But for a significant minority, maybe 7 percent of those with alopecia areata, the hair loss became chronic. Some progressed to alopecia totalis, total loss of hair on the head. At that point, the chances of a full recovery diminished substantially. Whatever mistake the immune system had made, it became permanent. And of this totalis subset, some went on to develop alopecia universalis—loss of hair on the entire body. For them, recovery was nearly impossible.

None of this sounded good, especially as I was speeding toward totalis and—who knows?—universalis after that. Two treatment options existed, neither of which worked without fail: immune suppression or irritation. Steroids suppressed the immune response and, basically, called off the attack dogs, allowing hair to grow again. Immune stimulation, on the other hand, worked in slightly more mysterious ways. Inflammation induced by an irritant distracted the immune system from less pressing projects, such as attacking hair follicles. Irritation would earn my hair follicles a reprieve. As neither approach was a sure bet, the dermatologist recommended that I try both.

I did, and neither worked—although I developed an oozing blister where I applied the irritant. My alopecia advanced until, by age sixteen, not a single hair remained on my body. I had joined the elite ranks, somewhere around 0.1 percent of the population, of those with alopecia universalis. I put on a hat, which I’d wear more or less nonstop until my early twenties, and tried to get on with my adolescence.

* * *

Not until my thirties did I look into what scientists had discovered in the roughly twenty years since that first bald spot appeared on my head. I wasn’t too hopeful; surely, I would have heard had a cure been developed. As I contemplated having children, I’d begun fretting about what lay hidden in my genes. The first genome-wide association study of alopecia, published in 2010, showed that the disorder, the most common autoimmune disease in the U.S., shared gene variants with several much worse autoimmune diseases, such as

rheumatoid arthritis, type-1 diabetes, and celiac disease.4 Soon thereafter, my first child, a girl, arrived. Now the results of my investigation had concrete applications. If alopecia suggested a tendency toward immune malfunction, and if that tendency was modifiable, I wanted to know how to better play the cards. I wanted to ensure that my progeny remained free of both allergic and autoimmune disease.

I was right about one thing. Treatments for alopecia hadn't advanced much since my childhood. They still consisted mainly of irritants and immune suppressants, and as neither approach corrected the underlying malfunction, both would require indefinite use. Prolonged exposure raised a host of secondary concerns. Repeated steroid shots, for example, were not only exceptionally painful, they thinned and discolored the skin. Irritants induced swelling, redness, and skin flaking. One powerful immune suppressant called cyclosporine increased the risk of skin cancer. No thanks.

However, the patterns of immune-mediated disease in general caught my attention. The incidence of both autoimmune and allergic diseases had recently increased, and to the degree that scientific literature conveys feeling, in this case it evinced alarm. Scientists threw around the word epidemic to describe the rising prevalence of asthma especially, a descriptor usually reserved for infectious diseases, like the prayer-inducing, body-wasting, dead-in-a-day cholera epidemics that terrified the world during the nineteenth century. Generally speaking, however, there was no asthma bacterium, no autoimmune virus. No new plagues were driving this particular pandemic. Instead, we seemed newly vulnerable to immune dysfunction.

If I possessed glasses that afforded me the power to see otherwise non-apparent allergic and autoimmune diseases, I'd be struck by the sheer abundance of people with these problems. Walking down Broadway in New York City, for instance, one of every ten children passing by would have asthma; one in six would have an itchy rash and sometimes blisters—eczema.5 One of every five passersby would have hay fever. If I could see allergic antibodies directly—immunoglobulin-E—I'd note that half the crowd around me was sensitized to dust mites, tree pollen, and peanuts, among other basically harmless proteins. I'd see pockets full of inhalers, and bags stuffed with allergy medicines. In the satchels of the most severely afflicted, I'd see pills of powerful immune suppressants, such as prednisone. I'd even see a few soon-to-be corpses; about 3,500 people die yearly from asthma attacks.

Americans spend perhaps $10 billion yearly on asthma-related drugs and doctor visits. Direct and indirect costs of asthma combined reach about $56 billion. I'd see these funds flowing from allergic and asthmatic wallets to doctors and drug companies. And I'd observe money not flowing from days missed at work, diminished overall productivity, and opportunities lost over a lifetime.

If I took the same walk with glasses that allowed me to see autoimmune diseases, I'd note that one in twenty passersby had one of eighty of these often debilitating conditions.6 One of every 250 people—it would take about a minute standing in a place like Times Square for such a person to pass by—would suffer from debilitating pain in his or her intestines, what's called inflammatory bowel disease.7 I'd see scarring and constriction. And in the most severe cases, I'd observe removed lengths of intestine, colostomies (surgically created exits for intestinal contents), and colostomy bags (containers for the effluence) hidden under clothes.

