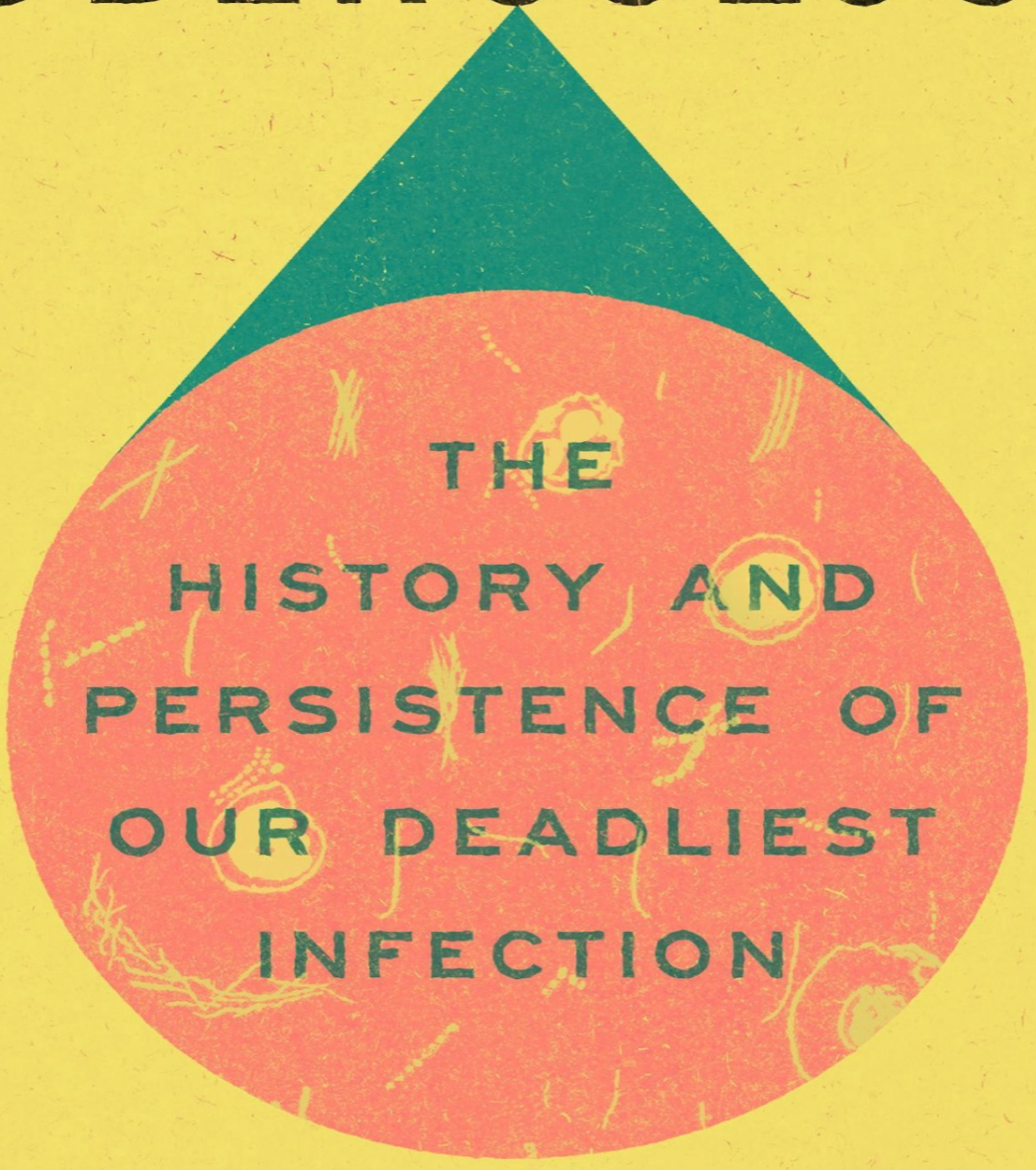
EVERYTHING IS TUBERCULOSIS



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EVERYTHING IS TUBERCULOSIS

THE HISTORY AND PERSISTENCE OF OUR DEADLIEST INFECTION

JOHN GREEN

CRASH COURSE BOOKS

CRASH COURSE BOOKS
An imprint of Penguin Random House LLC
1745 Broadway, New York, New York 10019

First published in the United States of America by Crash Course Books,
an imprint of Penguin Random House LLC, 2025

Visit us online at PenguinRandomHouse.com.

Library of Congress Cataloging-in-Publication Data is available.

Ebook ISBN 9781101592410

Cover design by Grace Han
Edited by Julie Strauss-Gabel
Design by Anna Booth, adapted for ebook by Andrew Wheatley

The authorized representative in the EU for product safety and compliance is Penguin Random House Ireland, Morrison Chambers, 32 Nassau Street, Dublin D02 YH68, Ireland, https://eu-contact.penguin.ie.

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This book is dedicated to
Shreya Tripathi, Henry Reider,
and TB Fighters everywhere

INTRODUCTION

GREGORY AND STOKES

AROUND THE TURN OF THE nineteenth century, the Scottish tinkerer and chemist James Watt began working on a new project.

He had already achieved fame and success for making steam engines more efficient, helping to fuel the industrial revolution that would radically reshape human history. The steam engine would lead to everything from air-conditioning to air travel to AirPods, while also unleashing over a trillion tons of carbon dioxide into the atmosphere, reshaping the planet's climate. Watt's innovation carried within it so much power that we named a measurement of power after him. Watt also made other important contributions to the human collection of tools and knowledge, inventing a machine that could copy sculptures and developing new strategies for manufacturing chlorine to bleach textiles.

But Watt hoped this new project would be his most important yet. He became obsessed with finding some kind of chemical solution to treat the lung disease known to physicians as phthisis.

Watt's daughter Jessy had died of phthisis at the age of fifteen in 1794. And now his son, Gregory, was ill with the disease, suffering from the classic symptoms of a persistent cough, night sweats, fever, and the physical wasting of the body that gave the disease its colloquial name: consumption. Gregory was in his early twenties, a skilled orator known for being phenomenally attractive—one friend described him as "literally the most beautiful youth I ever saw."

In a furious attempt to save Gregory, Watt helped invent a device that delivered nitrous oxide to the lungs, believing that shifting the amount of oxygen available to the body might help it heal. But the treatment proved unsuccessful. After many years of suffering, Gregory died of consumption in 1804 at the age of twenty-seven.

By 1900, phthisis had come to be known by a new name: tuberculosis. My great-uncle Stokes Goodrich was born that year in rural Tennessee. He was raised in a wood-frame house built by my great-grandfather Charles, a country doctor who rode his horse night and day around Franklin County delivering babies and dispensing medicine.

Stokes was a sickly child. In those days—and in these ones, too, I suppose—it was common to connect illness to some kind of deficiency, failure, or past mistake. A physician might conclude, as one German doctor did in the early eighteenth century, that a woman's life-threatening illness was brought on "by a dog which barked loudly at her." For baby Stokes, being given coffee and sweets by a family friend was thought to be the inciting incident. Thereafter, Stokes "developed the worst case of typhoid fever I ever saw recover," my great-grandfather later reported in a short memoir he wrote for our family.

In 1918, when Stokes was eighteen, he again nearly died during the Great Influenza pandemic when he became ill while working at a munitions factory. He survived, and in 1920 went to work for Alabama Power and Light, laboring as a lineman. As the 1920s progressed, Stokes experienced frequent bouts of what he hoped might be bronchitis. But the stubborn cough would not go away, and eventually, after coughing up blood, he sought medical attention.

Here is how my great-grandfather reported what happened next: "Stokes went to see a fine doctor in Gadsden, Alabama, who X-rayed him and discovered tuberculosis in the apex of his right lung. The X-ray

technician who made the film told me, ‘Dr. Goodrich, your son has miliary tuberculosis, and I have never seen a case that lived over two months.’ ”

Stokes was placed in a sanatorium in Asheville, North Carolina, one of many American cities that functioned as a tuberculosis colony of sorts. “Stokes had the best of care in the sanatorium but steadily grew worse, and on May 18, 1930, passed over the river to his Lord.”

My great-uncle was twenty-nine years old. I often wonder what it must have been like for my great-grandfather, having trained as a doctor, to be unable to save his own son from disease.

We are powerful enough to light the world at night, to artificially refrigerate food, to leave Earth’s atmosphere and orbit it from outer space. But we cannot save those we love from suffering. This is the story of human history as I understand it—the story of an organism that can do so much, but cannot do what it most wants.

Now we are two centuries removed from the deaths of Jessy and Gregory Watts, and nearly a century removed from the death of my great-uncle Stokes. Still, over a million people died of tuberculosis in 2023. That year, in fact, more people died of TB than died of malaria, typhoid, and war *combined*.

Just in the last two centuries, tuberculosis caused over a billion human deaths. One estimate, from Frank Ryan’s *Tuberculosis: The Greatest Story Never Told*, maintains that TB has killed around one in seven people who’ve ever lived. Covid-19 displaced tuberculosis as the world’s deadliest infectious disease from 2020 through 2022, but in 2023, TB regained the status it has held for most of what we know of human history. Killing 1,250,000 people, TB once again became our deadliest infection. What’s different now from 1804 or 1904 is that tuberculosis is curable, and has been since the mid-1950s. We know how to live in a world without tuberculosis. But we choose not to live in that world.

In 2000, the Ugandan physician Dr. Peter Mugyenyi gave a speech about the rich world's refusal to expand access to drugs treating HIV/AIDS. Millions of people were dying each year of AIDS, even though safe and effective antiretroviral therapy could have saved most of their lives. "Where are the drugs? The drugs are where the disease is not," Dr. Mugyenyi said. "And where is the disease? The disease is where the drugs are not."

And so it is with TB. This year, thousands of doctors will attend to millions of TB patients, and just as my great-grandfather could not save his son, these physicians will be unable to save their patients, because the cure is where the disease is not, and the disease is where the cure is not.

This is a book about that cure—why we didn't find it until the 1950s, and why in the decades since discovering the cure, we've allowed over 150,000,000 humans to die of tuberculosis. I started writing about TB because I wanted to understand how an illness could quietly shape so much of human history. But along the way, I learned that TB is both a form and expression of injustice. And I learned that how we imagine illness shapes our societies and our priorities. James Watt understood consumption as a mechanical failure by the lungs to ingest the proper ratio of gases. My great-grandfather understood his son's sickliness to have been driven by ingesting coffee and sweets in childhood. Others would understand TB as an inherited disease that affected certain types of personalities. Still others would argue that the illness was caused by demon possession, or poisoned air, or God's judgment, or whiskey. And each of these ways of understanding tuberculosis shaped not just how people lived and died of TB, but also *who* lived and died of it.

Today, we understand tuberculosis as an infection caused by bacteria. TB is airborne—it spreads from person to person through small particles contained in coughs, sneezes, or exhalations. Anyone can get tuberculosis—in fact, between one-quarter and one-third of all living humans have been infected with it. In most people, the infection will lie dormant for a lifetime.

But up to 10 percent of the infected will eventually become sick, a phenomenon we call “active TB.” People are especially likely to develop active TB if they have a weakened immune system due to other health problems like diabetes, HIV infection, or malnutrition. In fact, of the ten million people who became sick with TB in 2023, over five million also experienced malnutrition. And because the disease spreads especially well in crowded living and working conditions like slums and poorly ventilated factories, tuberculosis has come to be seen as a disease of poverty, an illness that walks the trails of injustice and inequity that we blazed for it.

The world we share is a product of all the worlds we used to share. For me at least, the history and present of tuberculosis reveal the folly and brilliance and cruelty and compassion of humans.

My wife, Sarah, often jokes that in my mind everything is about tuberculosis, and tuberculosis is about everything. She’s right.

CHAPTER 1

LAKKA

WHEN I FIRST VISITED LAKKA Government Hospital a few years back, I did not really want to be there.

Sarah and I were in Sierra Leone, a nation of nearly nine million people in West Africa, to learn about the country's maternal and neonatal healthcare systems. At the time, Sierra Leone had the highest maternal mortality rate in the world, with around one in every seventeen women dying in pregnancy or childbirth, and we'd traveled there to learn about and share stories of people affected by the crisis.[*]

So our trip was supposed to be oriented around the global maternal mortality crisis, not tuberculosis, and by our last day in Sierra Leone, I was exhausted and ill. (I possess a somewhat fragile constitution when it comes to health, and also when it comes to most other things.) But a doctor we were traveling with asked us to visit Lakka with him. He assured us that Lakka, a facility supported by the global health nonprofit Partners In Health, was basically on the way to the airport, and he needed to consult with the staff about a few cases.

At the time, I knew almost nothing about TB. To me, it was a disease of history—something that killed depressive nineteenth-century poets, not present-tense humans. But as a friend once told me, "Nothing is so privileged as thinking history belongs to the past."

When we arrived at Lakka, we were immediately greeted by a child who introduced himself as Henry. “That’s my son’s name,” I told him, and he smiled. Most Sierra Leoneans are multilingual, but Henry spoke particularly good English, especially for a kid his age, which made it possible for us to have a conversation that could go beyond my few halting phrases of Krio. I asked him how he was doing, and he said, “I am happy, sir. I am encouraged.” He loved that word. Who wouldn’t? *Encouraged*, like courage is something we rouse ourselves and others into.

My son Henry was nine then, and this Henry looked about the same age—a small boy with spindly legs and a big, goofy smile. He wore shorts and an oversized rugby shirt that reached nearly to his knees. Henry took hold of my T-shirt and began walking me around the hospital. He showed me the lab where a technician was looking through a microscope. Henry looked into the microscope and then asked me to, as the lab tech, a young woman from Freetown, explained that this sample contained tuberculosis even though the patient had been treated for several months with standard therapy. The lab tech began to tell me about this “standard therapy,” but Henry was pulling on my shirt again. He walked me through the wards, a complex of poorly ventilated buildings that contained hospital rooms with barred windows, thin mattresses, and no toilets. There was no electricity in the wards, and no consistent running water. To me, the rooms resembled prison cells. Before it was a TB hospital, Lakka was a leprosy isolation facility—and it felt like one.

Inside each room, one or two patients lay on cots, generally on their side or back. A few sat on the edges of their beds, leaning forward. All these men (the women were in a separate ward) were thin. Some were so emaciated that their skin seemed wrapped tightly around bone. As we walked down a hallway between buildings, Henry and I watched a young man drink water from a plastic bottle, and then promptly vomit a mix of bile and blood. I instinctively turned away, but Henry continued to stare at the man.

I figured Henry was someone’s kid—a doctor, maybe, or a nurse, or one of the cooking or cleaning staff. Everyone seemed to know him, and

everyone stopped their work to say hello and rub his head or squeeze his hand. I was immediately charmed by Henry—he had some of the mannerisms of my son, the same paradoxical mixture of shyness and enthusiastic desire for connection.

Henry eventually brought me back to the group of doctors and nurses who were meeting in a small room near the entrance of the hospital, and then one of the nurses lovingly and laughingly shooed him away.

“Who is that kid?” I asked.

“Henry?” answered a nurse. “The sweetest boy.”

“He’s one of the patients we’re worried about,” said a physician who went by Dr. Micheal.

“He’s a patient?” I asked.

“Yes.”

“He’s such a cute little kid,” I said. “I hope he’s going to be okay.”

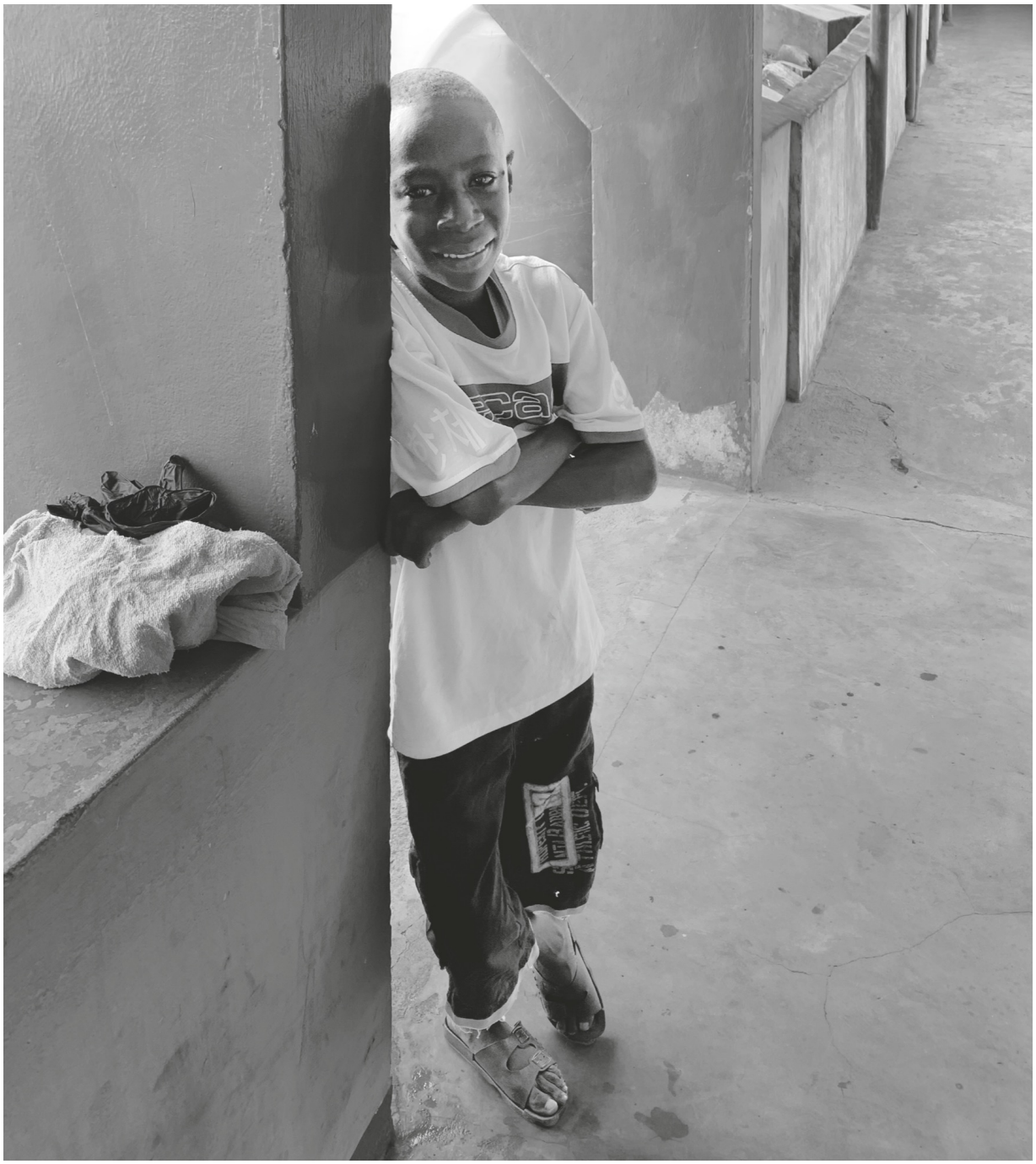
Dr. Micheal told me that Henry wasn’t a little boy. He was seventeen. He was only so small because he’d grown up malnourished, and then the TB had further emaciated his body.