Of every thousand passersby, I'd note one struggling to move legs or arms. These people have multiple sclerosis, a progressive autoimmune disease of the central nervous system. Their vision might blur when they read signs. Their legs might fail to cooperate when crossing the street. The worst cases, of course, wouldn't be out at all. They'd remain at home, perhaps in electric wheelchairs, maybe bedridden.

I'd note glucose monitors on one of every three hundred children frolicking in Central Park's playgrounds, children afflicted with autoimmune diabetes, which is usually childhood-onset.8 Their skin would bear

needle marks from the daily insulin injections required to avoid coma and death.

If my glasses came with headphones, I'd hear a cacophony of worry and desperation: asthmatic teenagers wondering if they'll be able to join friends in a game of baseball; more severe cases focused on walking slowly, so as not to lose breath; eczematics reminding themselves ceaselessly not to scratch, or if they've already scratched, berating themselves for the raw mess left behind.

Those with inflammatory bowel disease might be preoccupied with the pain, sometimes dull, sometimes sharp, that has characterized life since diagnosis. If it's not racking cramps on their minds, they'll likely be strategizing around bowel movements, which arrive all too frequently and with a painful urgency, and which sometimes contain blood. Those with MS might be wondering: How much longer before I can't walk? And everyone will regularly ask: Why can't doctors fix this? Where did this come from? Why me?

The National Institutes of Health estimate that between 14.7 and 23.5 million Americans have an autoimmune disease, or 5 to 8 percent of the population. The American Autoimmune Related Diseases Association puts the number at more than double that—50 million Americans. In the U.S., autoimmune disease ranks among the top ten killers of women. And that speaks to an omission I made for simplicity's sake in the above scenario. Roughly three-quarters of those afflicted with autoimmune disease are female. When I had my autoimmune glasses on, in other words, I'd be seeing mostly women.

Anthony Fauci, director of the National Institutes of Allergy and Infectious Diseases, once estimated that the direct and indirect costs of autoimmune diseases reached a staggering $100 billion yearly. (By comparison, we spend $57 billion on cancer and $200 billion on cardiovascular disease.) That may seem high, but bear in mind that autoimmune diseases, which are chronic in nature, generally strike in the prime of life, and require decades of costly symptom management.

These statistics apply to the richest countries in the early twenty-first century. But immune-mediated diseases weren't always this prevalent. Early hints of immune dysfunction during the late nineteenth century notwithstanding, the allergy and asthma epidemics gained steam during the 1960s, accelerated through the 1980s, and then plateaued by the early 2000s. In that period, depending on the study and the population, you'll find somewhere between a doubling and a tripling of asthma and allergies in the developed world.

Some autoimmune diseases show even more dramatic increases during the late twentieth century. A 2009 study found that the prevalence of undiagnosed celiac disease, a type of inflammatory bowel disease incited by proteins in grains, had increased more than fourfold since the mid-twentieth century.9 The incidence of multiple sclerosis has nearly tripled. And for some of these diseases, there's no end in sight. The incidence of type-1 diabetes, which more than tripled during the late twentieth century, is estimated to double again by 2020.

What has happened? In 2002, the French scientist Jean-François Bach published a seminal paper for anyone asking that question.10 The study, which appeared in the New England Journal of Medicine, had two graphs side by side, one showing the gradual decline since 1950 of once-common infectious diseases—hepatitis A, measles, mumps, and tuberculosis—next to another showing, over the same period, an increase of autoimmune and allergic disease in the developed world. Nearly everyone contracted mumps and measles in 1950. By 1980, almost no one did. Vaccines had almost eliminated both viruses. In an even shorter period—since 1970—new cases of hepatitis A infection fell to one-fifth their former level. And all the while, new cases of asthma, multiple sclerosis, and Crohn's disease doubled, tripled, and quadrupled, respectively.

Source: Bach, New England Journal of Medicine (2002).

The relationship that Bach so clearly demonstrates, that as infections decline over time, immune dysfunction increases, is evident between contemporaneous regions and populations. The incidence of allergic disease varies by a factor of 20 between the most allergic countries and the least. Vanishingly few children in Albania, for example, have allergy, but one-quarter of Australian children do.11 The incidence of type-1 diabetes varies even more markedly—350-fold between the most afflicted country, Finland, and the least, China.12 Are some ethnicities more vulnerable to these disorders than others? Maybe. However, when migrants move from low-risk to high-risk countries, the children born to them in their adopted homelands almost invariably suffer from immune-mediated diseases at rates equal to, and sometimes higher than, the local population. So, if not genetics, what explains the great disparity?

Epidemiologists used to assert that, generally speaking, these disorders increased as you moved from the equator toward the poles. In sub-Saharan Africa they were quite rare. In the U.K., they were all too common. And that seemed irrefutably true even thirty years ago. But evidence of a recent surge of asthma in countries like Brazil and Peru—and urban centers in the developing world everywhere—has undermined this once safely made generalization. Nowadays, you're more likely to hear that allergic and autoimmune diseases correlate with gross domestic product. And for now, that's holding true. The richer the country you call home—or in some cases, the higher your social class within a country—the more likely you are to have asthma, inflammatory bowel disease, and multiple sclerosis.

Critics discount these sweeping statistics for their reliance on questionnaires. Surveys are inevitably vulnerable to recall and cultural biases, they point out. But smaller studies that use objective measures such as wheeze and skin-prick tests, or testing for autoimmune antibodies, have repeatedly revealed the same basic pattern: Immune-mediated disorders arise in direct proportion to affluence and Westernization. The more that one's surroundings resemble the environment in which we evolved—rife with infections and lots of what one scientist calls "animals, faeces and mud"—the lower the prevalence of these diseases.13

BETWEEN THE STONE AGE AND THE NEOLITHIC, NO ASTHMA

In preparing for my Mexico trip, I often pondered another I'd taken, to a place where asthma didn't exist: the Bolivian Amazon. The anthropologists Michael Gurven from the University of California, Santa Barbara, and Hillard Kaplan from the University of New Mexico, Albuquerque, study a horticulturalist people living on the western edge of the Amazon basin. They're called the Tsimane, and they subsist, for the most part, directly off the jungle. They hunt monkeys, tapirs, and other animals with bows and arrows. (They happily use rifles, which some possess; but because they don't regularly participate in a cash economy, they often lack shells.) They fish with weirs, poison plants, and special arrows. And although they have plenty of contact with twenty-first-century Bolivians, their lifestyle is as close to Stone Age living as one can reasonably expect to find these days. That's why Gurven and Kaplan are here.

I caught up with Gurven, smiling, scruffy, and wearing a Phillies cap, at his clinic on the outskirts of a bustling, dusty town in the Bolivian lowlands called San Borja. Horses grazed in a nearby soccer field. Handsome, sand-colored cows wandered about. The occasional sow trotted by.

Gurven belongs to a school of anthropology called human behavioral ecology. The tools come from biology; the novelty is their application in anthropology. To hear him tell it, behavioral ecology emerged in reaction not to the cultural anthropology of the early and mid-twentieth century—Margaret Mead and her study Coming of Age in Samoa, for example—but to the period of anxious self-examination that followed. Was the very notion of studying humans imperialistic and exploitative? Could an outsider truly understand "the

other," or was she doomed to endlessly project herself on her study subjects?

Behavioral ecology, as applied to the study of people, as Gurven and his students explain to me around campfires during the coming nights, originates in a certain weariness, not necessarily with this self-questioning, justified as it may be, but with the retreat from even trying to comprehend those who inhabit different worlds. Yes, we inevitably project, but people who continue to live as we all once lived can teach us many things, and there are objective ways to measure these things. What's more, anyone interested in these lessons had better move fast. Whatever hunter-gatherers and horticulturalists remain in the world won't be at it for much longer.

Among the Tsimane, Gurven first studied human reciprocity and altruism, why people share in a world of limited resources. He asked questions like: How does a sick person get help in a world without health insurance? And why do people help the ailing when it costs them precious time and energy? He also explored how humans age under the more-or-less constant onslaught of infections. Even here, people live decades beyond their capacity to bear children. According to the most severe interpretations of Darwinian theory, that just shouldn't happen. But for Homo sapiens, it does. What are those extra decades for?

As part of his arrangement with the tribe, Gurven gives the Tsimane free medical care. He trucks them to his clinic from the remote villages along the tributaries of the Maniqui River. A doctor examines them. Technicians take stool, urine, and blood samples. In one darkened room, an ultrasound machine peers at their hearts and arteries. We'll revisit the specifics of Gurven's findings later, but, almost incidentally, he's found that the immune system of a horticulturalist living in the Amazon works differently than your average Londoner's or New Yorker's.

Over the past decade, Gurven's clinic has examined more than 12,000 people, almost the entire Tsimane population. In the 37,000 examinations conducted by his staff (they've seen many patients multiple times), no doctor has logged a single case of asthma.14 If rates approximated those in the U.S. and the U.K., you'd expect at least 1,000 asthmatics. As for autoimmune disease, he's seen fifteen cases—including eleven of vitiligo, a condition in which the immune system turns on pigment-producing cells in the skin, one of lupus, and one of rheumatoid arthritis. If autoimmune disease occurred with the same frequency here as in the developed world, he should have seen roughly six hundred cases. In Tsimanía, in other words, the prevalence of autoimmune disease is one-fortieth what it is in New York City.

What he does see are plenty of infections, which cause half of all deaths among the Tsimane. (Accidents and violence contribute an additional 14 percent.) And parasites are so universal as to be nearly unremarkable. There's lots of giardia and amoebiasis. A few have tuberculosis. Fewer still have a chronic flesh-eating parasite called leishmaniasis. And nearly everyone has hookworm.