“He seems to be doing okay,” I said. “Lots of energy. He walked me all around the hospital.”

“This is because the antibiotics are working,” Dr. Micheal explained. “But we know they are not working well enough. We are almost certain they will fail, and that is a big problem.” He shrugged, tight-lipped.

There was a lot I didn’t understand.

I saw Henry again as we were getting ready to leave. He was standing near the entrance to the hospital, and I asked him if I could take his picture. He said yes, and I photographed him a few times.



We scrolled through the pictures together. I tried to communicate that I was smiling from behind my mask. Henry wore no mask—his bacterial load was low enough that he posed no infection risk to others. As we chatted, I realized I was looking at him differently than I had when I believed him to be the son of a staff member. He no longer reminded me of

my nine-year-old son; now he was an emaciated young man. When he looked up at me, I saw yellow clouds in the whites of his eyes—a byproduct of the liver toxicity that frequently accompanies the treatment he was on. I noticed swelling on one side of his neck—which I would later learn is a telltale sign that TB has infected the lymph nodes. I asked him if he took medicine every day.

"Yes," he said. "I swallow them. Also they inject shot."

"Is that scary?"

His big eyes got bigger as he nodded.

Henry told me that the injections burned like a fire under his skin, and that the medications had many side effects, but the worst one was hunger. Active tuberculosis severely suppresses appetite, causing stomachaches and generally inhibiting the ability to eat, and once treatment commences and the infection begins to lessen, hunger roars back, which is a good sign—but only if one has enough to eat.

Years later, a young TB survivor told me about the hunger. I was at Lakka again, sitting in the immense shade of a mature mango tree, one of the only pleasant spots on the hospital grounds, which otherwise comprised patches of red clay and overgrown shrubbery. Three long, rough-hewn wooden benches were moved throughout the day to keep in the mango's shade. On the bench opposite me sat a young woman—we'll call her Marie—hunched forward, knees on elbows. Marie was so thin when she arrived at the hospital that she'd been unable to walk, and her chest X-ray revealed hardly any healthy lung tissue at all. She was five feet, three inches tall, and when she'd arrived at Lakka, she weighed less than seventy pounds.

Marie told me she dreamed night and day of eating as she recovered her health, that she thought of making mud soup and eating sticks. She thought about how crunchy they would be, imagining them as overstuffed with rich, soft nutrients inside. She could not think of anything but food, all the time.

Almost apologetically, a nurse sitting beside us said, “We feed everyone three times a day. Big meals. But it is not enough.” Not nearly enough, in fact, but the nurse explained that even three meals a day strained resources, because food was not considered to be an essential aspect of tuberculosis treatment, and so there was no funding for food. Some people became so hungry, she told me, that they left the hospital and stopped taking their medication, which increased the likelihood that the TB bacteria within them would continue multiplying, eventually developing resistance to first-line treatments. But they simply could not live with the hunger.

In Henry’s short, beautifully written memoir, he referenced hunger many times. He called Lakka “a place where hope and despair intertwined…. I found myself in a world where food was scarce, water was rationed, and clothing was inadequate for the chilly nights.”

After I first met Henry, I asked one of the nurses if he would be okay. “Oh, we love our Henry!” she said. She told me he had already gone through so much in his young life. Thank God, she said, that Henry was so loved by his mother, Isatu, who visited him regularly and brought him extra food whenever she could. Most of the patients at Lakka had no visitors. Many had been abandoned by their families; a tuberculosis case in the family was a tremendous mark of shame. But Henry had Isatu.

I realized none of this was an answer to whether he would be okay.

He is such a happy child, she told me. He cheers everyone up. When he’d been able to go to school, the other kids called him *pastor*, because he was always offering them prayers and assistance.

Still, this was not an answer.

“We will fight for him,” she told me at last.

SKIP NOTES

* Thanks to investments from Sierra Leone’s Ministry of Health in deep partnership with other organizations, maternal mortality in Sierra Leone declined by more than 50 percent in

the five years following our trip, a reminder that there is nothing permanent or unalterable about health inequities.

CHAPTER 2

COWBOYS AND ASSASSINS

AFTER I RETURNED FROM LAKKA to my home in Indianapolis, I began reading about the history of tuberculosis, which seemed to pop up everywhere from fashion to warfare to human geography, and I found that I simply could not shut up about the disease. Someone would mention New Mexico, and I'd jump in: "Did you know that New Mexico became a state partly because of tuberculosis?" Or, if a conversation turned to World War I, I'd respond, "Did you know that tuberculosis sorta kinda but not really caused World War I?" Or perhaps at a neighborhood Halloween party, I'd confront a ten-year-old dressed as a cowboy: "Did you know tuberculosis helped give us the cowboy hat?"

Which it really did, by the way: In the 1850s, a young man named John was living in New Jersey, working as a hatmaker, when he started coughing up blood. John visited the doctor and learned that, indeed, he had consumption. According to the prevailing wisdom of the time, his only real chance of survival was to head West.

The American West has long been associated with escape and freedom and last hopes. "West is where we all plan to go some day," Robert Penn Warren wrote in *All the King's Men*. "It is where you go when the land gives out and the old-field pines encroach. It is where you go when you get the letter saying: Flee, all is discovered." And it is where consumptives went to extend their lives.

In the nineteenth and early twentieth centuries, it was commonly accepted that consumption could be effectively treated by dry air, which

made a kind of sense—consumptive lungs seemed wet, and so did the humid, stagnant air in big American cities like New York and Baltimore, where consumption flourished. People fled to Arizona or New Mexico or California, which came to be known as the "land of new lungs." As one brochure boldly promised: "Come West and live."

Several cities, including Pasadena and Colorado Springs, were essentially created for consumptives and their families. But it wasn't only desert air that was mythologized. Doctors also recommended island air, or mountain air, or forest air, or Italian air. The justification for the so-called "travel cure" varied, except for one constant: Consumption thrived in cities, and so the solution must be rural. (This worldview was not unique to Europe and the U.S., although it was centered there. The Japanese poet Masaoka Shiki also traveled with consumption in the hopes of improvement.)

Now, our hatmaker John did not travel all the way to the West Coast, but instead headed from his home in New Jersey to the rough frontier town of St. Joseph, Missouri. It's hard to see how St. Joe's humid, stultifying air could be viewed as TB-friendly, but John ended up settling there for a while, and—wonder of wonders—began to feel better. For reasons we still don't fully understand, between 20 and 25 percent of people recover from active TB illness without treatment, and John was among that lucky minority.

Regaining his health over the next few years, John noticed something about the West: The hats sucked. Fur traders of European descent often wore bug-infested, brimless coonskin caps. Folks who made their way to Missouri from Texas and Mexico, meanwhile, tended to wear wide-brimmed straw hats that protected from the sun but leaked in the rain. So, after returning to the northeast with his consumption under control, John B. Stetson created a new sort of hat, which in time came to be known as the cowboy hat. (And even, sometimes, the Stetson.)[*]

I wasn’t kidding about New Mexico, either. Even after New Mexico became a U.S. territory in 1848, it was regarded with suspicion by many white Americans. After all, the majority of people living in the territory were Indigenous people or people who spoke Spanish as their first language. And so despite New Mexico having the institutions needed for statehood, a large enough population, and a strong majority of its voters seeking statehood, the U.S. Congress repeatedly turned down New Mexico’s attempts to fully enter the Union.

In order to please Congress, New Mexican officials realized they needed to recruit a larger white and English-speaking population, and thus began New Mexico’s quest to woo consumptives from the American Northeast and South with the promise of desert air, open skies, and world-class consumption care. That plan worked—by 1910, around 10 percent of *all* New Mexicans were tuberculosis patients, and with these new white residents, the U.S. Congress finally acquiesced, and New Mexico became the forty-seventh U.S. State in 1912.

Did TB cause World War I? Not really, but I would argue that it should be listed as a minor cause alongside the major ones—the alliance system of early twentieth-century Europe, the growing militarization and expansion of empires, etc.

As you may recall from history class, the Great War began after the assassination of the Austro-Hungarian Archduke Franz Ferdinand, which was—as assassinations go—somewhat farcical. On one side, we had the archduke’s wildly incompetent support staff, and on the other, a group of wildly incompetent assassins, half of whom were teenage consumptives.

In early 1914, the archduke and his wife made plans to visit Sarajevo, which was part of their Austro-Hungarian Empire—but a rather reluctant part. Many people living in the Balkan region wished to see their long-marginalized communities emerge as independent states, and among them were three teenagers from Belgrade—Nedjelko Cabrinovic, Trifko Grabez,

and Gavrilo Princip. All three were nineteen and connected with the Black Hand, a revolutionary group led by Serbian Army officers who wanted to liberate Serbia from the Austro-Hungarian Empire.

Cabrinovic, Grabez, and Princip were all seriously ill with tuberculosis. They knew they would die soon. As John Simkin put it, “They were therefore willing to give their life for what they believed was a great cause.” So when news spread that Franz Ferdinand would be touring Sarajevo (in an open-top car, no less), the three young men traveled there with the intention of assassinating the archduke. In Sarajevo, they joined up with three coconspirators, and each of the six was given a gun, bombs, and a cyanide pill, along with instructions to commit suicide once the archduke was dead.

The mission was an unmitigated catastrophe. Three of the men (the three without tuberculosis!) ended up unable or unwilling to act, but the boys from Belgrade were different. Cabrinovic was the first to see the archduke on the parade route; he threw a bomb toward Franz Ferdinand’s car but he missed, instead injuring several people in another car. Cabrinovic proceeded to take his cyanide pill, which contained too little cyanide to actually kill him, and then jump into a river where he hoped to drown, except the river ended up only being four inches deep, so he was quickly captured.

The archduke’s car took off after that, and the remaining conspirators gave up. At this point the archduke probably should’ve returned to his hotel after almost dying, but one of his companions convinced him to *continue driving around Sarajevo*, saying, “Do you think Sarajevo is full of assassins?”

Minutes later, their driver—who did not know the streets of Sarajevo well—took a wrong turn and stopped the car to shift into reverse. And where should that car stop except right in front of Gavrilo Princip. Princip shot both the archduke and Duchess Sophie to death, and then took his cyanide pill, which of course failed to kill him. Instead, he and all of his coconspirators were imprisoned. In the Austro-Hungarian Empire, it was illegal at the time to put teenagers to death, but the government didn’t need

a firing squad to kill them. All three would be dead before the end of the war, and all from tuberculosis.

It's fascinating to see the places where TB intersects with history, but there's risk in claiming that, for instance, Princip's consumption caused World War I, or that TB made New Mexico a state, and so on. Looking at history through any single lens creates distortions, because history is too complex for any one way of looking to suffice.

And anyway, while I'm fascinated by how TB shaped culture and history, what's most important to me is how culture has shaped TB. The infection has long exploited human biases and blind spots, wriggling its way through the paths injustice creates. Of course, tuberculosis doesn't know what it's doing, but for centuries, the disease has used social forces and prejudice to thrive wherever power systems devalue human lives—an experience that, back in Sierra Leone, Henry and his mother, Isatu, knew all too well.

SKIP NOTES

* Stetson would go on to live a long life and make a vast fortune, almost all of which he donated to endow schools, homeless shelters, and food banks.

CHAPTER 3

LOOK AT OUR RAILROADS

IT IS COMMON TO SAY that Sierra Leone is a poor country, but this is not the case. It is an exceptionally rich country with vast wealth in metal ores and especially in diamonds, which during centuries of colonialism encrusted many a British crown. After achieving independence in 1961, the new government struggled to transition away from this extraction-based economy. This was in part because systems to mine and export diamonds and minerals were more mature and robust than any other economic sector, and in part because independence didn't change the fact that many of the most valuable assets in Sierra Leone were (and still are) foreign-owned. Although the economy grew and life expectancy increased after independence, the country remained deeply impoverished.

The Sierra Leonean physician Dr. Bailor Barrie once told me, "If you want to understand why Sierra Leone is poor, you must look at a map of our railroads." And so I did. The map looks like this:

The railroads, built during colonial rule, did not connect people to each other. They connected the mineral-rich areas of Sierra Leone to the coast of Sierra Leone, where those minerals could be exported. The British Empire was in the business of resource extraction, and the systems built to support that business were resource-extraction systems. Were there schools? A few, to train servants of the empire. Were there clinics? A few, to heal servants of the empire. But the empire's role in Sierra Leone was primarily to take Sierra Leone's wealth, as quickly and efficiently as possible, *out* of Sierra Leone.

In the Global North, we still sometimes hear about the benefits of colonialism, how it brought roads and hospitals and schools to colonized regions, but this perspective is not supported by strong evidence. In 1950, life expectancy in Britain was sixty-nine. In Sierra Leone, after 150 years of colonial rule, life expectancy was under thirty, relatively similar to the life expectancy of premodern humans who lived five thousand or fifty thousand years ago. In general, colonial infrastructure was not built to strengthen communities; it was built to deplete them.

Sierra Leone was a British protectorate for centuries, but even before it was formally controlled by the British, it was terrorized by them. The historian Stephen Greenblatt introduced me to one account, written by a merchant named John Sarracoll, of a 1586–1587 voyage to Sierra Leone:

> The fourth of November we went on shore to a town of the Negroes…. It was of about two hundred houses, and walled about with mighty great trees, and stakes so thick that a rat could hardly get in or out. But as it chanced, we came directly upon a port which was not shut up, where we entered with such fierceness, that the people fled all out of the town, which we found to be finely built after their fashion, and the streets of it so intricate that it was difficult for us to find the way out that we came in at. We found their houses and streets so…cleanly kept that it was an admiration to us all, for that neither in the houses nor streets was so much dust to be found as would fill an egg shell. We found little in their houses, except some mats, gourds, and some earthen pots. Our men at their departure set the town on fire, and it was burnt (for the most part of it) in a quarter of an hour, their houses being covered with reed and straw.

This story of destruction and violence accelerated with the transatlantic slave trade. In the eighteenth and nineteenth centuries, roughly four hundred thousand people living in what is now Sierra Leone were kidnapped and sold into slavery, fueling terror throughout the region. Sometimes slave raiders broke into homes in the middle of the night and stole away entire families. Other times, children or young adults were kidnapped while hunting or gathering water. One young boy from the Mende community was kidnapped by Portuguese slave raiders while walking on a road known as the Kaw-we-li, or "War Road," because it was considered so dangerous. This boy was only around six years old when taken from his family and forced onto a slave ship. In adulthood, he went by the name Kaw-we-li, which some scholars theorize was likely the name given him by slavers: They called him after the road where they kidnapped him. It's possible that young Kaw-we-li lost the memory of whatever name his parents had given him, knowing himself only by the location of his kidnapping.

The slave trade directly caused millions of deaths, shut down trade routes, and upended social orders. Communities were devastated, not only because so many people were forced from their land and families, but also because most forms of economic activity—from traveling with goods to selling in a market—came with the risk of being kidnapped.

For Sierra Leone, the story is especially complex. In 1783, Britain emancipated some enslaved people who had fought for the British in the American Revolutionary War.[*1] At least four thousand newly emancipated soldiers were as a result relocated to Nova Scotia in Canada after the war. But many of the relocated emancipated people were unhappy in Nova Scotia, where they still faced discrimination alongside extreme weather, and so in 1791, a Black Loyalist leader named Thomas Peters made the case to colonial authorities that the community should be resettled in a new colony in West Africa. This became Freetown, which came to be the capital of the British-colonized territory of Sierra Leone. Over the ensuing decades, thousands more emancipated slaves or leaders of rebellions were

transported to Freetown, many of them Black Americans, Canadians, or Jamaicans. Their descendants are still known in Sierra Leone as Krios.

Beginning in 1807, the British Empire outlawed the slave trade (although individuals were still allowed to own slaves until 1833), and whenever the British Royal Navy found and captured a slave ship, the navy would "emancipate" the formerly enslaved and resettle them in Freetown regardless of their actual homes. And so Freetown grew, and with it, the city's vast diversity. The Krio people's language, also called Krio, contains many words from English but also from a huge variety of West African languages, and it is now the lingua franca of Sierra Leone. As Freetown became the economic center of the burgeoning colony, people from a huge variety of ethnic groups also came to the emerging city.

Henry, the boy I met at Lakka Hospital, had a Krio father—meaning that Henry's roots in the United States may stretch back much further than my own. (My people have only been in America since the late nineteenth century.)

It is true, and important to note, that many Sierra Leoneans *are* poor—indeed, according to the United Nations, 59 percent of people in Sierra Leone are "multidimensionally poor." But it is equally true that many Sierra Leoneans are not poor, that the country is economically and religiously and culturally diverse, and that any attempts to essentialize a nation of nine million people amounts to the kind of oversimplification that deceives via distillation. That noted, it's important to understand the nation has suffered repeated crises not primarily due to incompetence or corrupt officials (although both certainly played a role) but instead from the omnipresence of history.[*2]

Sometimes, folks try to explain Sierra Leone's impoverishment through the lens of pure physical geography—West Africa's rivers aren't long enough or navigable enough; there aren't enough good ports; the "resource curse" of mineral wealth causes economies to develop with a focus on

extraction instead of investment; etc. But these explanations overlook history. Until the fifteenth century, Europeans generally imagined West Africans to be rich and powerful. (Indeed, the wealthiest individual in human history was likely Mansa Musa, the fourteenth-century ruler of West Africa's Malian empire.) There is nothing inevitable or natural about the impoverishment of countries like Sierra Leone.

Henry's mother, Isatu, was born in 1968, seven years after Sierra Leone became an independent nation. She grew up in the southern Bonthe District. Her people were Mende, one of Sierra Leone's largest ethnic groups, and also one of the groups most ravaged by the Atlantic slave trade. Growing up, Isatu heard stories of the slave raids that continued long after the official abolition of slavery in the British Empire.

I want to be careful here not to imagine Isatu's life as merely poor, or merely oppressed, or merely anything. Yes, she grew up in a village where malnutrition was common, and she was often hungry, especially after her father's death when she was ten. But she remembers happy early years in Bonthe, "joy joy joy" when she went to school and attended church each Sunday.[*3] She also loved school (and excelled in it). She enjoyed being with her many friends, how there were always kids around who knew and cared for her. They invented games to play together, then fought over the rules of those games. When Isatu has described her childhood to me, I've found myself thinking about Nikki Giovanni's 1968 poem "Nikki-Rosa," which begins*:*

childhood remembrances are always a drag
if you're Black
you always remember things like living in Woodlawn
with no inside toilet
and if you become famous or something
they never talk about how happy you were to have

your mother
all to yourself and
how good the water felt when you got your bath
from one of those
big tubs that folk in chicago barbecue in

Once, when Isatu talked to me about her childhood, the interpreter used the word “woven.” “Myself and my friends were woven.”

I do not know what it’s like to grow up in a safe and stable village in southern Sierra Leone, but I know the joy of feeling woven into the social fabric, feeling a part of the world rather than apart from it, and that’s how Isatu has always described her early years to me. But then the war came.

In 1991, when Isatu was twenty-three, a long and truly horrific civil war erupted in Sierra Leone. The war would eventually claim the lives of over fifty thousand Sierra Leoneans and traumatize millions more. Part of the terror, Isatu explained to me, is that either side in the war could enter your village at any time, loot it, burn it, and massacre the community. The threat was not only personal death but communal death.

Isatu’s family, like many others, moved to Freetown, which was seen as insulated from the worst of the violence. During this period of relative safety in her life, Isatu met Henry’s father at a church service. Then, just as Isatu learned she was pregnant with Henry, the war came to Freetown. The rebels took over the city. “There was no particular place to stay because the war people would move around Freetown, killing people,” she told me. “I moved from one location to another, staying with friends and family. It was the hardest time of my life. I was pregnant, and I couldn’t find enough food. I couldn’t visit the clinic. Some days I didn’t have shelter at all. It took the grace of God to provide my survival.”

Henry was born at a clinic, but by the time his younger sister, Favor, arrived two years later, the clinics and hospitals in Freetown were so underfunded and understaffed that Isatu decided it was safer to give birth at home. When the war finally ended in 2002, Isatu was a thirty-four-year-old

mother of two young children. By then, her dreams of pursuing a college education had been left behind, and she worked in a market selling cooking oil and cleaning supplies alongside her sister.

When I first spoke with Isatu, she asked the name of my spouse and children. “Sarah, Henry, and Alice,” I told her. We were speaking through an interpreter, so I was surprised when she answered me in English: “Wife Sarah, child Henry, child Alice,” she said back to me. “I will pray for them.” Sometimes, when people tell you that you’re in their prayers, it sounds like lip service. But not when Isatu says it.

Skip Notes

*1 It’s worth pausing to consider what it means in the context of American history and our conceptions of freedom that Black people fighting for the British were far more likely to be emancipated than those fighting for an independent U.S.

*2 Corruption contributes to Sierra Leone’s poverty, but 1. Government incompetence is not unique to poor countries and 2. While corruption is a significant problem around the world, on a very basic level, Sierra Leone simply does not have enough money to build a functioning healthcare system. Countries like Germany and the U.K. devote about 12 percent of their total gross domestic product to healthcare. If Sierra Leone spent the same percentage of its economy on healthcare, the country would have about sixty dollars per person per year to spend on health, which wouldn’t pay for two months of my Lexapro prescription, let alone a functioning healthcare system.

*3 Around 70 percent of Mende people in Sierra Leone are Muslim, but interreligious communities and marriages are common. Isatu and her family are evangelical Christians.

CHAPTER 4

THAT WEALTH NEVER WARDED OFF

IT IS A STRANGE FACT of human history that we tend to focus so little on disease. In my college survey course about the history of humans, I learned of wars and empires and trade routes, but I heard precious little of microbes, even though illness is a defining feature of human life. As Virginia Woolf wrote in *On Being Ill*, considering "what wastes and deserts of the soul a slight attack of influenza brings to light…it becomes strange indeed that illness has not taken its place with love, battle, and jealousy among the prime themes of literature."

Some of this may be due to the nature of pain itself. As Barbara Duden has written, "Pain is in the body. It leaves no trace for the historian, unless complaints about it are recorded." But I wonder if we also ignore illness because of our bias toward agency and control. We would like to imagine that we captain the ships of our lives, that human history is largely the story of human choice. Perhaps this is why rumors have swirled for millennia that Alexander the Great died of poisoning even though he almost certainly died of typhoid or malaria. We simply don't want a world where even the most powerful emperor can be felled by mere infection. But history, alas, is not merely a record of what we do, but also a record of what is done to us.

We know consumption has been with us for a very long time. Mummies in ancient Egypt from five thousand years ago show the telltale deformities that accompany tuberculosis of the bone, wherein the bacteria scrape out

tiny holes in the skeleton, leaving bones that resemble dead coral. TB was one of the few infectious diseases present in both the Americas and Afroeurasia before the Columbian Exchange began in 1492; archaeological evidence indicates that TB was in the Americas at least two thousand years ago,[*1] and it has been present in China for at least five thousand years. But recent genetic evidence indicates that the story might go back much further—our species is perhaps three hundred thousand years old, but it seems that other species of hominids were being infected with consumption-like illnesses three *million* years ago. In fact, tuberculosis is listed in *Guinness World Records* as the oldest contagious disease.

In ancient China, TB was known by a term that translates to "lung exhaustion." In ancient Hebrew, TB was called *schachepheth*, meaning "wasting away," and is mentioned in the Tanakh. The famous Greek doctor Hippocrates wrote about TB, too, which as we've learned was known in Greek as *phthisis*, derived from a word meaning "to decay."

"Of all maladies," Hippocrates tells us, "it was the most virulent and the most difficult, and exacted the most deaths." Hippocrates advised his students not even to attempt treating phthisis, because they would inevitably fail, which would make them look like poor healers.

By 200 CE, a new Chinese term for consumption had emerged: *huaifu*, meaning "destroyed palace." A Chinese medical textbook at the time read, "Toxic Drugs bring no cure; short needles cannot seize [the disease]."

All these names—whether they focus on the destruction of the bodily palace or physical disappearance—reference an important facet of tuberculosis, which is weight loss and wasting caused by lack of appetite and extreme abdominal pain. This is also why TB was widely known as "consumption" until the twentieth century—it seemed to be a disease that consumed the very body, shrinking and shriveling it. Over eight hundred years ago, Daoist priests began referring to the illness as *shīzhài*, or "corpse disease," because the illness transforms a living being into a cadaver.

Unlike many other diseases, for most of human history, consumption appeared indiscriminate, killing the rich and the poor, the foolish and the brilliant. Charles Dickens called consumption the disease that "wealth never warded off," and indeed among its victims was the richest individual of the nineteenth century, Jay Gould. John Bunyan called consumption "the captain of all these men of death" for its ubiquity and severity. Victims of consumption included Henry VII of England, Paul Laurence Dunbar, Eleanor Roosevelt, Lin Huiyin, Simón Bolívar, Franz Kafka, Louis XIII of France, John Keats, Sultan Mahmud II, and all three Brontë sisters.

Because TB infects and kills so slowly, it does not resemble a plague that sweeps through a community, where one family member gets sick and everyone else in the household is sick a few days later. It also differs from an illness like cholera that affects mostly the destitute. And it wasn't like a cancer or heart disease that affects one person but rarely seems to spread. At times, consumption seemed to strike whole families; at other times, it seemed to strike randomly. (Louis XIII died of TB, for instance, but his spouse and children did not.)

Precisely because consumption was so fundamentally different from other infections, classical understandings of the disease varied widely. In ancient China, huaifu was generally understood to be contagious, but lifestyle factors could worsen it. (One book claimed you could get consumption "by overworking one's mind and exhausting one's energy, injuring one's *ch'i* and loosening one's sperm.") In ancient Greece, Hippocrates thought it to be an inherited condition, writing that "consumptives beget consumptives." Other Greeks, including the physician Galen, believed the disease to be contagious. In ancient India, consumption was understood to be caused by excessive fatigue, anxiety, and hunger. In other communities, it was seen to be the result of a curse, or a poison, or demon possession.

Some classical thinkers did even approach a germ theory of disease long before microscopy could confirm it. Around a thousand years ago, the Persian scholar and poet Ibn Sina wrote that tuberculosis and other illnesses were caused when the body was “contaminated by tainted foreign organisms that are not visible by naked eye.”

Even seven hundred years after Ibn Sina first proposed that consumption might be caused by invisible organisms, there were no effective treatments. Ibn Sina hailed the use of garlic in TB treatment, and garlic does have antimicrobial properties, but isn’t nearly strong enough to treat the disease effectively. Indian texts recommended, among other treatments, eating meat, drinking alcohol, and rest. Adequate nutrition and rest *are* good treatments for active TB infection, although they are most effective at preventing the emergence of active TB. In Europe, bloodletting was a common (and wholly ineffective) treatment; in the Americas, herbal remedies were used, some of which were useful if not curative. Global medical treatment for TB has ranged from rubbing buzzard fat on the chest to ingesting human milk, from animal sacrifice to acupuncture. Identifying effective treatment was made more difficult by the fact that people sometimes seemed to recover only to get sick again, or seemed to get sick after treatment only to recover later. Consumption made very little sense.

And at least in that respect, it hasn’t changed much. Tuberculosis is, on many levels, a weird disease. Infections can lie dormant for decades, or for a lifetime. The illness has an unpredictable course—it may kill its victims within a few months, or over many years, or not at all. Treatment can appear effective only for the illness to come roaring back for reasons we still don’t fully understand.

Much of this oddness is related to the infectious agent itself. Right now, over two billion people have been infected with a microorganism called *Mycobacterium tuberculosis*. This speaks to just how infectious tuberculosis can be: The average untreated case of active tuberculosis will spread the infection to between ten and fifteen people per year.[*2] One might acquire TB on a crowded city bus, or from lying next to a sick person at night, or working near them. Less commonly, we can also contract

tuberculosis from other mammals—by eating infected seal meat, or by drinking raw milk from infected cows.

M. tuberculosis is a near-perfect human predator in part because it moves very slowly. The bacteria has an uncommonly slow growth rate. While *E. coli* can double in number about every twenty minutes in a laboratory environment, *M. tuberculosis* doubles only about once *per day*. And so infections simply take much, much longer to make an infected person sick, as the number of bacteria remains lower, allowing the immune system lots of time to mount a defense against the pathogen.

But there's a problem: *M. tuberculosis* grows so slowly because it takes a long time to build its unusually fatty, thick cell wall, which is a formidable enemy to the immune system. White blood cells struggle to penetrate the cell wall and kill the bacteria from within. In fact, it's so hard for infection-fighting cells to penetrate the bacteria's cell wall that, instead, white blood cells usually surround it, creating a ball of calcifying tissue known as a tubercle.[*3] The TB bacteria can survive within these tubercles, replicating very slowly, consuming dead tissue as food. This type of infection, sometimes known as latent tuberculosis, will often last a lifetime without ever making a person sick. Most people infected with TB will never become ill because the tubercles will continue to hold the bacteria within them, preventing active disease from developing. But in 5 to 10 percent of infections, the immune system can't produce enough white blood cells to surround all the bacteria with tubercles, and *M. tuberculosis* is able to grow and grow within the lungs or elsewhere. The body is slowly overwhelmed by infection (and the immune system's resulting inflammation), eventually leading to death.

Most active tuberculosis illnesses occur within two years of initial infection, but sometimes the infection can lie dormant for decades before suddenly exploding into active disease. Often, the factors leading to active disease are clear—a compromised immune system from HIV, malnutrition, stress, or air

pollution all might trigger the disease into life. Immunosuppressive drugs that treat autoimmune disorders like Ulcerative Colitis can also cause TB infections to become active disease, which is why Americans often hear tuberculosis listed among potential side effects in drug commercials. But other times the causes are mysterious—something in the body's balance shifts, and slowly the body is overwhelmed with bacteria.

Once the disease becomes active, its course is extremely unpredictable. For reasons we don't fully understand, some patients will recover without treatment. Some will survive for decades but with permanent disability, including lung problems, devastating fatigue, and painful bone deformities. But if left untreated, most people who develop active TB will eventually die of the disease. Their lungs collapse or fill with fluid. Scarring leaves so little healthy lung tissue that breathing becomes impossible. The infection spreads to the brain or spinal column. Or they suffer a sudden, uncontrollable hemorrhage, leading to a quick death as blood drowns the lungs.

Let's turn back now to 1804, the year that James Watt's son Gregory died of tuberculosis. In May of that year, Napoleon Bonaparte was named Emperor of France; in July, Aaron Burr shot Alexander Hamilton. I understand if this all feels like ancient history, but it's really not. As of 2025, around 117 billion modern humans have lived. Over 100 billion were born before 1804. Almost everything that ever happened to us, and almost everyone who ever happened, happened before 1804.

And yet, human health was vastly different in 1804. Indigenous South Americans had discovered quinine bark as an effective tool for preventing malaria, and West Africans and Turkish peoples had developed inoculation as a strategy for reducing the risk of smallpox. There were effective remedies for some illnesses—plants with antimicrobial properties were widely used in traditional medicines, as were natural anti-inflammatory compounds (especially salicylic acid, which would eventually be used to

create aspirin). But relatively few high-quality health interventions existed, and the overall human life expectancy in 1804 was not much different than it had been a thousand years earlier.

Surgery was generally fatal, if not from the operation itself, then from bacterial infections that emerged days afterward. No city in the world had a good sewer system or consistent access to clean water. People who received treatment from doctors were not much more likely to survive an illness than people who were treated by faith healers, and most of what we now think of as “modern medicine” did not exist. Imagine a contemporary healthcare clinic, and then, one by one, remove the items that weren’t yet available in 1804: Of course there were no antibiotic medications to treat bacterial illnesses (the first antibiotic became available in 1945) or antiviral medications (1967). There were no antiseptics or knowledge of them, because no one knew microorganisms might cause disease.

There were no reflex hammers, those little tools used to test nerve conduction in the knees and elbows, until 1888. There were no otoscopes, the tool that uses light and magnification to visualize the eardrum, until the 1830s. The first stethoscope, an essential tool for listening to the heart and lungs and GI tract, wasn’t developed until 1816. There were also no X-rays (1895) and no blood pressure cuffs (1881), all of which meant there was really no way to see or understand the inside of a human body while that body was still alive.

Because the body was a wholly exterior phenomenon, European medicine saw great significance in the flow of various bodily fluids from the interior to the exterior. They believed the so-called “four humours” of blood, phlegm, yellow bile, and black bile needed to remain in balance for the body to be healthy. Illness was thought to be a dysregulation of those humours—too much or too little of one or another, and so the treatment for an illness might require bleeding via leeches, or shifting the amount of bile in the body by inducing vomiting, or instructing patients to spit out all their phlegm rather than swallowing it.

To try to understand the state of medicine in this era, I sometimes think of the eighteenth-century physician Johann Storch, whose patient histories

were the subject of Barbara Duden's brilliant book *The Woman Beneath the Skin*. In one passage, Dr. Storch tries to imagine what might've happened to a pin that had been swallowed by a tailor's assistant. "It could exit from nearly anywhere: from a lump next to the navel, through the urine, with the excrement, through the vulva, at the inside of the calf through an abscess, or after eighteen years through the leg." All of which is to say that three human lifetimes ago, a trained physician in Germany had no idea that the human body contained a digestive tract.

And so how did one arrive at a diagnosis? Largely through patient history and observation, which remain hallmarks of a doctor's visit today, even if we now have better ways of observing the body and its machinations. The classical physician was a kind of detective[*4] whose job was to listen carefully to a person's story, pay close attention to their appearance, and then use that information to identify a culprit.

By 1804, consumption was overwhelmingly the most common cause of death globally, and so healthcare providers around the world knew the classic symptoms: If a patient wakes up with their bedclothes so drenched in sweat they need to be wrung dry, that's a concern. Losing weight was another worrying sign, as was a persistent dry cough that eventually transitioned to coughing up phlegm. Doctors also considered consumption if a patient reported a long illness that had slowly become worse—losing the ability to run or walk over several months or years, or finding that a cough does not go away even after a year of treatment.

And then there is the blood. When a physician learned that a patient was coughing up blood, or even phlegm tinged with blood, consumption immediately became the most likely diagnosis. Blood in the sputum became such a diagnostic hallmark for consumption that even today entire plots can be woven around it. When we see the blood in Satine's handkerchief in *Moulin Rouge*, or Violetta's in *La Traviata*, or Velementov's in *The Great*, or Arthur Morgan's in *Red Dead Redemption 2*, we know that it portends their forthcoming tragic end. My favorite stand-up comedy routine about tuberculosis (yes, I have a favorite stand-up comedy routine about tuberculosis) begins with Naomi Ekperigin saying that the U.S. has become

so troubled that “if America was a character in a movie…this would be the part in the movie where America coughs into a rag and then pulls it away and sees blood.” The crowd erupts, because even now, when most Americans know very little about TB, they still know about the blood in the rag.

Today, we understand that these familiar symptoms are associated with tuberculosis of the lungs. But TB can also invade other parts of the body and express itself very differently. Classically, what we now understand to be TB was seen as several different diseases. From the pancreas to the spinal cord to the lymphatic system to the brain, a tuberculosis infection can cause a wide array of illness, from brain swelling (tuberculous meningitis) to the rupture of infected lymph nodes through the skin (scrofula) to tuberculosis of the bone, which can cause lifelong disability through destroying hip, spine, or limb bones. TB affecting the spine, known as Pott’s disease, is a common and terribly painful cause of a hunched back (the fictional hunchback of Notre Dame suffered from Pott’s disease).

Back in 1804, there was still disagreement over whether all these illnesses had the same cause, but upon autopsy, physicians did notice similarities between what consumption did to the lungs and what it did to the rest of the body. The telltale tubercles, those clumpy spheres of white tissue, appeared not just in the lungs but also in other organs. Like the needle swallowed by the tailor’s assistant, it seemed that consumption could end up anywhere for any reason.

SKIP NOTES

*1 It seems likely that seals infected with TB carried the disease from Afroeurasia to the Americas.

*2 The average case of influenza, meanwhile, spreads to between one and two further people. For Covid-19, it’s between 1.4 and 2.4.

*3 “Tubercle” describes a rounded, swollen shape. Potatoes are tubercular, for example.

*4 It’s no coincidence Sir Arthur Conan Doyle, who created the deductionist detective Sherlock Holmes, was—as we’ll learn later—a physician and tuberculosis researcher.

CHAPTER 5

WHIPPED AWAY

HENRY CRIED A LOT AS a baby. Isatu often worried that he was sick, not least because there was never enough food for him. But by the time Henry was three, he seemed healthy enough, and he had escaped the dangers of infancy. The war was over at last, and Henry loved playing with friends in their Freetown neighborhood—in fact, he got his preschool uniform dirty so regularly that Isatu felt she was perpetually washing his clothes by hand, or else fetching the water for washing. She remembered once when Henry was five years old, he and a friend rode a bicycle together down a hill, but the bike fell apart halfway down, and Henry landed headfirst in a drainage ditch during the peak of rainy season. She had to take him to the hospital after that one. He was a boisterous and wildly energetic child—which is why she knew something was wrong when he began to act more and more lethargic around the age of six.

Meanwhile, Henry's younger sister, Favor, really did seem to have favor on her side. She rarely struggled with illness, kept her school clothes immaculate, and was an excellent kindergarten student. Henry remembered waiting for her outside of school each day, and walking home with her, feeling like the protective older brother even though they weren't very far apart in age.

As Henry grew sicker, every part of Isatu's life grew difficult. Her husband—Henry and Favor's father—left their home, and although he continued to stay in touch and provide occasional support, Isatu felt a profound abandonment that he was living elsewhere. "I had many

challenges with money," she told me. "I went out to sell perfume and oils, but there was no one to buy them, and it made me discouraged. Whenever I had money, I invested it in Favor and Henry's schooling. I always paid their school fees. I knew there is no future without education. But with Henry sick, we had many constraints."