He also sees plenty of the wear and tear that comes from an active life: prolapsed uteruses, the result of having many children (the average Tsimane woman has nine), and hernias from heavy lifting. But the diseases of civilization, including cancers of the breast, prostate, ovary, colon, and testicle, are absent. And so is cardiovascular disease.

Are the Tsimane special, genetically immune perhaps? Others studying unacculturated Amerindians in the Amazon have explicitly noted the same absence of allergic disorders, and the suite of diseases so common in modernity.15 Maybe Amerindians as a group are genetically invulnerable to these diseases. Perhaps, but not likely. Scientists have made similar observations among peoples in Europe, Africa, and Asia. The repeated observation is that people living in "dirtier" surroundings have less allergy and autoimmunity. The reverse holds true as well: Anyone seems able to develop asthma if exposed to the right conditions. And these

conditions prevail in places like New York City, London, and Sydney.

WHAT DOES A PLACE WITHOUT ASTHMA LOOK LIKE?

The day after I find Gurven, we drive an hour through cane fields and pasture to a red-hued river. We pile into a motorized dugout canoe, its sides shored up by planks. The month is August, the Southern Hemisphere winter, and it's chillier than one might anticipate for the jungle. A wind called el surazo—the southerly—blows off the vast pampas to the south. (Later I'll learn that this particular winter was so cold that fish and pink river dolphins washed up dead throughout Amazonia.)

After more than an hour of motoring past snowy white egrets, the same species that steps gingerly through the marshland of New York City's Jamaica Bay, we arrive at a Tsimane settlement called Chacal. "Gringolandia," Gurven says softly as several Coleman tents—Gurven's base camp—come into view. "The Tsimane don't live in tents."

There's no central village per se, just a freshly painted yellow school-house next to a field where the men play soccer nightly. The Tsimane live scattered along the river, each family or group of families tending fields of rice, corn, and manioc. Some credit their decentralized way of life with helping them resist Spanish influence. The would-be colonizers found no central authority to usurp, no priests or kings to co-opt. And the Tsimane simply retreated deeper into the jungle before the Spanish advance, which began in the seventeenth century.

Soon enough, we're walking along a narrow path running parallel to the river. As a clearing becomes visible through the underbrush, a Tsimane guide with a boyish face and solemn demeanor named Arnulfo makes a soft hooting sound. Gurven takes up the call as well. High-pitched and elongated like the last syllable of an owl's hoot, the cry serves as a kind of jungle courtesy, notifying those up ahead that we're approaching.

As we pass into the clearing, Gurven and Arnulfo announce their greetings in Tsimane. A group of young boys plays with tops carved from tree nuts. Hammered-in nails serve as points. The children stare at the newcomers expressionless at first, their brows in furrows, but they've seen outsiders before, and they quickly resume their game, winding string around their tops, and then setting their toys spinning with practiced yanks. Two women seated on a large woven mat return the greetings. A little girl lies prone in the lap of one woman, who searches patiently through her hair, extracting lice and nits, and crushing them between her teeth. The men are all gone for the day, we learn, on a hunting trip. We say our goodbyes—it's not good form to visit the women without men present, Gurven explains later—and continue walking.

We see fields of corn, lots of dogs, canoes, exquisitely woven mats, waist-high mortar-and-pestles, and everywhere tools made from jungle materials. It's this mastery of the jungle that strikes me, a twenty-first-century New Yorker with a computer-addled, Internet-spoiled brain, as most impressive. The Tsimane carve slim dugout canoes from tree trunks, and push them through the rivers with long poles. Mats are woven from palm fronds, as are the roofs on their huts. Useful trees and plants surround their jungle homesteads—papaya, banana, and a tutuma tree that bears large gourdlike fruits that they then fashion into bowls. They use ginger root to treat insect bites. They sleep on elevated platforms. As Gurven explains, here, one's worth doesn't derive from one's possessions, but instead from one's skills at extracting resources from the jungle. "You could lose everything, yes, but then you just build a new house, get fish, go hunting. Lots of individuals have that ability," he says. "There's a kind of freedom in that."

I could go on about how extraordinary Tsimane adaptations are, but really, I'm here to observe what I can't see directly: the hidden microbial and parasitological landscape. I want to know what that place where the

immune system doesn't malfunction looks like. And so how does it look? The answer is, alive.

To Gurven's chagrin, the Tsimane often draw drinking water directly from the muddy river. It's likely teeming with bacteria. Pigs, chickens, dogs, and the occasional pet spider monkey wander about freely. They each bring their unique blend of microbes. Tsimane women make an alcoholic drink by chewing and spitting boiled manioc and letting it ferment. In other words, they regularly imbibe what your average New York health food store touts as "live cultures." And of course, a majority has hookworms embedded in his or her gut.