It started with that lethargy, and a cough. She took him to a clinic where they suspected tuberculosis, but the first tests came back negative,[*1] and so Isatu was told that he might have malaria or another illness. But he got worse—soon he was soaking through his bedding with sweat each night and feeling too ill to attend school. Finally, both he and Isatu were diagnosed with tuberculosis. They began treatment—the standard protocol of four different drugs, taken daily at a clinic and paid for by the Global Fund, which supports efforts to fight malaria, TB, and HIV/AIDS. The inconvenience of walking to the clinic each day, or else paying for transportation, further complicated their already precarious lives, but they kept up with treatment—for a time.

When Henry first got sick, he was treated—like almost everyone diagnosed with TB in poor countries—with the RIPE drugs,[*2] a combination of Rifampin, Isoniazid, Pyrazinamide, and Ethambutol. You probably don't need to remember the names of these drugs, but it's worth noting that all four of them are over fifty years old, and many forms of tuberculosis have developed resistance to one or more of the drugs, a condition known as drug-resistant TB, or DR-TB. When Henry was first diagnosed with TB, rapid molecular tests that can quickly identify drug resistance had not yet been developed, and drug resistance could only be established through the time-consuming and expensive process of culturing bacteria from Henry's sputum and then testing it against each individual drug in the regimen. And so instead, it was (and remains) common to treat all TB cases with these old first-line drugs, and then only test for drug resistance if the first-line treatment fails.

Drug resistance is sometimes present in the strain of *M. tuberculosis* from the beginning; other times it develops after treatment begins, especially if treatment is not adequate. We'll never know how or when Henry's drug resistance developed, but most likely he was responding well to treatment until his father, who'd briefly reentered Henry's life, insisted on taking Henry off the medication regimen. After the first three months of treatment, Henry's father felt certain the drugs had failed. He demanded Henry stop taking the pills and then sought help from a traditional faith healer. "This is not a disease for the doctors," as Henry's father put it. Instead, it was a disease to be treated by God through God's healers.

It is easy to criticize this choice, but it's also worth remembering that in 2006 there was little reason to trust the healthcare system in Sierra Leone. In the wake of the civil war, many systems in the country had collapsed, including healthcare. Most hospitals lacked running water and electricity. Many didn't have paid staff, or functioning X-ray machines. Medications—including medications for TB—frequently went out of stock, so it's possible Henry wouldn't have been able to finish his treatment regardless.

Also, for a while at least, the faith healer's interventions—prayers, special teas—seemed to be working. And unlike the medical system, traditional healers treated Henry and his father like people. Henry was not viewed as an infectious case to be feared, but as a human child to be healed.

We pay a lot of attention to how we treat illness, and much less to the critical question of how we *imagine* illness. In Christian Europe, the disfiguring illness leprosy (which is caused by a bacteria similar to *M. tuberculosis*) was long heavily stigmatized, so much so that lepers were often cast out of society, even as they were also sometimes considered destined for heaven, since Christ healed lepers. In her book *Curing Their Ills,* Megan Vaughan tells us, "In early medieval France a priest performed the ritual of separation in which the leper stood in a grave whilst the priest

threw three spadefuls of earth on her or his head, announcing that they were ‘dead to the world’, but would be ‘reborn in god.’ ”

But this way of imagining leprosy is not inherent to the disease—in fact, Vaughan points out that in precolonial Africa, leprosy was not especially feared or stigmatized, and certainly was not seen as a cause for removal from the social order.

When I was initially diagnosed with an anxiety disorder in the late 1980s, the first SSRI medications were very new. Pathological anxiety was not seen primarily—at least among my peers and caregivers—as a biomedical phenomenon, but instead as an overdeveloped personality trait. It was as likely to be healed by faith as by science. (In fact, I found great relief from my anxiety through religion and ritual.) Today, in my community, anxiety is more likely to be imagined as an illness to be treated through the healthcare system. I would argue this shift happened largely because the healthcare system got better at treating anxiety—not only with pharmaceutical interventions (although those have been very effective for many, including me) but also with stronger evidence-based therapy practices like exposure and response prevention therapy for obsessive-compulsive disorder or cognitive behavioral therapy for general anxiety disorder.

And so we must remember that illness is not only a biomedical phenomenon, but also a constructed one, and how we imagine leprosy or OCD or tuberculosis matters. In a place where the formal healthcare system is not particularly effective at treating an illness, it is easy to imagine how more trusted spaces and people—like churches and faith healers—can be a better bet than doctors and hospitals.

Henry’s improvement didn’t last. Eventually, Isatu was able to get Henry back on the RIPE drugs, but now they seemed less effective. “Then Favor got sick,” she told me once. “Life became so, so difficult. Hopeless. I

worked in the market, but everything I put my hand on, the money whipped away on sickness."

Favor had developed a cyst in her throat that interfered with her vocal cords and made her voice hoarse and whispery. Isatu believed in the medical system even if her husband didn't, and so she took Favor to the hospital, where they told her that Favor would need surgery on her larynx to remove a tumor. The tumor was likely benign in the sense that it wouldn't spread to other parts of the body, but it was potentially fatal because if not removed, it could grow to close off Favor's windpipe.

Now, Isatu struggled even more to try to raise the money for Favor's surgery. Her days were long, and they did not feel like her own—after getting the kids to school, she would try to sell in the market all day until it was time to come home and feed them whatever she could, whatever had not whipped away on sickness. "When they would come home," she remembered, "sometimes there was nothing to eat. I would make them sugar and milk and curry, but with no rice because we didn't have money for any rice, just milk and spices. I would pray for more food. I could see how hungry they were."

In stark contrast to Henry's slow, decade-long decline with TB, Favor got sick quickly. Soon, she was eating little and struggling to speak. But they simply couldn't afford the surgery. Isatu saved every leone she could, and accepted help from friends and family, but just as the family was closing in on the funds necessary to pay for the surgery, Favor died at home. She was seven.

Henry was nine then, and he was utterly devastated. "I really miss her," he later wrote. "We always made jokes together at home. I was not able to eat fast or eat much because of my sickness, and Favor would always try to feed me and get me to eat a lot. She used to tell on me to mom, but I could never get angry with her. We studied together. She was very good at mathematics. I really, really miss her."

A few years after Favor's death, and just over a decade after the civil war ended, Sierra Leone was devastated by an outbreak of the hemorrhagic fever Ebola. The already fragile healthcare system completely collapsed. Because most clinics lacked clean water and protective equipment like gloves and masks, many healthcare workers were infected with Ebola. At least 221 Sierra Leonean healthcare workers died of Ebola between 2014 and 2016, including many of the nation's most experienced physicians, nurses, and community health workers.

Ophelia Dahl, co-founder of Partners In Health, has described such crises as "acute-on-chronic." Chronic impoverishment and failures of resource distribution fuel acute crises like the Ebola epidemic. Ebola is difficult to treat even in rich countries but was almost impossible in Sierra Leone in 2014.[*3]

In response to the crisis, funding flooded in. Nonprofits and disaster response organizations rushed into Sierra Leone (as well as neighboring Liberia and Guinea) to build temporary Ebola Treatment Units and provide trained healthcare staff to support them. I visited one such unit in 2019 in eastern Sierra Leone. A young man who survived Ebola walked me around an abandoned series of concrete slabs with crumbling walls. This place, the survivor explained, had been imagined as a school by a nonprofit that ran out of funding, and so it was never completed. It became a makeshift Ebola treatment facility where the young man shared a cot with his son, sweating in the heat, trying to keep down Pedialyte, watching people (including his wife and mother) die all around him.

And now, as he showed me the place in 2019, it was once again a ruin. The Ebola survivor explained to me this was common in Sierra Leone: People come in with a one-year or three-year grant to do this or that, and then at the end of the grant period, they leave with a half-finished project. In the case of Ebola response, Dahl said that you could "hear the sucking sound" of global money leaving as the crisis started to wind down. But for many Sierra Leoneans, hardly anything had gotten better. Yes, Ebola had been successfully addressed, but the healthcare system was weaker than ever—clinics were no better resourced than they had been before the crisis,

few new healthcare workers had been trained, and much of the existing healthcare workforce had been killed by Ebola.

Amid the Ebola outbreak in 2015 and 2016, Henry became very ill. He sweated through his bedsheets at night, often had fevers, and was vomiting and coughing up blood. But he also desperately wanted a normal life. Most of all, he wanted to be able to sit for his exams to get into secondary school. He knew education was the key to a successful life, a life where he might be able to travel and work, a life where he could be "a person in society," as he once wrote. But his reemergent, rampaging tuberculosis threatened to deny him those opportunities.

Henry and his mother were slow to seek treatment this time, in part because accepting the reality of his illness would be so devastating for his future prospects, but also because even as Ebola began to decline, Henry and Isatu still knew that it spread at healthcare facilities. Many people living with TB were unable to take their medicine as clinics closed or became seen as dangerous. "It was during Ebola that we really saw the explosion in drug-resistant TB," Dr. Bailor Barrie told me once. "Because people could not get their medication, and so the disease had the chance to evolve resistance."

In Freetown, Henry prayed every night for health. He didn't want to leave this world, and he especially didn't want to leave his mother.

By 2016, Henry had lived off and on with active TB for more than half of his life. The illness waxed and waned for most of his childhood, retreating after treatment or simply due to a stronger immune system and less malnutrition. But now the disease roared into undeniability as he prepared for secondary school. He became tired studying for his exams, and it didn't help that he coughed all night, often spitting up blood. He had no appetite and began to lose weight. Despite the family's fears, Isatu brought Henry to

Connaught Hospital in Freetown, one of the best hospitals in the country, where he was again diagnosed with tuberculosis.

By this time, those extremely accurate and quick molecular tests were available in rich countries. Within a couple hours of providing a sputum sample, Henry could have known not only that he had tuberculosis, but which drugs would treat his particular infection. He could have known that his TB was resistant to two of the first-line RIPE drugs. He could have known that his TB was also resistant to a second-line drug. He could have immediately begun taking appropriate, widely available medication that would've cured him within eighteen months. But although these molecular tests had been around for a few years, they were expensive and unavailable to Henry, or indeed to most other patients who desperately needed them. Instead, Henry was diagnosed via chest X-ray, which showed advanced disease but gave no indication as to whether Henry's particular infection was resistant to first-line antibiotics.

And so through Connaught Hospital, Henry again began taking the standard RIPE cocktail for TB, a regimen of between ten and twenty pills per day. Henry faithfully showed up each day and took his pills. He seemed to get better at first, but the drugs were incapable of fully clearing the infection. He grew sicker. In time, he no longer needed to travel to Connaught to take his meds, because he was a full-time patient there. Henry remembered being afraid: "Loneliness and despair cast their long shadows, testing the very core of my spirit," he wrote. His roommate at Connaught died of tuberculosis, and Henry could feel himself getting sicker. He began to notice swellings in his neck and above his collarbone—a sign the TB had invaded his lymphatic system. In time, this scrofula would become so large that it ruptured through the skin, leaving an open wound that seared with pain.

After two years receiving treatment at Connaught Hospital, and after a time-consuming and laborious process of growing bacterial cultures from

Henry's sputum and testing different antibiotics against those cultures, Henry was finally determined to have multidrug-resistant tuberculosis. His doctors told him that he needed to be transferred to Lakka Hospital immediately, as it was the only MDR-TB treatment facility in Sierra Leone. He would need an entirely new medication regimen, including highly toxic injected antibiotics. On his last night at Connaught Hospital, Isatu lay with Henry in his hospital bed, and together they cried through the night. "Everyone knows," one TB patient told me, "what Lakka means. Lakka is the place you go to die. You go in, and you do not come out."

Skip Notes

*1 When Henry first became ill in the early 2000s, TB was still diagnosed in Sierra Leone primarily by microscopy—placing a slide stained with a person's sputum under a microscope and looking for the rod-shaped TB bacteria. Unfortunately, this diagnostic method misses around half of all TB cases—and infections in young children are especially likely to be missed by microscopy. Even more unfortunately, in 2025, microscopy continues to be the leading test for tuberculosis, and continues to be very unreliable.

*2 RIPE is the acronym most commonly used in the U.S. In the rest of the world, this drug combination is generally referred to as HREZ. The global health community loves acronyms so much that they will make up more than one to describe the same thing.

*3 While Ebola is often portrayed as a death sentence, over 75 percent of people who've been treated for Ebola in the U.S. have survived.

CHAPTER 6

TIGER GOT TO HUNT

SOMETHING LIKE 90 PERCENT OF people die of disease, a phenomenon so entrenched in human life that we attribute most such deaths to "natural causes." Many of us feel a certain relief when we learn that someone has died "naturally," especially when the death occurs at what we think of as an appropriate age.

When I was twenty-one, I worked for several months as a student chaplain at a children's hospital. My supervisor was a Presbyterian pastor with the soulful eyes and calm voice her job demanded. On my first day of training, she said to me, "Death is natural. Children dying is natural. None of us actually wants to live in a natural world."

Treating disease—whether through herbs or magic or drugs—is unnatural. No other animals do it, at least not with anything approaching our sophistication. Hospitals are unnatural, as are novels and saxophones. None of us actually wants to live in a natural world. And yet we tell ourselves that some—and only some—lives end naturally (which really means "acceptably" or "well"). We construct ideas about what constitutes a good time and manner of death. I recently asked my ten-year-old daughter what constituted a natural death. "Well, you have to be old," she said. "At least seventy-five. And you should probably be asleep."

But that's not the only definition of a good or natural death. In Elizabethan England, any death that occurred immediately after a confessional prayer was good, because it sped the path to heaven. (You'll recall, for instance, Hamlet's hesitation to kill his praying uncle, lest old

Claudius receive a free ticket to immortal glory.) To the Romans, a death in battle was a good death, at least for men. As Horace put it, “Dulce et decorum est pro patria mori.” (“It is sweet and noble to die for one’s homeland.”) By World War I, the poet Wilfred Owen turned that idea on its head, arguing that deaths from war were “obscene as cancer” and calling Horace’s line “that old lie.”

Owen’s idea that cancer is obscene is also a construction, as is our current idea that for a life to be truly good, it must end in old age, and probably asleep. Our understandings of good illnesses, and good deaths, are perpetually shifting.

Before vaccination, C-sections, infection control, and antibiotics, the death of children was routine. About half of all humans ever born died before the age of five. Child death was so common that it had to be acknowledged as natural. And so the acceptable times to die in much of the premodern world were 1. Early in childhood, or 2. Late in adulthood.

But tuberculosis has long been known for sickening and killing those between twenty and forty-five, during the one period of life when you were supposed to be relatively insulated from illness and death. While TB is also common among children and elderly people, consumption was known as “the robber of youth” in the eighteenth and nineteenth centuries because of its ubiquity among young adults. How could we explain this disease that should not be, that was at once omnipresent and terrifyingly unnatural?

I want to pause here to note a defining feature of humans, which is that we like to know why things happen, especially why really bad things happen. And if a reason is not immediately apparent, we will find one. I am reminded of a poem in Kurt Vonnegut’s novel *Cat’s Cradle*:

Tiger got to hunt,
Bird got to fly;
Man got to sit and wonder, “Why, why, why?”

Tiger got to sleep,
Bird got to land;
Man got to tell himself he understand.

Vonnegut reminds us that we are both inclined toward curiosity *and* inclined toward arriving at some kind of comprehensible conclusion. “Nothing is more punitive than to give a disease a meaning,” Susan Sontag famously wrote, and yet we go on giving disease meanings anyway. These illness narratives are often not just a strategy for conceptualizing the pain of others, but also a way of reassuring ourselves that *we’ll* never feel that pain.

The way we symbolize disease ends up shaping the way we experience and respond to disease. As we’ve seen, in various places and times TB has been seen as an illness of the living corpse, or of demon possession. It has also been stigmatized as a drunkard’s illness, or an illness caused by sexual immorality, or poor hygiene. One eighteenth-century consumptive, a Mr. Rookes, died—according to his doctor—due to engaging in “six unnatural things.” These were overeating, drinking alcohol, constipation, overexercise, staying awake late, and perturbation of the mind.

But the illness could never be entirely dismissed by stigmatizing gluttony and exercise, because it was not only the constipated alcoholics who suffered from consumption. The disease was simply too common to be an illness of moral failing. Also, it affected not only the poor and marginalized, but also the rich and powerful.

Toward the end of the eighteenth century, Europe (and especially Britain) saw an unprecedented rise in consumption cases, a phenomenon the physician Maurice Fishberg called “the frightful tuberculization of humanity.” The disease became not just the leading cause of human death, but *overwhelmingly* the leading cause of human death. One analysis quoted in Vidya Krishnan’s *Phantom Plague* found that “roughly 15% of all deaths in London before 1730 were due to the disease, a percentage that nearly doubled [by] the early 1800s.” When almost a third of all people shared the same fate, it became impossible to construct consumption as merely a

disease of the drunk or demon-possessed. There were simply too many cases for consumption to be understood as a disease caused by immorality or weakness. Something had to be done—if not about the disease, then at least about our imagining of it.

And so, in the eighteenth and nineteenth centuries, Europeans came to romanticize consumption, to see the illness as beautiful and ennobling. It's tempting to imagine this romanticization as the opposite of stigmatization—rather than discounting people as stigma does, romanticization lifts them up as paragons of beauty or intellect or some other virtue. But really, I see these as complementary strategies used to make the sick into an "other," a group of people fundamentally distant and different from the rest of the social order. Mental illness, for example, is often romanticized as bringing on creative genius or other superpowers. Brilliant detectives like Sherlock Holmes and Adrian Monk are portrayed as having their powers of detection enhanced by their obsessive thought spirals even as they are also stigmatized for their eccentric and off-putting behavior. But as someone with OCD, I can report with some authority that both the stigma and the romance are unfair. People living with OCD are not generally good detectives—in my case at least, OCD does not make me good at noticing anything other than my own obsessive fears. But I find it similarly problematic to stigmatize my compulsive behaviors as revolting or odd.

Imagining someone as more than human does much the same work as imagining them as less than human: Either way, the ill are treated as fundamentally other because the social order is frightened by what their frailty reveals about everyone else's.

I would argue that the proper way to understand the utterly surreal romanticization of TB is that as the disease exploded in cities, stigma alone simply could not answer the "why, why, why" of consumption. Instead, people began to conclude that consumption was caused by a personality especially attuned to the fragile and fleeting loveliness of life. This made a

kind of sense—in northern Europe, consumption was widely understood to be an inherited disease, passed on, like personality traits, from parents to children. And so it reasonably followed that consumption might be accompanied by other traits, like beauty and brilliance and sensitivity.

This romanticization continued for a very long time: In the 1909 book *Tuberculosis and the Creative Mind*, Dr. Arthur Jacobson maintained that TB offered a "divine compensation" in exchange for illness: TB patients' lives "are shortened, physically, but quickened psychically in a ratio inversely as the shortening." Maybe the nineteenth-century Romantics would die early, but oh, the poems they would write.

In his classic book *Epidemics and Society*, Frank M. Snowden notes, "Lungs are more ethereal than bowels." Although consumption in its late stages can involve large volumes of diarrhea and vomiting, phthisis was widely viewed as a disease of the air. It was an illness of the breath, of the place where the body interacts with the atmosphere, a process so sacred that the Hebrew word *ruach*, the Chinese word *chi*, the English word *spirit*, and the Inuit word *sila* all derive from words meaning *breath* or *breathing*. Breath *is* life—respiration is the most visible and irrefutable sign that we are still here. To inspire is to breathe in; to expire is to breathe all the way out.

And so it was easy to make this disease of breath a disease of the spirit, the chi, the sila, and the ruach. Consumption was believed to bring the creative powers to new levels, helping artists get in deeper touch with the spirit as their worldly bodies literally shrank away.

As evidence for consumptive brilliance, scholars pointed to the fact that everyone from Stephen Crane to Frédéric Chopin died of consumption. (Less attention was paid to the fact that, in a world where over a quarter of all people were dying of the disease, it should not be particularly surprising that many artists and writers were dying of it.) One London magazine in 1825 wrote, "It is a striking fact that genius is often attended by quick decay

and premature death." Another linked phthisis to authors in particular, writing that an author's "waywardness, peevishness, irascibility, misanthropy, [and] murky passions…are referable to their constitutional peculiarities and condition: In simple words, their mental eccentricities result from the derangement of bodily health." This is precisely what I mean when I say that romanticization is not a kind or generous way of treating the ill. I am an author, and I for one am deeply offended by the notion that my waywardness, peevishness, irascibility, misanthropy, and murky passions are caused by a derangement of bodily health, even as I am impressed by a nineteenth-century magazine's ability to absolutely nail my personality.

Inheriting TB also meant you had inherited other traits—the "tuberculous personality" involved a melancholic outlook on the world, and a deep understanding of the beauty and frailty of life. Female artists were occasionally mentioned in analyses of how the disease quickened genius—the Brontë sisters, after all, died of TB, and Elizabeth Barrett Browning likely did as well—but the focus was on men and how TB enhanced their talents. It is said that Victor Hugo's friends joked with him that he could've been a truly great novelist…if only he'd contracted consumption. Lord Byron wrote, "I should like, I think, to die of consumption…because then the women would all say, 'See that poor Byron—how interesting he looks in dying!' "

It's hard to overstate how profound the link between consumption and creative genius was in eighteenth- and nineteenth-century Europe and the U.S.[*] When TB rates declined in the U.S. toward the end of the nineteenth century, some physicians worried it would harm the quality of American literature, with one writing, "By way of compensation for good health we may lack certain cultural joys."

Even in twentieth century, Maurice Fishberg wrote in his book *Pulmonary Tuberculosis* that young TB patients "display enormous

intellectual capacity of the creative kind. Especially is this to be noted in those who are of the artistic temperament…. They are in a constant state of nervous irritability, but despite the fact that it hurts their physical condition, they keep on working and produce their best works." This idea was known as *spes phthisica*, or consumptive spirit, which Dr. David Morens defines as "a condition believed peculiar to consumptives in which physical wasting led to euphoric flowering of the passionate and creative aspects of the soul."

Even artists themselves believed in spes phthisica. The consumptive American poet Sidney Lanier wrote to his wife, "I would think that I am shortly to die, and that my spirit hath been singing its swan-song before dissolution. All day my soul hath been cutting swiftly into the great space of the subtle, unspeakable deep, driven by wind after wind of heavenly melody."

We find this romanticization in fictional accounts of consumption as well. "There is a dread disease," Charles Dickens wrote in *Nicholas Nickleby*, "which so prepares its victim, as it were, for death; which so refines it of its grosser aspect…in which the struggle between soul and body is so gradual, quiet, and solemn, and the result so sure, that day by day, and grain by grain, the mortal part wastes and withers away, so that the spirit grows light and sanguine with its lightening load." Dickens did not name the disease—nor did his readers need him to. This concept of the body being refined of its grosser aspect, of the mind flowering as the body wilts, continues to reverberate, of course—both because the sick are still sometimes viewed as unusually proximal to the divine and sacred, and because those with smaller bodies are often treated as more valuable or beautiful by the social order.

Consider the way Harriet Beecher Stowe describes the death of a child from consumption in *Uncle Tom's Cabin*: "The child felt no pain…and she was so beautiful, so loving, so trustful, so happy, that one could not resist the soothing influence of that air of innocence and peace which seemed to

breathe around her…. It was like that hush of spirit which we feel amid bright, mild woods of autumn."

Notice the adjectives here—loving, trustful, happy, soothing, bright, mild. Who among us would not want to live such a life, and die such a death? How natural! How lovely! And there is something to it—people usually die of tuberculosis slowly, over months or years, as opposed to a terrifying epidemic like cholera or typhoid sweeping through a city. People dying of consumption often, although not always, appear to die peacefully, or at least quietly. But mostly, this was a lie a society told itself to fathom losing so much of itself to one disease.

There were, however, some who saw the absurdity of this lie. Alexandre Dumas, for instance, satirized the romanticization of the moment. He deadpanned that in 1823 and 1824, "it was the fashion to suffer from the lungs; everybody was consumptive, poets especially; it was good form to spit blood after any emotion that was at all sensational, and to die before reaching the age of thirty." But Dumas himself also embraced a romantic ideal of consumption; as David Barnes points out in *The Making of a Social Disease*, "the disease's wasting effect on the body is portrayed as enhancing feminine beauty" in Dumas's novel *The Lady of the Camellias*.

Perhaps the most famous nineteenth-century victim of tuberculosis was the English poet John Keats, who, like the Brontës, seemed to have a familial predisposition toward the disease. (In fact, Keats probably developed TB after caring for his consumptive brother.)

As a former medical student, Keats knew what it meant when he saw a spot of blood in his sputum, telling a friend, "It's arterial blood. That blood is my death warrant. I must die." Keats's friends and colleagues clearly associated his genius with his illness. Percy Shelley (who also lived with phthisis) wrote to him, "This consumption is a disease particularly fond of people who write such good verses as you have done."

As he grew more gravely ill, Keats wrote of the pain and inability to get adequate oxygen to his body: “We cannot be created for this sort of suffering.” He would wish for death, but then wish for life, because “death would destroy even those pains which are better than nothing.” He was twenty-four when he wrote those words, and had only five months to live.

Keats’s friend and caregiver Joseph Severn reported that the poet would sometimes awake in tears, devastated to still be alive and in such pain. A few months before his death, Keats wrote what amounted to a will. He had no money. “My estate real and personal consists in the hopes of the sale of books publish’d or unpublish’d,” he wrote, and then atop the note, he scrawled and underlined a single line of iambic pentameter: “My chest of books divide among my friends.”

In *Ode to a Nightingale*, Keats wrote, “Youth grows pale, and spectre-thin, and dies.” It proved a prophecy. When Keats’s body was autopsied a couple of days after his death, the doctor wrote, “The lungs were completely gone.” This was—and is—the truth of death by tuberculosis. The afflicted often drown as blood and pus fills their lungs. They die starved of oxygen, desperate for air. One physician and anthropologist described a typical tuberculosis death: “He is seized with a sharp agonizing pain in the chest…. The facial expression is that of profound agony, the eyes prominent, the lips livid, and the forehead clammy.”

Of course, romanticizing the troubled young artist isn’t unique to tuberculosis—I think of the way people talked about Billie Holiday’s substance abuse disorder, or Vincent van Gogh’s mental illness. There’s something about the candle snuffed out prematurely that captures our imagination—it is the thought, perhaps, of the books and paintings and songs that might’ve been, or the idea that artists simply burn too bright for this world.

Consumption, as the robber of youth, played a special role in this relationship between youth, artistry, and health—and not only in Europe.

The Indian poet Sukanta Bhattacharya died at just twenty of tuberculosis in 1947. Bhattacharya wrote gut-wrenchingly of the Bengal famine, which was orchestrated by British colonial authorities and resulted in the deaths of perhaps three million people. Bhattacharya's death was probably hastened by his own experiences during the famine—as we've seen, malnutrition can trigger a tuberculosis infection into active disease. "Our history will be shaped by / Hungry stomachs," he wrote. An Indian newspaper referred to him as "the John Keats of Bengal," and like Keats he was credited with a wisdom beyond his years tightly linked to his suffering. But unlike Keats, he was an unabashedly political poet, and I am especially haunted by one of his lines, written when he had very little time left to live: "Your pleasure signals our death."

But I find no writing about TB so moving as that of Masaoka Shiki, the Japanese poet often credited with revitalizing the haiku form in the late nineteenth century. Shiki suffered from spinal and pulmonary tuberculosis and spent the last five years of his life in bed. His pain was agonizing and constant. (He adopted the pen name Shiki after a Japanese cuckoo bird that was believed to sing until it spat up blood.) Shiki was far less romantic about suffering than European contemporaries, but like Lanier he felt the creative urge right up until his death. In fact, he wrote a few haiku just hours before he passed away in 1902, beginning with one that translates to:

the gourd flowers bloom,
but look—here lies
a phlegm-stuffed Buddha!

This phlegm-stuffed Buddha wrote extensively about the experience of spending one's life in bed, slowly drowning.

Pain from coughing
the long night's lamp flame
small as a pea

Across many of his eighteen thousand poems, Shiki elliptically and brilliantly captures the pain and the isolation of illness. One series of haiku begins:

It is snowing!
I see it through a hole
In the paper door

The poet can celebrate snow, even if he can only see it through a hole in the door. But he feels himself more observer than participant:

All I can think of
Is that I am lying
In a house in the snow.

The consumptive poet cannot be in the snow, only lying in a house in the snow. For me, anyway, this way of understanding chronic illness—as being of the world but also not permitted by circumstances or the social order to be entirely *with* the world—is a sentiment applied from within rather than from without, a way of thinking about the limits and opportunities of disability that acknowledges difference and loss without othering or romanticizing. It's not trustful or loving or soothing or mild. It's true.

SKIP NOTES

* As we will see, the romanticization of the phthisis patient wasn't limited to the West, either.

CHAPTER 7

THE FLATTERING MALADY

YOU WERE EITHER BORN WITH spes phthisica or without it. We've seen how this belief in a genetic component to consumption led to the belief that consumptives were predisposed to sensitivity and artistic genius. Women were also believed to be elevated by their illness, sometimes in their artistic gifts, but most obviously in their physical beauty.

Women with consumption were believed to become more beautiful, ethereal, and wondrously pure. As Charlotte Brontë put it in a letter she wrote as her sister was dying of the disease, "Consumption, I am aware, is a flattering malady."

Patients with active tuberculosis typically become pale and thin with rosy cheeks and wide sunken eyes due to the low blood oxygenation and fevers that often accompany the disease, and these all became signals of beauty and value in Europe and the United States. Henry David Thoreau wrote in his journal, "Disease and decay are often beautiful—like the pearly tear of the shellfish or the hectic glow of consumption."

Phthisis was deeply associated with feminine beauty in Northern Europe. Small, waifish bodies can now seem so associated with beauty (and health!) that it can feel innate or instinctual to find smaller bodies more attractive than larger ones. But that's not inherent to humanity (and indeed was not a significant bias of humanity until relatively recently). That said, it's important to note that the idealization of the small body did not mean the end of consumptive stigmatization. Once again, we see the commingling of romance and stigma in the way women's bodies are

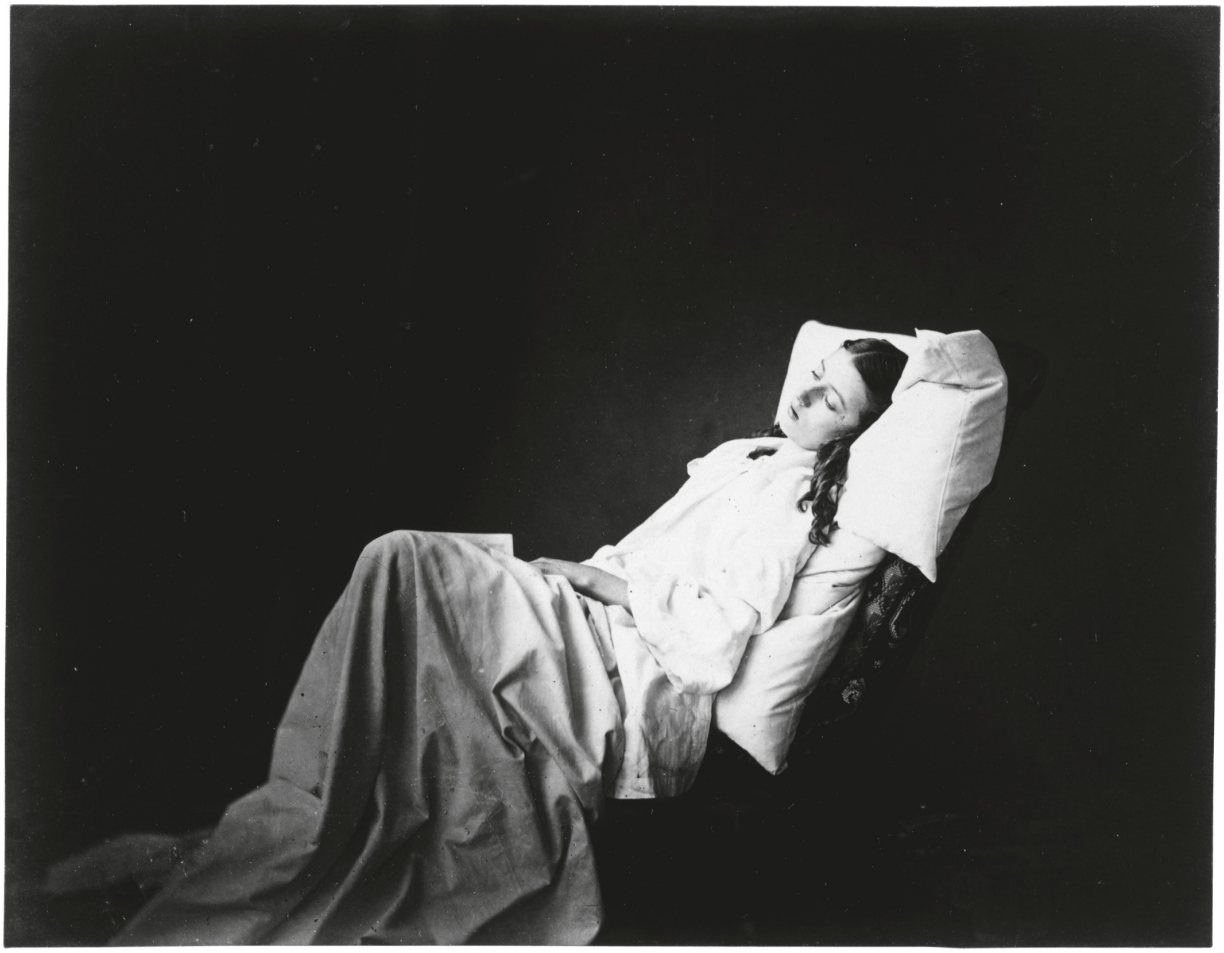
imagined, sometimes within a single sentence, as when one eighteenth-century magazine extolled the virtues of a consumptive body type: “The beauty of women is greatly owing to their delicacy, or weakness.” One romantic word to describe the beauty standard—delicacy—followed by a stigmatizing one—weakness.

The English actor Eliza Poe, whose beauty was widely admired, looked stereotypically tubercular—her rosy cheeks, alabaster skin, wide eyes, and tiny body were all the result of consumption, which killed her in 1811, when she was in her early twenties and her son, Edgar, was two. Edgar Allan Poe would go on to describe many of the women in his stories and poems as similarly wispy, pale, and large-eyed before he himself possibly died of tuberculous meningitis.

By the time of Eliza’s death, “consumptive chic,” as Carolyn Day termed it, had taken over European beauty standards. Women were discouraged from physical activity and too much time in the sun. “Languid and listless ladies sporting pale complexions were all the rage,” Day writes. The French novelist Henri Murger told of a young consumptive’s corpse that the glow of her face made it look like “she had died of beauty.” Even Henry Gilbert’s 1842 medical treatise *Pulmonary Consumption* contained an ode to the beauty of female consumptives—and I don’t mean that figuratively. The poem goes:

With step as noiseless as the summer air,
Who comes in beautiful decay? Her eyes
Dissolving with a feverish glow of light;
And on
Her cheeks, a rosy tint, as if the tip
Of beauty’s finger faintly press’d it there:
Alas! Consumption is her name.

We can see these shifting beauty standards in European art of the time. Consider Henry Peach Robinson's *Fading Away* (1858), in which Robinson combined five different photographic negatives to create a single combined portrait. In an early study for *Fading Away*, a young model—Sarah Cundall, the daughter of a grocer—is portrayed as pale, waiflike, and weak from consumption. And yet she is also clearly meant to be beautiful, complete with the sheet covering her lower body mimicking the folds of a dress in a Renaissance sculpture.



She Never Told Her Love, an albumen silver print from glass negative by Henry Peach Robinson, 1857. Early study for *Fading Away*.

In the finished *Fading Away,* an image of Cundall has been combined with other negatives to create an image of a “good death.” Victorian English viewers were scandalized by Robinson’s photograph because it depicted such a private human moment so realistically that many thought the young waif really was fading away into death. But others were charmed by the artwork—in fact, Prince Albert bought a print of the picture. In these photographs we see the fully romanticized image of the alluringly pale young woman not dying amid violence or disfigurement, but fading away peacefully (and passively) from consumption.

Fading Away, a combination print by Henry Peach Robinson, 1858.

Around the time of this photograph, some women applied belladonna to their eyelids, albeit in minimally toxic amounts, to dilate their pupils so they’d have that wide-eyed consumptive look.[*1] Magazines also offered instructions for how to apply red paint to the lips and cheeks to capture the hectic glow of consumptive fevers. I probably do not need to point out that

these standards of beauty are still informing what is considered to be feminine beauty in much of the world.

The romanticization of TB also intersected with fashion for both men and women, although the interaction was complicated, and we must be careful not to conflate correlation with causation. It's often been said, for example, that corsets sought to emulate the experience of consumption by being restrictive enough to limit women's breathing and physical activities, but more recently, historians have found that most corsets were not particularly restrictive and these connections have been overblown. In fact, men (including physicians) at the time argued that corsetry might restrict blood flow, and that corsetry was a villainous force that *caused* consumption by forcing blood to stagnate in the lungs. Other articles of clothing (including menswear that tightened at the waist) were also seen as fueling phthisis. One article claimed that wearing thin shoes was "an equivalent for the hacking cough and hectic flush." So while male historians may have connected corsets to consumption, the contemporary fashion historians I've spoken to do not believe women's dress at the time sought to mimic or encourage the symptoms of consumption. Instead, fashionable dressing was viewed as a risk factor for consumption in Europe precisely because the patriarchal social order disapproved of it.

I came across a comment on a video about tuberculosis recently in which a woman named Jil wrote, "As a fat person, I used to wish for a wasting disease like tuberculosis. It's…it's messed up." Dozens of people replied to that comment with their own experiences of being complimented for weight loss associated with life-threatening illness, or their fantasies of tapeworms and other illnesses that would shrink their bodies. The idea of becoming sick in order to look healthy or beautiful speaks to how profoundly consumptive beauty ideals still shape the world we share.

But as pervasive as these beauty standards are, we must remember that they aren't universal. In Sierra Leone, being small and thin like Henry

brought to mind not beauty but stunting and unwellness.[*2] In fact, Henry fit all the ideals of the consumptive—the wide eyes, the visible cheekbones, and the creative temperament. He wrote beautiful poems, and his interest in writing blossomed during his illness. He was preternaturally brilliant and deeply sensitive, expressing deep emotions of yearning and love in his memoir and poetry.