In short, the Tsimane live in what scientists call "a living environment." Who cares? Much evidence suggests that surroundings like this protect against autoimmune and allergic disease, and for a simple reason: This is the type of environment the immune system has evolved to expect. And when it doesn't encounter the abundant stimulation contained herein, it falls into disarray.

Life here is not easy, of course.16 Infant mortality, which has improved since vaccinations arrived during the 1990s, remains high. One in five children dies before his or her fifth birthday. By age fifteen, an additional 5 percent have succumbed to disease. Essentially, one-quarter of all children born don't survive to adolescence, and that's an improvement over the early twentieth century. (On the other hand, two of every five Tsimane live to age sixty, one of Gurven's central and somewhat counterintuitive findings.) Despite the ubiquity of infectious and parasitic disease, however, the Tsimane do not appear sickly or starving. They're often missing several front teeth, a result of their fondness for sugarcane and citrus fruit, says Gurven, but otherwise, they seem robust and healthy.

On our return trip, we'll motor down the river, and drive through cane fields on muddy dirt roads. To return home, I'll take a small plane from San Borja over the imposing wall of the Andes to the west, spend a layover in the nation's capital, 12,000-foot-high La Paz, and then head back to New York City via Miami in a jet.

That trip passes through a well-defined gradient of allergic disease. I'll have traveled from an area of nonexistent allergies (subsistence living in the jungle) to one of slightly higher (the no-frills Bolivian town) to one of even higher (a large city in a developing country) to a place with the highest allergy prevalence of all (a large city in the developed world).

The gradient I just described in space also exists in time. If you retrace your own lineage back a few generations, you'll probably find hay fever and asthma lessening with each one. You (like me) may have lifelong asthma and food allergies, for example. Your parents, meanwhile, maybe had seasonal hay fever. But relatively few of your grandparents' generation—or great-grandparents, as the case may be—suffered from sneezing or wheezing of any sort.17 This pattern likely relates not to new exposures, but to the removal of old ones—exposures of the sort still prevalent in Tsimanía.

Repeated observations like these, backed by piles of experimental evidence indicating that the immune system responds differently depending on its history of exposures, have prompted some immunologists to question the basic assumptions underlying their field. Our understanding of the immune system rests on work mostly carried out during the twentieth century, but by that time, we were living in evolutionarily novel circumstances. In other words, we may have made a mistake equivalent to studying and cataloging an exotic-seeming ecosystem, only to discover that we weren't in the jungle at all; we were actually at the Bronx Zoo.

Or as the Duke University scientist William Parker puts it, "We as immunologists are now faced with the unsettling realization that the immune system we have spent all of our effort and energy studying

over . . . the past fifty years has turned out to be dramatically different than the system derived by natural selection."18

And that brings us to the heart of the matter.

UNDERSTANDING THE IMMUNE SYSTEM AND ITS DISCONTENTS

You've probably heard peripherally about the many allergens, such as dust mites, peanuts, and tree pollen, which cause allergies. Maybe you've heard reference to the infections and toxic pollutants that provoke autoimmune disease. Without suggesting that these ideas are totally unfounded, here's an alternative and much simpler model for engendering immune dysfunction. To produce these disorders, you don't need to add something new to your body. All that's necessary, in fact, is the removal of a single critical component of the immune system, and the human organism will collapse in a firestorm of autoimmune and allergic disease.

Immunologists learned this lesson from real-life case studies. In 1982, scientists at Oregon Health Sciences University in Portland described the case of an infant who'd died from multiorgan autoimmune disease—type-1 diabetes, thyroiditis, eczema, diarrhea, and a self-destructive immune response to viral infection.19 Seventeen other male infants from the boy's extended family had perished the same way, but no girls. The scientists suspected they had a genetic mutation in the X chromosome on their hands.

Boys have only one X chromosome, from Mom. So while girls, who have an X chromosome from each parent, can always refer to workable instructions in their second X chromosome, boys are stuck with whatever defective genes their single X chromosome contains. These boys had apparently inherited a gene that precipitated an immune-system meltdown.

Two more decades passed before geneticists identified the culprit. The gene was named FOXP3 (forkhead box P3 in its full ungainliness).20 When switched on, FOXP3 changed how white blood cells operated, turning them from aggressors into peacekeepers. In the case of those boys, a spontaneous mutation had disabled the gene. As a result, they couldn't restrain immune aggression. They went thermonuclear on invaders, causing severe collateral damage. And they couldn't tolerate even their own tissues. Mystery solved. Case closed. Except that the finding upended the current understanding of the immune system.21

For decades, immunologists had envisioned a system that avoided attacking the self by deleting self-reactive immune cells, and by employing the molecular equivalent of a hall pass system. Cells that belonged—"your" cells—displayed a unique badge (called the major histocompatibility complex, or MHC). Invaders didn't have this badge, and patrols picked them off handily. But here we had cells that possessed the mark of belonging, and were attacked anyway. What's more, healthy individuals tolerated a teeming community of microbes in the gut, organisms that didn't display the requisite hall pass but nonetheless escaped notice. Clearly, the old ideas needed revising.