But of course he did not live in the nineteenth century, and he also wasn't white.

If the pale, thin, wide-eyed, rosy-cheeked beauty standard has proven astonishingly durable, the conflation of whiteness with consumption would prove even more devastating to human health and equity. One 1807 essay, "On the Beauty of Skin," wrote: "A white skin, slightly tinged with carnation, soft and smooth to the touch, is what we commonly call a fine skin." Fair skin with rosy cheeks and prominent, round eyes all call to mind the final days before death from consumption. The cheeks are rosy from fever; the skin is white from deoxygenation; the cheeks and eyes are prominent because the body is, as one recent TB survivor put it, "becoming a skeleton." But to read these descriptions of beauty or refinement is to be overwhelmed by the word "whiteness." *The Ladies' Toilette* tells us, "Whiteness is one of the qualities which it is requisite for the skin to possess before it can be called beautiful." The 1837 book *Female Beauty* lays it out plainly: "Whiteness is the most essential quality of the skin."

Those esteemed for their beauty in Europe and the U.S. at the time were described as having "alabaster" skin, or "marble," or "translucent." In fact, in the most famous portrait of Eliza Poe, it is difficult to tell where her consumptive skin ends and her white dress begins.

As Frank M. Snowden observes in *Epidemics and Society*, white physicians in Europe and the U.S. generally agreed that consumption was, as some

eighteenth-century observers put it, a disease of civilization. Everyone knew that rural communities were less vulnerable to consumption. “Fond as I am of London,” one mother wrote after both she and her daughter became ill, “there seems a fatality against my living in it.” But in a highly racialized social order, conceiving of phthisis as a “civilized” disease also meant that it could not be a disease of uncivilized people, which furthered the racialization of consumption.

In Europe and the U.S., most white doctors believed that phthisis—as it was inherited by those with great sensitivity and intelligence—could *only* affect white people, and it was sometimes known as “The White Man’s Plague.” One American doctor, for instance, called it, “a disease of the master race not of the slave race.”

As Snowden writes, “In the United States, the prevailing wisdom was that African Americans contracted a different disease. The disinclination even to give it a name speaks volumes with regard to the prevailing racial hierarchy and the lack of access to medical care by people of color.” This phenomenon extended to all colonial empires. Many European colonialists believed that TB did not exist in South Asia or Africa, even though physicians working in colonized communities knew otherwise. As one wrote in 1829, “It is a generally received error that pulmonary disease in India is rare and readily cured.”

What we see here is yet another example of how our understandings of tuberculosis are shaped by social forces—which in turn shape how and where tuberculosis is able to thrive. In India, where TB was supposedly “rare and readily cured” in 1829, it was in fact neither. There was always extensive illness and death from consumption in colonial India; it just went largely undetected and uncounted by colonial authorities. After all, the entire premise of colonialism relied on white supremacy, and the entire premise of spes phthisica maintained that only superior and civilized (read: white) people could become consumptive. Acknowledging that consumption was common among enslaved, colonized, and marginalized people would have undermined not just a theory of disease, but also the project of colonialism itself.

Skip Notes

*1 Belladonna also had other medicinal uses—it was long used by optometrists to dilate pupils for eye exams.

*2 Once, on a visit to Sierra Leone, I met up with an old acquaintance I hadn't seen in five years. "Look how fat you've gotten!" he told me, approvingly. "A little, I guess," I answered, feeling self-conscious. "No, no," he said, patting my stomach. "You've gotten very fat!"

CHAPTER 8

THE BACILLUS

OUR HISTORICAL OVERVIEW HAS FOCUSED on northern Europe and the U.S., where consumption was considered inherited for most of the nineteenth century, but that certainly wasn't the case everywhere. Rates of phthisis appear to have been lower, for example, in China, where Daoist physicians argued the disease was infectious beginning in the twelfth century CE. Consumption was rarer in southern Europe as well, where the illness was also understood to be infectious. As the writer George Sand tried to find a place for consumptive Frédéric Chopin to stay in Spain, Sand wrote a friend, "Phthisis is scarce in these climates and is regarded as contagious." But of course phthisis was scarce in those climates precisely *because* it was regarded as contagious. "We went to take residence in the disaffected monastery of Valdemosa," Sand goes on, "…but could not secure any servants, as no one wants to work for a phthisie…. We begged of our acquaintances that they give us some help…a carriage to take us to Palma from where we wanted to take a ship back home. But even this was refused us, although our friends all had carriages and wealth."

Toward the end of the nineteenth century, consumption began to decline in northern Europe and the U.S. as well. In the process, romanticization of the disease was abandoned. The decline happened in part because, as quality of life rose for the wealthy and the emerging middle class, they were less likely to live or work in crowded spaces where consumption can flourish.

Increasingly, it was the poor who seemed to get sick, and so people began to turn their eyes away from “the languorous, fainting young women and their romantic lovers,” wrote René and Jean Dubos. “They noticed instead the miserable humanity living in the dreary tenements born of the Industrial Revolution. In the ‘tentacular cities’ they saw hosts of men, women and children, pale too, often cold and starving, working long hours in dark and crowded shops, breathing smoke and coal dust. Tuberculosis was there, breeding suffering and misery without romance.”

Indeed, this is how we now understand the surge of consumption in the eighteenth and nineteenth centuries: It arose not from civilization or white skin or a sensitive personality, but from industrialization. The rise of cities and sweatshops meant crowded markets, factories, and streets, which proved an ideal breeding ground for TB. And so just as Britain was ground zero for the Industrial Revolution, it was ground zero for the explosion of tuberculosis. Similar outbreaks have occurred in the twentieth century in India and Nigeria as they industrialized. “TB’s parallel journey with capital,” as the investigative journalist Vidya Krishnan put it, appears in outbreak after outbreak.

And so TB revealed itself to be not a disease of civilization, but a disease of *industrialization*; of crowding and intermingling in huge cities with packed tenements and factories where coughed-up particles could linger in the stale air. As the century progressed, more folks began to argue —with increasingly strong evidence—that consumption seemed to be an infectious disease. But the inheritance camp still held much sway in the debate. In 1881, a major medical textbook identified the causes of consumption as “hereditary disposition, unfavorable climate…deficiency of light, and depressing emotions.”

But the very next year, all these causes would be challenged as it became clear that consumption was caused by the spread of “a vexatious little organism” called *Mycobacterium tuberculosis*. First identified by the German doctor Robert Koch, the discovery of *M. tuberculosis* would radically reshape our understanding of the disease as well as our strategies for containing it—both medically and psychosocially.

In his paper announcing the discovery of the bacteria that caused TB, Koch seemed to recognize the romanticization of the illness. He seemed to feel a bit defensive in trying to make the case that the world's leading cause of death was, in fact, a big deal. "If the importance of a disease for mankind is measured from the number of fatalities which are due to it, then tuberculosis must be considered much more important than those most feared infectious diseases—plague, cholera, and the like."

History is often imagined as a series of events, unfolding one after the other like a sequence of falling dominoes. But most human experiences are processes, not events. Divorce may be an event, but it almost always results from a lengthy process—and the same could be said for birth, or battle, or infection. Similarly, much of what some imagine as dichotomous turns out to be spectral, from neurodivergence to sexuality, and much of what appears to be the work of individuals turns out to be the work of broad collaborations. We love a narrative of the great individual whose life is shot through with major events and who turns out to be either a villain or a hero, but the world is inherently more complex than the narratives we impose upon it, just as the reality of experience is inherently more complex than the language we use to describe that reality. I think that's all worth remembering when it comes to Robert Koch. Koch made important discoveries, but they happened amid a host of other insights related to microorganisms, because breakthroughs in understanding were being made by a wide variety of people who could share findings efficiently through medical journals. (In fact, fifteen years before Koch's discovery, Edwin Klebs proved a TB chain of transmission, but his research was minimized because it wasn't shared widely enough and because he wasn't able to isolate and identify the infectious agent itself.)

And just as Koch's discovery didn't erupt out of nowhere, neither did his catastrophic downfall.

In 1869, Koch was a twenty-six-year-old small-town doctor in what is now western Poland. Young doctors were common at the time—the years of postgraduate education and residency we now associate with physicians did not yet exist, not least because there wasn't *that* much to learn. Koch was struggling to establish his practice until a local baron accidentally shot himself. Using the new sanitation techniques beginning to take hold in Europe, Koch was able to prevent infection and save the baron, which brought him a small measure of local acclaim.

Koch was fascinated by wound infections and the invisible creatures that some believed might cause them. His wife, Emma, bought him a microscope as a present so he could see these organisms himself.[*]

Koch tried again and again to enter the world of proper scientists—he wanted an appointment at a university, and for his papers to be published in the finest journals—but despite the meticulous research that would become his trademark, success eluded him until he made a major discovery in 1876, elegantly proving that anthrax was caused by bacteria. Koch looked at a tissue sample from an animal known to have recently died from anthrax. After staining the sample, he noticed rod-shaped creatures still squirming under the light of the microscope. He then injected tissue from the diseased animal into a healthy rabbit whose tissue samples showed no signs of these squirming microorganisms.

Soon, the previously healthy rabbit was dead, and a post-mortem tissue analysis revealed a "moderate number" of those rod-shaped organisms we now know as the bacterium *Bacillus anthracis*.

But this still didn't prove causation. Perhaps some other aspect of the dead rabbit's tissue had killed the healthy rabbit, and the presence of bacteria was a coincidence. So Koch took a sample from the dead rabbit, allowed the bacteria to grow in a solution made from chicken egg, and then took a sample from the egg solution and injected it into another rabbit, which also soon died.

Koch had proven the bacterium caused anthrax by isolating the bacteria and establishing a chain of transmission, which still remains an important tool in bacteriology and virology. His resulting paper was a sensation, at least as medical journal articles go, and Koch became a star, invited to visit the great institutes of the time and lecture on the emerging field of infectious disease.

Koch soon made his best-known discovery when he showed that bacteria he'd isolated from a tubercle—those sphere-shaped clumps of white blood cells that contain tuberculosis bacteria within them—could lead to a chain of transmission similar to the one seen with anthrax.

He grew a culture of the rod-shaped organisms found within a tubercle and then injected the bacteria into a guinea pig, which became very sick indeed. Koch had proven that tuberculosis (at least in guinea pigs) began not with a genetic predisposition, but with an infestation of these tiny, squiggling bacteria.

When Koch presented his paper proving that TB was caused by what he called *Mycobacterium tuberculosis*, "there was no applause, no murmuring, no debate," Thomas Goetz writes in his brilliant book *The Remedy*. "The crowd was simply, utterly, absolutely speechless." The scientist Paul Ehrlich would later write, "I hold that evening to be the most important experience of my scientific life."

By the end of the nineteenth century, the replication and acceptance of Robert Koch's research meant that the era of consumption, an inherited condition that grew the soul by shrinking the body, ceased to exist. The era of tuberculosis, an infectious disease of the poor and marginalized, had commenced. In fact, the way we understood "consumption"—that bright, mild, kind disease that Harriet Beecher Stowe described—was so different from the way we understood "tuberculosis" that even though they are the same disease, one could be forgiven for thinking they were entirely different. Consumption, after all, was a flattering malady, a genetic disorder

enriching the soul even as it slowly destroyed the body. Tuberculosis was a horror, an invisible contamination proliferating within you and then spreading to anyone near you.

Skip Notes

* Robert would go on to repay Emma's generosity by having a long-term affair with a teenager named Hedwig; Koch eventually left Emma for Hedwig in a move that was largely in keeping with his relentless ambition and self-absorption. But that was in the 1890s—after Koch had become famous, and rich, and powerful, and disgraced.

CHAPTER 9

NOT A PERSON

WE SEE THE PROFOUND SHIFT from an inherited disease of intellect to a contracted disease of filth in the racialization of tuberculosis. As late as 1880, white American physicians still argued that consumption did not occur among Black Americans, who, it was claimed, lacked the intellectual superiority and calm temperament to be affected by the White Plague. But after Koch identified *Mycobacterium tuberculosis* in 1882, all that changed.

Racialized medicine no longer maintained that high rates of consumption among white people was a sign of white superiority; instead, racialized medicine maintained that high rates of consumption among Black people was a sign of white superiority. One white doctor's 1896 treatise asserted that African Americans were disproportionately dying of tuberculosis due to their smaller chest capacity and increased rate of respiration, for example.

None of this was true, of course. Black people were not more susceptible to TB because of factors inherent to race; they were more susceptible to tuberculosis *because of racism*. Because of racism, Black Americans were more likely to live in crowded housing, an important risk factor for TB. Because of racism, Black Americans were more likely to be malnourished, another risk factor. Because of racism, Black Americans were more likely to experience intense stress, and they were less likely to be able to access healthcare. To cite one story among thousands, Thomas Albert White, a Black veteran of World War I, saw his TB infection turn

into active disease after a chemical warfare attack. He returned to the U.S. and was sent by the federal government to a series of TB hospitals throughout the country, all of which denied him entry despite government orders to admit him. White eventually died of his illness without access to care.

Similarly, in the early twentieth century, Irish and Chinese immigrants to the U.S. were widely viewed as having a racial susceptibility to TB. But then as now, tuberculosis does not travel primarily through paths forged by race, except insofar as human power structures force it to.

This racialized medicine *was* challenged—it was obvious hogwash from the beginning, and there was pushback against it, especially from Black healthcare workers. In 1909, a Dr. Stile in Tennessee claimed "the negro is to blame for his own susceptibility to tuberculosis." In response, an anonymous Black physician wrote to a medical journal that this sort of racialized medicine "smacked more of the cheap politician seeking notoriety and office by playing to passion and prejudice than a doctor discussing, philosophically, a scientific subject for the diffusion of knowledge." So there was resistance; it just too often went unheard.

This bias against marginalized people and the healthcare workers directly serving them has proven to be one of the great facilitators of tuberculosis over the last century. How might the contemporary story of tuberculosis be different had we listened to African American physicians like Dr. A. Wilberforce Williams, who noted over a century ago that the real cause of TB was not race but "poverty, bad housing, bad sanitation, bad working conditions, long hours, high rent, [and] poor food"?

The mostly white medical establishment in the U.S., meanwhile, tended to focus on so-called "race susceptibility," the idea that something inherent in the genetics of Black people caused tuberculosis—a kind of spes phthisica for the infectious age. Some white doctors even argued that the "susceptibility" was caused by the end of slavery in the U.S. In his famous 1896 essay "The Effects of Emancipation upon the Mental and Physical Health of the Negro of the South," Dr. J. F. Miller argued (falsely) that

tuberculosis was a “rare” disease “among the negroes of the South prior to emancipation.”

In truth, the disease was “rare” because enslaved people had no access to diagnosis and lived in a world where white physicians presumed that consumption among Black people was either uncommon or impossible. But Miller instead argued the real cause of the disease was that “even now, after thirty years or more of freedom, he [the Black person] takes but little thought for to-morrow, but to-morrow, nevertheless, comes to him and oftimes finds him wholly unprepared to meet its exacting demands.” Miller argued the only way to restore Black people to health was to return to the institution of slavery.

This was the world in which Black people lived with tuberculosis in the U.S.—one where they were told by the medical establishment that their illness was caused by weaknesses and susceptibilities inherent to their race, or else by freedom and citizenship itself. And so even after we understood that TB was an infection, we continued to blame it on the sufferer, but with a radically racialized and stigmatic lens that caused more harm to the ill than even previous forms of stigma.

It’s not an exaggeration to say that tuberculosis became a form of racialized violence. In Canada and the United States, for example, many Indigenous children were removed from their homes and forced to attend residential schools. As early as 1907, experts were sounding the alarm that this project seemed “almost as if the prime conditions for the outbreak of epidemics had been deliberately caused.” The death rate from TB in Canada’s residential schools appears genuinely unprecedented in human history.

The Canadian Public Health Association has estimated that in First Nations communities, around 700 of every 100,000 people died annually of tuberculosis in the 1930s and 1940s. Indigenous people were more than ten times as likely to die of TB than white Canadians. But in residential schools, the rate was *8,000* per 100,000—meaning that 8 percent of all kids

confined in these schools died of tuberculosis *each year*. And these inequities persist—today, Inuit people are over 400 times more likely to contract tuberculosis than white Canadians. As Lena Faust and Courtney Heffernan have written, “These deaths should not be dismissed as an unavoidable consequence of a long-standing epidemic, but as the result of deliberate neglect and mistreatment on the part of the architects of the residential-school system.”

People who are treated as less than fully human by the social order *are* more susceptible to tuberculosis. But it’s not because of their moral codes or choices or genetics; it’s because they are treated as less than fully human by the social order.

This brings us back to an important facet of understanding human responses to illness—stigma and the ethical narratives we construct around illness.

My dad had cancer twice when I was a kid, and I saw some of this up close. People said he had cancer because his parents had smoked, or because he didn’t exercise enough, or because he didn’t eat broccoli, or whatever. And it’s true that secondhand smoke and poor diet are risk factors for cancer, but it is also true that the vast majority of people whose parents smoked do not get cancer when they are a thirty-two-year-old father of two toddlers. Framing illness as even *involving* morality seems to me a mistake, because of course cancer does not give a shit whether you are a good person. Biology has no moral compass. It does not punish the evil and reward the good. It doesn’t even know about evil and good.

Stigma is a way of saying, “You deserved to have this happen,” but implied within the stigma is also, “And *I* don’t deserve it, so I don’t need to worry about it happening to me.” This can become a kind of double burden for the sick: In addition to living with the physical and psychological challenges of illness, there is the additional challenge of having one’s humanity discounted.

People living with TB today have told me that fighting the disease is hard, but fighting the stigma of their communities is even harder. The TB survivor Handaa Enkh-Amgalan writes movingly of this in her book *Stigmatized*, in which she recounts being told by doctors to hide her diagnosis lest it make her unmarriable and permanently separated from her community. TB expert Dr. Jennifer Furin once had a patient weep upon learning she had tuberculosis rather than lung cancer. “But we can treat this,” Dr. Furin told her patient. “This is curable.” Still, the young woman wished she’d been diagnosed with cancer because it would have brought less shame to her family.

Stigma is very complex, of course, but researchers have identified certain hallmarks of highly stigmatized illnesses. Chronic illnesses are more likely to be stigmatized than acute ones, for instance, as are illnesses with high levels of perceived peril. And critically for understanding tuberculosis, stigma can be compounded if a disease is understood to be infectious. Finally, the origin—or perceived origin—of a disease also matters. If an illness is seen to be a result of choice, it is more likely to be stigmatized. Mental illness is often viewed as a choice or a moral weakness, as are some kinds of heart disease and cancers. And even when there’s no evidence of a clear link between character and illness, we will invent one: It was long believed, for example, that cancer resulted from social isolation, or from bottling up one’s feelings. Even when these explanations are cruel and dehumanizing, we embrace them—because tiger got to sleep, and bird got to land, and man got to tell himself he understand.

Like all chronic illnesses that involve high levels of perceived peril, TB has been heavily stigmatized throughout history, and continues to be. Today, TB is often seen as a mark of disgrace because of its association with poverty, but it’s also often associated with perceived choice and moral failures.