Scientists, meanwhile, experimentally produced a range of autoimmune disorders by doing exactly what the FOXP3 mutation had done—disabling or hindering peacekeeping cells. Self-directed white blood cells obviously existed in healthy animals; they were a natural part of a functioning immune system. Order was maintained not by destroying these cells, but by restraining them. Disease arose not because lunatic lymphocytes escaped extermination (the old thinking), but because ineffective or absent suppressor cells failed to rein them in. The allergic and autoimmune diseases bedeviling us in modernity stemmed from a failure to police the police.

By the late 2000s, a revised model had emerged. Soon after birth, a wave of autoimmune cells populated the organism. They helped in defense, anticancer immunity, and tissue repair. A wave of peacekeeping cells quickly followed these initial pioneers, restraining them and establishing equilibrium. But keeping the peace in the long run required more suppressor cells. This secondary squadron emerged only after contact with the outside world—with certain parasites and microbes. This dependence was truly weird. It meant that our ability to self-regulate, to maintain homeostasis, was oddly reliant on external stimuli. What a design flaw—unless you considered the human organism in its proper context.

By all measures save sheer size and weight, you're mostly not you at all. The commensal bacteria in your gut, maybe 3 pounds worth, outnumber your cells by ten to one. The collective genome of this microbial community is a hundred times larger than yours, a hefty novel to your trifold pamphlet. That community harbors representatives from the three major branches of life on earth: bacteria (prokaryotes), yeasts (eukaryotes), and archaea (microorganisms that inhabit, among other extreme niches, deep-sea hydrothermal vents). You are really an ecosystem, a mutually dependent aggregation of life-forms, what scientists call a superorganism.

Now the reliance on "external" inputs makes a little more sense. How could your genetic self—the You that began when Dad's sperm fertilized Mom's egg—possibly ignore the voice of the majority? The seemingly absurd mistake that prompts immune-mediated disease makes a little more sense as well. Remove or change those stimuli, and of course you'd expect the immune system to lose its bearings. Those signals both guide and stabilize your immune function.

And that, unfortunately, is the story of the past century—the reason some think that the human immune system now malfunctions so spectacularly. We routinely fail to tolerate everything—innocuous proteins (allergies), our own tissues (autoimmune disease), and our commensal flora (inflammatory bowel disease)—because we've done environmentally what that FOXP3 mutation did genetically. By changing our inner ecology, we've hobbled the critical suppressor arm of our immune system.

So here's the question: Can we replace these stimuli? Can I take what's protective about the Tsimane environment and reintroduce it to mine? And can I do it without killing myself in the process, without losing the unprecedented improvement in both quality and length of life that characterizes the developed world?

INFESTED WITH WORMS IN MEXICO

And that brings us back to my impending experiment. I pull off the highway into a eucalyptus-lined parking lot where I'll meet my hookworm donor, a medical school dropout named Garin Aglietti. Warehouse-sized outlets of major American brands—Marshalls, Nike, Levi's, McDonald's—surround us. I join a group of forlorn-looking elderly people waiting under a tent. A bus passes by here to ferry them across the border. They belong, I presume, to the daily migration of Americans who travel to Mexico to buy cheap drugs.

Aglietti arrives in a tan Jeep Cherokee with Nevada plates. He's wearing baggy jeans, a blue shirt, and silver-rimmed wraparound sunglasses. He removes them to reveal blue eyes in a round, open face. In brief, Aglietti's story goes like this: In the 1990s, he developed psoriasis, an autoimmune disorder of the skin. He'd also suffered from asthma for most of his life. Mostly he fretted over the conditions known to accompany psoriasis, such as cardiovascular disease and autoimmune arthritis. All-too-frequent chest pains incited a cascade of worry. "I felt like it was killing me," he tells me. "I was way too young to be getting chest wall pain."

Allopathic medicine—also known as modern medicine—didn't offer much by way of treatments. Then in the

early 2000s, Aglietti heard about a Japanese scientist named Koichiro Fujita. Working in Borneo in the 1990s, a time when Japanese children seemed increasingly prone to developing eczema, Fujita had noticed that Bornean children had exquisite skin and no allergies. They also harbored plenty of parasites. Was there a link?

Back in Tokyo, Fujita took the extraordinary step of self-infecting with tapeworm. His hay fever cleared up. His skin became clearer and less muddled. He started preaching that the modern world was too clean for our own good. Corporate funders began withdrawing support from his lab.