When I visit with TB survivors, almost all of them cite stigma as the greatest challenge. In much of the world, it’s common for children

diagnosed with TB to be dropped off at a hospital or treatment center and then abandoned by their families. A Sierra Leonean man who'd survived a yearslong bout with drug-resistant tuberculosis told me he was afraid to visit Freetown because he did not want to run into anyone from his vast extended family, all of whom had shunned him after he got sick. He would message friends and family on WhatsApp only to be told by them that he was cursed and they wanted nothing to do with him.

A young woman who was abandoned by her family told me, "To them I am not a person." There were times that she wished she had died of TB rather than being cured of it, because the ongoing stigma was so profound. When people found out she'd survived TB, they would stop showing up for visits. The neighborhood knew, and everyone treated her like she was different. Some in her community said she got TB because her family had been punished by God. Others said it happened because they were poor, because their roof leaked, or because her mother was involved in dark magic. It may be easy to dismiss this kind of superstition, but we all unjustly stigmatize others. We all engage in the punitive act of giving a disease a meaning.

Henry was fortunate not to be abandoned by his parents, but many in his extended family severed their ties with him. Isatu, though, clung closer than ever to her only living child, and Henry felt that love at Lakka. In a poem, he wrote,

Mom you are special and beautiful
You stand closer
When everyone ran away
Especially my cousin ran away
But you stood firm.

I often think about that poem, about the astonishment of the tense change between “You stand closer” and “when everyone ran away.” Isatu’s presence remains present, even as everyone else’s presence is past.

CHAPTER 10

A STUDY IN TUBERCULIN

ROBERT KOCH WAS NOT THE only heralded genius of germ theory in his era. The French doctor Louis Pasteur rose to fame in the 1860s via his (thoroughly French) research into the role microorganisms play in the fermentation of wine. In the early 1880s, Pasteur not only corroborated Koch's findings that anthrax was caused by bacteria, but also developed a vaccine for anthrax by heating the bacterium to the point where it could no longer produce spores and then injecting it into nonhuman animals. Pasteur showed that after vaccination with these killed bacteria, the animals became inoculated against fatal anthrax infection, thereby establishing not just a cause of anthrax but a solution to it as well.

To understand what happened in the medical world after the discovery of Koch's bacillus, we need to consider a bit of geopolitics. At the time, the French and the Germans had just wrapped up the Franco-Prussian War, which was understood to be a great victory for the German people, and indeed for German nationalism as a whole. They'd laid waste to France through superior military technology but also with better medical care: The use of antiseptics during wound treatment was common among German doctors, but not among French ones, and so German casualties had been significantly lower. Medical advances had come to be seen as essential to German success. It was, therefore, deeply disappointing to German authorities when the German Koch's discovery of anthrax was usurped just a few years later by the French Pasteur's vaccine for anthrax.

I find it interesting that even here, in the supposedly pure world of science, we feel the weight of historical forces pressing in upon discovery. Our desire to create outsiders, the competition for resources among communities that would be better off cooperating, and our long history of warfare all come together in this moment of discovery.

Both French and German politicians celebrated links between medical discovery and national success. After Koch identified the bacteria that causes cholera, a banquet was held in his honor. The announcement for that banquet read in part, “Just as 13 years ago the German people celebrated a glorious victory against the hereditary enemy of our nation, so does German Science today celebrate a brilliant triumph over one of humanity’s most menacing enemies.”

But then Koch and his German brethren were scooped again, as Louis Pasteur soon developed a vaccine for cholera. And so it began to seem that Germans discovered causative agents, but only the French discovered actual cures. While Koch too became famous, he was perpetually envious of Pasteur’s success at producing real remedies. And that may be why, at the height of his fame, Robert Koch abandoned his trademark intellectual rigor and declared that he had discovered a cure for tuberculosis.

In the late 1880s, Koch began testing a potential treatment for tuberculosis using a substance he derived from the bacteria. He began with animals (Koch essentially invented the concept of using white mice for experiments), but soon decided his curative agent was safe to test on humans. He injected the translucent brown liquid, known as “Koch’s Serum,” or tuberculin, into four initial subjects: himself, his mistress Hedwig, and two assistants.

All got sick within a few hours after the injection, becoming feverish and suffering “unusually violent shivering.” But then, after less than a day of illness, they got better and fully recovered. This appeared similar to the machinations of Pasteur’s anthrax and cholera vaccines—you’re exposed to a relatively innocuous version of a pathogen, experience an immune response, and thereafter are inoculated from illness. It seemed to Koch that the body had achieved the same “fever cure.”

But Koch went even further. When he injected the serum into patients with tuberculosis, he observed that some of the tissue riddled with tubercles seemed to die off. He claimed his serum not only protected against tuberculosis infection, but also cured the disease in those already living with it.

When Koch began to share news of his remedy, the entire world took notice. Given the effectiveness of Pasteur's serums at preventing anthrax and cholera, it made sense to the public that Koch's serum also worked, especially given Koch's reputation for care and precision in his research. And so thousands of TB patients traveled to Berlin to receive Koch's cure. Many were desperately sick, and would die in train cars or hotel rooms or on the street as they awaited the remedy.

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In early 1891, news of Koch's serum reached Great Britain via a rare special supplement to the *British Medical Journal*. On the southern coast of England, another small-town doctor with big ambitions opened his mail one day and began reading of this astonishing substance. This young doctor read that Koch had discovered "a remedy which conferred in the animals experimented upon an immunity against inoculation with the tubercle bacillus, and which arrested tuberculous disease."

This idea was so thrilling, the physician later wrote, that "a great urge came upon me suddenly that I should go to Berlin. I could give no clear reason for this, but it was an irresistible impulse and I at once determined to go." And so that very day, Dr. Arthur Conan Doyle left his medical practice, packed a bag, and began his journey toward Berlin. Conan Doyle would go on to become not just one of the world's most famous novelists, but also the man who helped burst the "deceptive bubble" of Koch's cure.

It's worth trying to imagine how simultaneously thrilling and horrifying the germ theory of disease was when it first emerged. As Louis Pasteur put it,

“If it is terrifying to think that life may be at the mercy of the multiplication of those infinitesimally small creatures, it is also consoling to hope that Science will not always remain powerless before such enemies.” Pasteur acknowledged the terror felt by many—it is truly the stuff of horror movies to learn that unseen organisms are squirming in and on you, replicating in their billions until they take over your body and sicken or kill you. But he also saw the hope that accompanies better understanding.

Still, germ theory ushered in a very different world. We had imagined that having minimized deaths from lions, bears, and other predators that we had become “civilized,” a species dramatically above all others, the great power in a world of lesser life. As Conan Doyle would later write of *M. tuberculosis*, “What an infernal microbe it is!…How absurd that we who can kill the tiger should be defied by this venomous little atom.”

I should acknowledge, I guess, that one reason I’m interested in TB is that I have obsessive-compulsive disorder, and my particular obsessive worries tend to circle around microbes and illness. Before the germ theory of disease, we did not know that around half the cells in my body do not, in fact, belong to my body—they are bacteria and other microscopic organisms colonizing me. And to one degree or another, these microorganisms can also *control* the body—shaping the body’s contours by making it gain or lose weight, sickening the body, killing the body. There’s even emerging evidence that one’s microbiome may have a relationship with thought itself through the gut-brain information axis, meaning that at least some of my thoughts may belong not to me, but to the microorganisms in my digestive tract. Research indicates that certain gut microbiomes are associated with major depression and anxiety disorders; in fact, it’s possible that my particular microbiome is at least partly responsible for my OCD, meaning that the microbes are the reason I’m so deeply afraid of microbes. [*]

I imagine that had I lived in 1800, I would've still suffered from OCD, but I couldn't have feared microbial infections, because they were unknown to us. Now, though, I feel microorganisms everywhere—on the keys of my keyboard as I type, on my skin, in my mouth. Microbes challenge my very understanding of myself—what am "I," in the end, if half of me isn't me, and the half of me that isn't me dictates some of "my" thinking and feeling? What does it mean to be a person whose consciousness, whose love and longing and fear, can be snuffed out by an overgrowth of bacteria that neither love nor long nor fear? How absurd that I can be murdered by that venomous little atom!

When young Dr. Arthur Conan Doyle arrived in Berlin, he sought a meeting with Dr. Koch and, finding that impossible, showed up at Koch's doorstep. A butler invited him into the entryway, but Conan Doyle was eventually turned around without ever meeting the great German physician. Instead, Conan Doyle traveled around Berlin, seeking to understand the nature of this mysterious serum and its effect on the body. What he discovered was quite different from the remedy Koch seemed to promise. "There can be no question that our knowledge of [the serum] is still very incomplete." Conan Doyle observed that while tuberculin *did* seem to have an effect on TB patients, that effect did not appear curative. Instead, the serum seemed to kill infected tissue without eliminating the bacterium itself. "It is as if a man whose house was infested with rats were to remove the marks of the creatures every morning and expect in that way to get rid of them," he wrote. Put another way: Tuberculin was in the business of picking up rat shit, not in the business of killing rats.

René and Jean Dubos would later write that Conan Doyle's essay was "of such intelligent understanding that little of importance has since been added to his analysis of the subject." Conan Doyle immediately saw what Koch did not: that the serum tuberculin caused a strong immune response in people who'd been infected by TB, but that this immune response did not

improve the body's ability to fight off the disease. As a result, tuberculin often made TB patients *more* sick, not less. It also didn't work as a vaccine—tuberculin causes an immune response in people who've previously been infected with TB, but it doesn't prevent infection.

But Conan Doyle realized that particular facet of tuberculin could be useful as a public health tool: Because the serum caused immune responses only in people already infected with TB, tuberculin could be used to diagnose tuberculosis infection. There was a reason Dr. Koch, his mistress, and his two lab assistants all became sick after being injected with tuberculin: All four of them were already infected with tuberculosis. (Indeed, almost everyone in nineteenth-century northern Europe was.) As we've seen, around 90 percent of people infected with TB will never become sick, because the body successfully walls off the TB in tubercles. But exposure to tuberculin causes an immune response even in asymptomatic infections. While tuberculin cannot treat TB, it can *identify* TB, because only those who are infected with *M. tuberculosis* will have an immune response to tuberculin.

And so tuberculin did end up being useful—small amounts can be injected under the skin to see if a person develops swelling at the injection site, a sign the immune cells of the body recognize the TB bacterium because they've seen it before. Tuberculin skin tests can't reliably determine whether someone has active disease or which treatments are required for a particular infection, but it's a useful screening tool, especially in countries where TB infection is relatively rare.

But because Koch hastily promised not a test but a cure, thousands of people died in Berlin and around the world while taking Koch's serum. As René and Jean Dubos write, "It soon became obvious that tuberculin killed many more patients than it helped." Koch ended up disgraced, and struggled to rebuild his reputation, not least because he clung to the idea that his cure really was a cure. Conan Doyle went home to England and within a decade published his first Sherlock Holmes story, all about a detective who uses reasoning and evidence to reach rigorous conclusions

about causes of death, meaning that Holmes's work was not so distant from that of his author.

SKIP NOTES

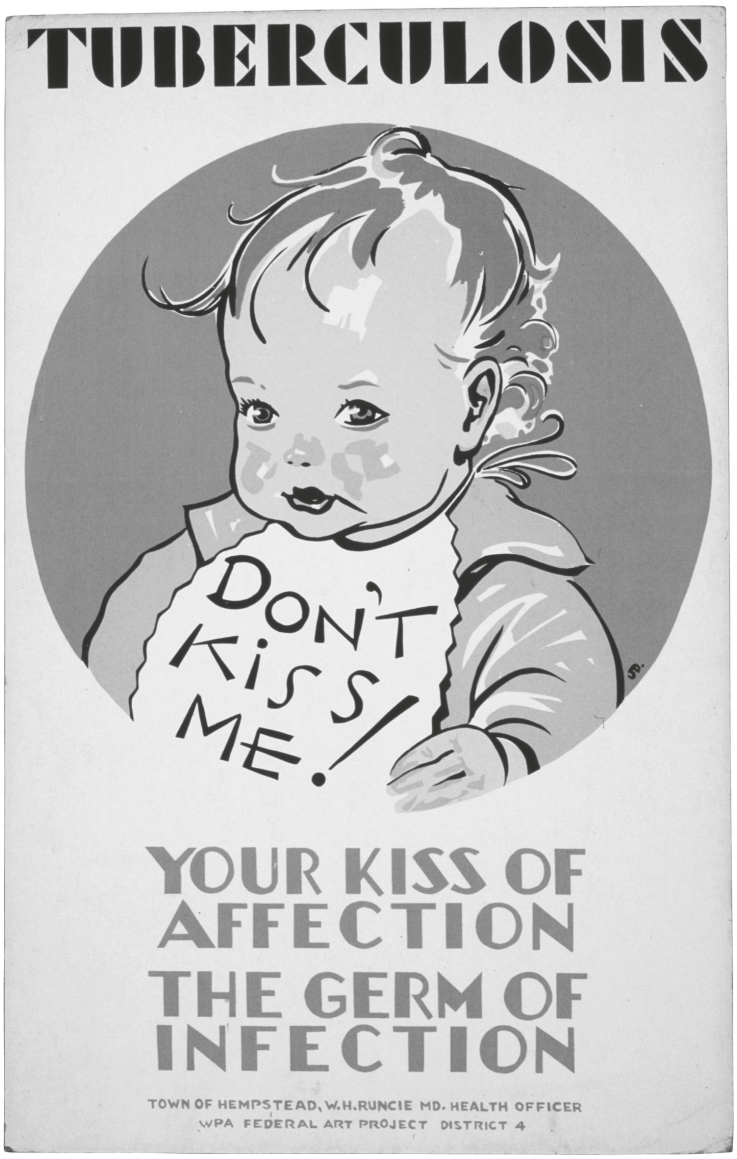
* Certain microbiomes are also correlated with the human body craving particular foods—meaning that when you're hungry for carbs or protein or whatever, it may in fact be your bacteria that are hungry for that food.

CHAPTER 11

TREPIDATION AND HOPE

IT WAS SOON UNDERSTOOD THAT tuberculosis spread mostly through the air and via infectious coughs and spit, but one could also catch it from milk contaminated with bovine tuberculosis, or in other ways. Many theorized that a popular method of transmission was inhaled dust that contained TB bacteria, or else insects that spread the germs from the ill to the healthy.

Attention first came to focus on the kinds of places and environments that seemed to foster outbreaks of tuberculosis—crowded housing tenements, dirty factories, fly-ridden environs, and the endless streams of tobacco and other spit spewing from the public. The United States of America, Charles Dickens once noted, was “a nation of spitters.” People spit on trolleys and on sidewalks, on restaurant floors and even in the home. Many public health efforts focused on discouraging or even outlawing spitting (public spitting continues to be illegal in many American municipalities), which likely did have some effect on rates of transmission. The movement to convince folks to cover their coughs and sneezes with a handkerchief or, failing that, a hand prevented even more transmission of TB (and other respiratory diseases). Public health authorities also discouraged folks from kissing babies, which may have lowered some infection risk, but definitely led to a great series of twentieth-century posters.



The quest for safe and hygienic spaces touched every aspect of life in the early twentieth century. Library books were often disinfected regularly, lest TB and other infectious diseases spread from household to household via reading. Streets were watered before sweeping lest invisible germs contained within dust particles spread to street sweepers and then into their homes and neighborhoods.

Another major focus of public health efforts was the housefly. Common in the tenements and factories where rates of TB were especially high, flies became hated and feared, as they were believed to carry *M. tuberculosis* "from the spit of the consumptive to the nipple of the baby's bottle, from

the garbage can to the lips of the sleeping child, and from the dead body to the fresh fruit," as one 1910 article put it. People were encouraged to screen in all windows and porches, and to cover their food to keep flies from it. But while houseflies can spread many diseases among humans, tuberculosis is not one of them, and anyway, houseflies are very unlikely to make you sick. As one bacteriologist recently put it, "The only way that you could come into problems is if you actually actually ate them."

People also obsessed over dirt, dust, and the places on the body where germs might find purchase. This again changed fashion, grooming, and social habits. "There is no way of computing the number of bacteria and noxious germs that may lurk in the Amazonian jungles of a well-whiskered face, but their numbers must be legion," argued Dr. Edwin F. Bowers in a 1916 magazine article called "The Menace of Whiskers." Fear of TB germs getting caught in beards led to what *Harper's Weekly* called "The Revolt against the Whisker," ushering in an era of clean shaves. For women, hemlines grew shorter as anxiety rose that floor-length dresses might pick up TB germs off dirty floors. But as Nicole Rudolph points out in *Sins against Our Soles*, "Hygiene involved far more than just the health of the body." Hygiene continued to be an excuse for treating fashion as a villain. One couldn't have too long a hemline, lest one risk carrying TB particles into the home. But one also couldn't have a short hemline, because then one might catch cold, which was believed to be a cause of tuberculosis. In short, no fashion would suffice unless it was defined as hygienic by the patriarchal medical establishment. And moral hygiene—being clean not just in body but also in mind and action—continued to be seen as essential to controlling tuberculosis. One could not drink too much, or overindulge in any other vice, without risking inviting TB into one's body.

Of his time at Lakka, Henry wrote in his memoir, "Every morning at the break of dawn, the nurses would arrive with a tray of medications, a bitter reminder of the battle within. These pills, each with its own set of side

effects, were ingested with a mix of trepidation and hope." In the days before the development of anti-TB medications, there was no tray of pills each morning, but otherwise the experience was quite similar. There was cause for hope even before cures became available—people *did* recover from TB, albeit not usually, and so there was always the possibility of survival. If one just did everything right, lived hygienically both in body and mind, listened to the doctors and the prevailing medical wisdom, then tuberculosis needn't be fatal.

That mix of trepidation and hope, so deeply felt by all who walk through the valley of serious illness, often sent people traveling in the nineteenth and early twentieth centuries. The idea of traveling to cure TB existed long before Koch and his bacillus—almost two thousand years before Koch, the tremendously influential Roman doctor Galen recommended "sea voyages" as among the cures for consumption. Aretaeus of Cappadocia recommended travel to Turkey, where time spent among the god Apollo's famous cypress groves could heal those "weak in the lungs." Since consumption was an illness of cities, it made sense to retire to quieter locales where the air was "clean" or "pure."

But nobody could quite agree on the definition of clean or pure. Should one travel to a mountaintop, as the characters do in Thomas Mann's *The Magic Mountain*? Should one move to the woods, or to the seaside, or to the desert? Should one live outdoors or indoors? Do the lungs need sunlight to recover, or only clean air? Should the air be dry as well?

Each doctor and community answered these questions differently as the concept of the sanatorium spread throughout the world in the early twentieth century. We needed dedicated facilities for the treatment of TB patients, which would serve both to improve their personal health and to end chains of exposure by removing the tubercular from their communities. Some were built in mountain climates—like the one in Asheville, North Carolina, where my great-uncle Stokes died—others in deserts or rural areas adjacent to cities. Some were even constructed in cities themselves, albeit usually on the outskirts.