Aglietti decided to follow Fujita's lead. Tapeworms have an intermediate and definitive host. In the former, they form a cyst; in the latter, they live as an intestinal worm. In 2005, Aglietti traveled to Kenya, toured cattle slaughterhouses searching for tapeworm cysts, found two, and swallowed them. Soon thereafter, Aglietti's psoriasis plaques softened. A few months later, they'd almost entirely disappeared. But once a tapeworm matures, it begins releasing rather large, semi-self-propelled egg-filled sacks called proglottids. They slither out one's rear and down one's leg in search of new intermediate hosts.

When they began passing, Aglietti felt as if sweat were dripping down his leg in the absence of any perceivable heat. "It's just a very unclean feeling psychologically," says Aglietti. "I just couldn't deal with it." He terminated the experiment with antiworm drugs. After passing a three-foot-long tapeworm, he set off in search of another, less psychologically disturbing parasite. This time, he settled on hookworm. Now he sells hookworm to others in Tijuana.

As we walk along the highway toward Mexico, Aglietti asks me almost gingerly why, with my apparently extensive knowledge of parasites, I didn't travel to some corner of the developing world, as he did, and acquire parasites naturally. I don't have the time, I say. But as we pass through turnstiles into a walled corridor, the no-man's-land that separates the two countries, I'm wondering the same thing.

No doctor or scientist I've yet met would recommend traveling to Tijuana to acquire hookworm. Not only is this approach completely outside the realm of what's proven to work scientifically, those like Aglietti who offer the service—at least two operations exist as of this writing—do so outside the scientific and medical establishment. No standards of quality or care exist save those that are self-imposed. And there's just as little accountability if anything goes wrong.

The cons of what I'm about to do are therefore significant. Illness and death are the most obvious. But I'm most worried about encouraging Aglietti, who seems perfectly nice, and his ilk. I'm not sure they deserve more attention than they've already received. On the other hand, self-infecting with hookworm has become an underground phenomenon of sorts, an unconventional treatment for often desperately ill people. I want to see what these individuals go through, how the process works.

And that brings me to the pros: I've heard fantastic tales of remission from people who've come this way before. Some I can confirm. Many more I cannot. There's nothing like seeing with your own eyes to settle questions like these. The potential benefits are also considerable—not worrying about peanuts, not wheezing, no more hay fever, no red, swollen eyes when cats jump in my lap. Sprouting a full head of hair would really be icing on the cake. Most important, success might point the way toward the Holy Grail of prevention—not for me, but for my children.

We pass through another revolving gate that's strangely reminiscent of the unjumpable, floor-to-ceiling turnstiles in the New York City subway, and we're suddenly at a small plaza with a fountain in Mexico. No more American chain stores. Small shops with colorful signs dominate. A friendly young man with thick

black eyebrows and hair gelled into spikes pulls up. He drives us to a neighborhood near the ocean. We park in front of building with a Mexican flag waving from the second-floor balcony. A sign says UNIDAD DE MEDICINA HOLÍSTICA—Office of Holistic Medicine.

While Aglietti confers with the doctor upstairs, our driver, Andrés, the doctor's son, tells me he's twenty years old, and was just admitted to medical school. He adds that his lifelong asthma forced him to stop playing sports years ago. Some months ago he infected himself with hookworm, and now it's much better. He began playing soccer again.

Aglietti returns and tells me the doctor is ready. I follow him to a clean, spare office on the second floor. A T-shirt with SAY HELLO TO MY LITTLE FRIENDS over an image of a gaping hookworm maw—four flat fangs lining the top, and vague indents where the eyes should be—is pulled over the back of a chair. The four "teeth" suggest Ancylostoma duodenale, generally considered more pathogenic than the Necator americanus I'll receive today. N. americanus has just two teeth that are boxier and, somehow, less sinister-looking.

Dr. Jorge Llamas enters, dressed in black slacks and blazer, and worn black loafers. He has a paunch, jowls, and a robust head ringed with trimmed white hair. Others who've passed this way have expressed great affection for him, and I can see why. He projects an easygoing, friendly manner that's reassuring and soothing.

"We're divorced from nature," he tells me. "And it's hurting us." He relates a story about an American woman who, after having lived in Acapulco for a time, returned to the U.S. to find she'd acquired parasites. She had them removed. Suddenly she was diagnosed with Crohn's disease. He mentions that as a child in Guadalajara, his father took him to the beach often, where swarms of mosquitoes fed on him. "It made my immune system strong," he says. He's never had allergies. He rails against the modern obsession with cleanliness. Everyone is mindlessly following the U.S.'s lead, he says. And everyone is getting U.S. diseases. "We need to stop and think."

He ends his holistic doctor spiel to take my medical history. Do I wake up at night? (Yes.) How many times, and what happens when I do? (I go back to sleep.) Just go back to sleep? (Yes.) How often do I exercise? (Three times a week.) What's my religion? (None.)

"That must be a lonely existence," he says, and notes something on my chart. He begins explaining the "known" emotional states associated with asthma and alopecia—stress and depression, respectively. "We create our own realities," he says at one point. "We're even creating this reality right now."

As we continue drifting into what I'm fairly sure is pseudoscience, I grow disconcerted. I'm here to acquire parasites, which is among the stupider things I've done. But the experiment is meant to probe what I imagine are universal principles of systems biology—relationships forged over mind-numbingly long periods of coevolution. None of which is hocuspocus. I attempt to correct course. I ask to see Aglietti's blood work. I've found no evidence that hookworms can transmit viruses between people, but they're born as eggs in one human's bowel movement and, after hatching into larvae and piercing the skin, they pass directly into another human's bloodstream. The precautionary principle applies.

Soon we're shuffling through a year or two's worth of tests. I confirm that Aglietti is clear of the major viruses—HIV, cytomegalovirus, hepatitis—as well as Strongyloides stercoralis, a nasty worm that, uniquely among soil-transmitted helminths, can reproduce in the host. I'm as satisfied as I'm going to be.

"Are you nervous?" asks Llamas.

“Do I look nervous?”

He shrugs. “A little.”

We move to a room at the back of the building. Aglietti has donned a light blue doctor’s overcoat with Worm Therapy embroidered in script over his right pectoral. He’s smiling and seems excited. With a pipette, Llamas removes what I’m assuming is larvae-laden water from a beaker, and squirts it onto an absorbent bandage. Given my apparent health, Aglietti and Llamas have recommended thirty worms, not the twenty or twenty-five I was assuming.

The bandage goes on. Within a minute, I feel a tickling, itching, nearly burning sensation—rather like a mild case of stinging nettles. That’s the microscopic larvae burrowing through my skin. Before anyone knew a parasite caused it, the distinctive itch had gained notoriety around the world, earning monikers like “ground itch,” “miners’ itch,” “water pox,” or the more poetic “dew poison.” Now scientists understand that hookworm larvae leave their outer cuticle, discarded inside-out like a sock, embedded in your skin. Your immune system responds savagely. But the now-naked larvae are already long gone.

Each larva will find its way into a capillary, and hitch a ride on my venous blood flow, like rafters on a river. They’ll pass through the thunderous pump of my heart, which causes me no small degree of anxiety. And once they’ve arrived at the capillaries of my lung, they’ll burrow out of the circulatory system, into the bunch-of-grapes-like sacs called alveoli. They’ll then follow the coordinated sweeping motion of millions of hair-like cilia up- and outward—the so-called mucociliary escalator—over the pharynx, where windpipe and food pipe branch, and plunge down into the esophagus.

They’ll miraculously survive the hydrochloric acid bath of my stomach and finally—after an odyssey through my body lasting several weeks—arrive at my small intestine, the final destination. They’ll latch on to my intestinal wall. They’ll mate. Large individuals will reach a centimeter in length. The females will lay perhaps 10,000 microscopic eggs daily, all the while grazing on intestinal tissue to the tune of 0.04 milliliter of blood per day. Assuming they all survive, that’s eight drops for every ten worms, or twenty-four drops daily paid to host a thirty-strong colony—not much, but not nothing either. And they can live for five years, maybe longer. The eggs, which require a week or two in tropical conditions to become infective larvae, will pass out with my stool—which, in New York City, means they end up in a wastewater treatment plant.

I might get a mild cough in a week or so, Aglietti explains. Flulike symptoms are common. Then “epigastric pain” once the worms attach. If I start coughing, I shouldn’t spit out the discharge.

“Swallow it,” he says. “That’s your medicine.”

Then Aglietti, who’s periodically glanced at his wristwatch since the bandage went on, says, “Okay, we’re past the possibility of anaphylaxis.” He’s referring to a potentially fatal allergic reaction usually associated with bee stings or, these days, peanuts. Anaphylaxis is treated with a shot of Adrenalin, which he has handy. Llamas hands me a box containing three pills of mebendazole, a deworming drug. “This is your out,” he says. “Here in Mexico we take two. But in the U.S., being the U.S., they take three.”

By now I have a headache. I’m filled with feelings of disgust, hope, and wonder—disgust with myself for agreeing (with myself) to this experiment; hope that the experiment may do some good; and wonder at the parasite’s biology, its ability to pierce skin, navigate circulatory systems, and, in the coming weeks, arrive at my small intestine. Underlying these sentiments is a recently acquired, quasi-religious faith in evolution—confidence that the organism knows what it’s doing, and won’t kill me in the process. For an

obligate parasite, a dead host is, after all, a useless host. For better or worse, we’re now in this together.

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