The sanatorium became a fixture in the U.S. As Sheila Rothman writes in *Living in the Shadow of Death*, "By 1900, 34 sanatoriums with 4,485 beds had been opened in the United States. Twenty-five years later, there were 536 sanatoriums with 673,338 beds." At the height of the sanatorium, there were nearly as many beds to treat tuberculosis patients as there were hospital beds for all other illnesses combined.

Clean air, rest, and sunshine were believed to "infuse new hope and courage," as one person put it, and so sanatoria focused on controlling the behavior of patients and requiring them to be largely immobile and outside whenever possible.[*1] In the U.S., entire cities were founded by and for people with tuberculosis, including Pasadena, California, and Colorado Springs, Colorado. Southern California came to be known as especially salubrious, and tens of thousands of people relocated there—a movement of people rivaling the Gold Rush. These "lungers," as they were known, settled in western towns and the sanatoria that sprung up within them. If patients survived, they often stayed in their new hometowns and began families, reshaping the geography of the United States.

Henry described "a sense of interminable monotony" at Lakka, and in that way it was similar to life in sanatoria, which tended to be excruciatingly dull for the patient. The job of the "invalid," as patients were commonly known, was to improve their health. The word "invalid," of course, gets at the core of what it meant to live with chronic illness—you were a person outside of society, invalid in the social order, separated from your family and your community. Even if you convalesced at home, you were still kept from many of the rhythms of daily life. You might not have the energy or health to go shopping, or attend church, or visit family. And for those in sanatoria, life became carefully circumscribed in the name of physical and mental hygiene. The sick were often told to move very little, discouraged even from writing letters or combing their own hair. They were also told not

to feel too intensely, or drink alcohol, or have sex—all exciting behaviors that could excite the tuberculosis within.

This meant lying still in beds or chairs, which were wheeled outdoors whenever possible so that invalids could soak in sunshine and clean air. Boredom is a constant theme of memoirs and letters home from TB patients. As one sanatorium visitor wrote, “I do nothing all day but lie here staring at the mountains…. I wish they would rearrange them a bit.” But patients did find ways to amuse themselves, as humans always will. Patient-run newsletters and newspapers flourished in larger institutions, and in some cases there were even patient-run radio stations. Gossip was a source of connection and excitement, especially when it came to romances, which were frequent and occurred in spite of all attempts at controlling them. Patients were often told that romance and gossip would be deleterious to their health, and yet most invalids could not help but be, well…human.

Family members were at times told not to visit the sick, not only because visits risked spreading the infection, but also because visits were seen as detrimental to health. One sanatorium told parents, “Remember your child is sent to the Sanatorium because it is ill and needs treatment; and if the best results are to be gotten and the child is to recover in the shortest period, it must be left to our care with little or no interruption from parents, relatives, and friends.”

Rothman describes one sanatorium as “too prisonlike to be a hospital, and too hospitallike to be a prison.” In order to maximize the chance for a cure, one doctor wrote, “The smallest details of the patient’s life are controlled by the supervising physician and nothing of any importance is left to [the patient’s] judgment.” This included what to eat, when to sleep, who to see, and even what to think about. Failure to give your life over to this total supervision was tantamount to suicide. As Megan Vaughan writes in *Curing Their Ills*, “patients were frequently warned that their fate depended on their obeying the myriad rules of the institution.”

Many of these patients were children, including Gale Perkins, who contracted tuberculosis in her bones as a young child in the mid-1930s while living with her family in Boston. Although Gale traced her illness back to a TB sufferer she shared a childhood home with, kids with bone-involved TB often contract it from drinking contaminated cow's milk. Either way, bone tuberculosis could be especially devastating to children, causing painful deterioration of the bones, which in Perkins's case necessitated traction and body casts during her twelve long childhood years spent in one sanatorium.[*2]

Gale was just three years old when she arrived at Lakeville Sanatorium in Massachusetts, and like many patients, she was told that a positive attitude and absolute fealty to her physician's treatment plan were essential to her survival. She recalled, "The night was frightening to me—the darkness would start to settle in, then the stillness. The children sobbed, calling for their mothers. The nurses would come in and say, 'Silence, everyone!' " (Here we see another connection with life at Lakka a century later for Henry, who wrote that at night, "the hospital corridors seemed to stretch into infinity.")

At many institutions, crying in public was strictly forbidden, because it would harm morale and thus one's overall chance for a cure. Everything about life in Gale's sanatorium was oriented around control of the patient—patients were told when (or whether) they could read and write, how often (or whether) they could stand up, how often (or whether) they could receive visitors, and on and on. Patients spent many hours every day curing, which meant sitting or lying absolutely still, not straining even to speak or laugh.

For young Gale, visitors were very rare, and she was frequently chastised for her bad attitude and behavior. Whenever she wet the bed or cried audibly, Gale was punished. "Punishment consisted of being isolated," she wrote. "No one could talk to me; I could not play with any of my toys; a screen would be placed around my bed so I could not see the rest of the children." This happened when she was just four years old.

She found salvation in her best friend Angie, a child of Greek immigrants. They loved being friends because it afforded them two Easter

celebrations—Gale's Roman Catholic Easter and Angie's Greek Orthodox one. While Gale often got in trouble for talking or squirming in her bed when she was supposed to be quietly and calmly "curing," Angie was the perfect patient. "She would read her book of prayers every day in the early morning and evening. She would also tell me that she said prayers for me, to help me stop getting in trouble," Gale would remember. Angie was intent upon recovering and leaving the hospital partly so that she could be reunited with her sister Pauline, who wrote her frequent letters, and her father, who faithfully delivered those letters during visits each week.

One day, young Gale overheard a terrible secret about her best friend Angie: Angie's sister Pauline, who wrote her weekly, had in fact already died of TB. "But her father didn't want her to know," Gale recalled, because it might cause the kind of emotional shock that was deemed dangerous to the TB patient. So to encourage the daughter he still had, this father wrote letters that mimicked the handwriting and style of the daughter he'd lost.

Having been told that patients could not be exposed to bad news without risking their health, Gale never told Angie that her sister had died. But it didn't matter. "I saw them wheeling a stretcher with a body on it down to the morgue. I knew right away it was my best friend Angie." Gale was eight years old.

In all the heartbreaks of reading about tuberculosis, perhaps none has stayed with me quite like the image of a father trying to write in his dead daughter's handwriting to his living daughter, in the hopes that she wouldn't be crushed by the truth. In Angie's father we see the humanity of people whose lives are torn asunder by TB—a humanity that is too often denied or minimized through stigma or romanticization. He was just a father trying to do right by his kids—and then, when he couldn't, trying to do right by his kid.

This speaks so much to me about the psychosocial components of living with tuberculosis. Patients were told that good moral and physical hygiene could save them. The disease was framed, as disease so often has been, as a moral quandary—if you don't wear high heels, and you don't live unnaturally in a city, and you don't drink, and you don't cry at night when you're four years old and miss your mother, then you will survive. But patients like Gale *knew this was a lie*. They knew it because they saw their friends die. That discordance makes it even more difficult to "keep a positive attitude," which was enshrined as a treatment strategy for TB, just as it is now enshrined as a treatment strategy for diseases that are sometimes survivable but often aren't, like cancer.

Angie and Gale's experience also reminds us of another facet of sanatorium life: Control. The patient—a word that takes on new layers of meaning when one considers that many TB survivors lived in sanatoria for years or decades—had to be coddled, but most of all the patient had to be controlled, with their movements and choices and access to information severely limited. In the U.S., we still often use the phrase "TB control" in public health departments, whereas for illnesses like cancer we're more likely to use the phrase "cancer *care*." This "control over care" dynamic, as the physician and anthropologist Paul Farmer termed it, is seen with many infectious diseases.[*3]

Eventually, in her adolescence, Gale was able to go home more often, but disease and its fears followed her even after she received streptomycin, the first drug synthesized to treat TB, in the late 1940s. As another childhood resident of sanatoria wrote, "There was such a stigma to having tuberculosis that people who had it would try to hide it in an effort to avoid prejudice and hostility." Many were instructed to tell friends and family that they'd been hospitalized not for TB but for polio—although, of course, polio was also an infectious disease, it was less stigmatized than TB.

Gale survived. She married, had three kids, and led the occupational therapy unit at a small hospital. She was also able to pursue a passion she first discovered at the Lakeside Sanatorium trying to cheer up the other kids with TB by making silly faces: She became a clown.

In thinking about Gale, I am reminded so much of Henry, who also endured so many lonely nights, who also saw friends die, who also made silly faces to cheer up his fellow patients, and whose movements and choices were also controlled by a public health system wary of trusting the TB patient.

Many patients did recover in sanatoria—rest and adequate nutrition are better for the body than malnourishment and stress—but recovery rates don't seem to have been much higher at sanatoria than for those who lived at home. Nonetheless, separating millions of the ill from their homes did decrease the spread of the disease within families, and combined with better overall nutrition and safer housing, rates of tuberculosis declined globally in the first half of the twentieth century. In the U.S. and other wealthy countries, the rate of decline was precipitous—between 1882 and 1930, when my great-uncle died of TB, overall mortality from the disease in the U.S. dropped by around 80 percent.

But those improvements were not evenly distributed in the American population—the declines for African Americans and Chinese Americans were much lower, and for Indigenous people, there was very little decline at all. And, for everyone, tuberculosis remained fundamentally incurable.

SKIP NOTES

*1 The reclining wood-slatted chair known as the Adirondack Chair was invented for TB patients, allowing them to rest outdoors without needing their beds wheeled outside.

*2 Eliminating the risk of bovine TB is one of the reasons we began pasteurizing milk; no doubt Dr. Koch would be bummed to learn we don't call it Kochizing milk.

*3 It's critical to control outbreaks of infectious disease, but such efforts can be counterproductive if elements of care are abandoned in the pursuit of control. Many TB

survivors have described to me the dehumanizing process of receiving their medication, for instance. More than one has told me that they were told to stand in a corner and then thrown their medication from across a room because healthcare workers were so afraid of TB. But with appropriate masking and infection controls, there's very little risk to healthcare workers if they hand medications directly to those living with TB. This sort of basic humanizing treatment goes a long way toward helping those with TB complete their treatment regimens, which is to say that care-focused treatment often controls the disease better than control-focused treatment.

CHAPTER 12

THE CURE

IN THE DECADES AFTER THE discovery of Koch's bacillus, small improvements emerged. Better diagnostics meant the disease could be identified and treated earlier, especially once chest X-rays emerged as a diagnostic tool.

X-rays could see evidence of the disease before it became symptomatic. By the 1930s, there were a variety of ways to "see" into the living body—the stethoscope allowed us to hear the heart and lungs; surgery, while it remained dangerous, was less likely to be fatal thanks to antiseptics. But the X-ray was different, because it revealed the body visually without the need for cutting the body open. No longer would the skin render the body's interior opaque.

Dr. Alan Hart helped pioneer the use of chest X-rays to diagnose tuberculosis. Hart was married to a woman and practicing medicine in San Francisco in 1918 when he was outed as a trans man by a former colleague. Dr. Hart was chased out of town on the back of headlines like "Girl Poses as Male Doctor in Hospital" (he was not posing, of course) and spent much of his life moving from town to town to escape various forms of transphobia. Hart was also a novelist, and wrote of one of his characters, "When it came to outrunning gossip he found he couldn't do it," which was Hart's experience as well—he moved seven times in nine years all around the U.S. in search of safety, but it always proved fleeting. He did manage to get a graduate degree in radiology, though, and helped show how chest X-rays could show very early signs of tuberculosis, thus allowing patients the

opportunity to rest and get adequate nutrition sooner, which contributed to better outcomes. Chest X-rays continue to be an essential diagnostic tool; mobile chest X-ray machines that can be carried via backpack now serve rural communities, so Hart's popularization of this diagnostic method continues to save lives.[*1]

But even with better diagnostics, effective treatments proved difficult to come by. A popular intervention involved intentionally collapsing the lung so that it could "rest," which had limited benefit.[*2] The primary strategies for healing TB continued to be rest, travel, and adequate nutrition, all of which had been promoted for thousands of years.

Prevention strategies saw faster progress. The widespread phenomenon of cows infecting humans with tuberculosis decreased with the advent of tuberculin-based testing of cow herds alongside the pasteurization of milk. And most critically, thanks in part to those cows, a vaccine emerged. The Bacillus Calmette-Guerin vaccine, or BCG, was named for its French developers, who hypothesized that just as exposure to the cowpox virus inoculated humans against smallpox, exposure to attenuated bovine tuberculosis bacteria might inoculate humans against tuberculosis. The researchers eventually stumbled upon a strain of bovine tuberculosis that was far less virulent, which they grew in a medium that mixed potatoes with beef bile. That strain became the basis for the BCG vaccine, which first became available to people in 1921.

Over a century later, BCG is still our only vaccine for TB—although by 2024, there were at last promising candidates in development. The efficacy of BCG is one of the most debated questions in all of public health, but what's agreed is that:

1. BCG is effective at preventing severe illness in children, especially children under the age of five.

2. BCG is not particularly effective—and maybe not at all effective—at preventing infection, serious illness, or death in adolescents or adults.

Even if the vaccine is administered multiple times, it simply does not seem to prevent much, if any, illness among most adults and teens.

3. For reasons we don't fully understand, efficacy seems to get worse as one gets closer to the equator.

Today, it's common to receive the BCG vaccine in infancy if you're born in a country with high rates of tuberculosis—most children in Sierra Leone receive it, for instance—and because it prevents a lot of child deaths, it is an important tool for prevention. But the vaccine could never, and will never, stop TB on its own.

A 1941 history of pulmonary tuberculosis proclaimed, "The El Dorado of cure seems still far in the distance." But in fact, that cure was only a few years away, which is a reminder that we can never see the future coming.

We tend to solve the problems we pay the most attention to, and in 1941 we paid a lot of attention to tuberculosis. It was the world's leading infectious cause of death and disability, and millions of people globally were residents of sanatoria. Even in the 1940s, TB infections and disease remained common—twentieth-century TB survivors include Beatle Ringo Starr (who was institutionalized with TB as a teenager), the novelist George Orwell (who died of TB in 1950, just as curative treatment was becoming available), the writer Thomas Wolfe (who died of tuberculous meningitis in 1938), and the actor Vivian Leigh (who lived with TB for over twenty-five years).

As late as 1941, only around a quarter of tuberculosis patients could expect to recover. For these lucky few, their immune systems would eventually find a way to restore balance between the body and the disease, walling off the bacteria in tubercles and allowing the patient to go on living. Some small percentage would live with the disease as invalids—never quite recovering, but not dying of it, either; these folks lived in limbo before

eventually dying of something else, often exacerbated by a decades-long battle with chronic illness. But more than half of TB patients with active disease died of their sickness, no matter how clean the air they breathed or how still they lay while breathing it.

And then, quite suddenly, tuberculosis became a treatable disease, and then a curable one. At first blush, it may seem to be a quirk of history that several effective anti-tuberculosis drugs were developed in the 1940s and 1950s. But in fact, all these drugs were the product of decades of research on bacteria and how to kill them. Throughout the late nineteenth and early twentieth centuries, researchers circled closer and closer around antimicrobial fungi and compounds. In Sweden, the physician Jürgen Lehmann became intrigued by a paper indicating that exposing tuberculosis to aspirin caused *M. tuberculosis* to take in more oxygen; he hypothesized that a different acid compound might inhibit the bacterium's growth by slowing its metabolism. This turned out to be true, with the compound para-aminosalicylic acid (commonly known as PAS) proving the most effective at inhibiting the bacteria's growth.

Meanwhile, graduate students at Rutgers University, including Albert Schatz and Elizabeth Bugie, isolated an antibiotic known as streptomycin, publishing their findings in early 1944. American soldiers with severe infections began receiving the drug a year later. The first patient died. The second survived but was blinded (an occasional side effect of the medication). The third patient was a critically ill young Army officer named Bob Dole, who would survive, and go on to become a U.S. senator, Republican nominee for president, and noted Viagra spokesperson.

In Massachusetts, Gale Perkins began receiving streptomycin in the late 1940s, when she was sixteen, and was finally able to permanently leave her sanatorium, where she'd lived since she was three. Everywhere these drugs appeared, TB death rates plummeted—in the U.K., rates of tuberculosis deaths fell by 90 percent in the decade after streptomycin was first introduced.

To augment the power of streptomycin, a previously existing drug, isoniazid, also proved effective against TB. By 1952, another previously

existing drug, pyrazinamide, was shown to kill tuberculosis bacteria.

Soon, physicians started experimenting with combinations of these drugs to cure TB, and by the mid-1950s combination therapy including all three of these drugs was tested and approved. Tuberculosis became curable for a great many of those infected. It's hard to overstate these drugs' transformative impact on TB treatment: Even today, both isoniazid and pyrazinamide are part of the first-line RIPE treatment protocol.

By the late 1950s, the illness was broadly curable. Sanatoria, which overflowed with patients just a decade earlier, emptied out in the United States and Europe. The discovery of two more antibiotics—ethambutol in 1961 and rifampin in 1966—led to the advent of that RIPE protocol, and soon people began to proclaim that TB was on the edge of eradication. Louis Pasteur's encouragement that "science will not always remain powerless before such enemies" had come to fruition, and now it would only be a matter of time before TB ceased to be a public health concern.

But as physician and researcher Annik Rouillon wrote in 1991, "In the long history of tuberculosis in man, despair follows hope, triumph and tragedies succeed each other." Here again we see the cost of human biases, and how the repercussions of those biases are borne by the poorest and most marginalized among us. Even as TB became curable, the cure often did not reach the places that needed it most. By 1980, the RIPE treatment protocol had been in use for decades in the U.S. and western Europe. Efforts at case-finding and prevention meant that rates of TB had dropped in rich countries so dramatically that tuberculosis felt like what it should have been: history.

But in dozens of countries, treatment either wasn't available or reached patients only sporadically. From India to Bolivia to Cambodia to Ethiopia, low- and middle-income nations continued to have TB death rates higher than those seen in the U.S. before the antibiotic era. In Ethiopia, for instance, TB mortality rates in 1990 resembled those in the U.S. in 1882, the year Robert Koch identified the venomous little atom of *M. tuberculosis*. It was as if the cure did not exist—because the disease was where the cure was not, and the cure was where the disease was not.

These failures largely resulted from rich communities understanding tuberculosis through the lens of racism and colonialism. It was too often assumed that delivering RIPE protocols to poor communities was either impossible or else inadvisable, because—as S. Lyle Cummins put it at the beginning of the antibiotic age, the “African native” was too “child-like in respect to mentality and outlook” to be trusted to take or dispense medicine as prescribed.

SKIP NOTES

*1 Hart eventually moved to Connecticut, where he fundraised for TB patients who couldn’t afford treatment and served as a public health official. He and his wife, a professor at the University of Hartford, lived in Connecticut for the last fifteen years of Hart’s life, before he died in 1962 at the age of seventy-one.

*2 It did help in one sense: *Mycobacterium tuberculosis* is highly aerobic, so it loves oxygen, and the collapsing of a lung deprived the bacteria in the lung of oxygen, thereby making it harder for the bacteria to survive. Also, collapse therapy probably stopped some TB deaths that would’ve occurred due to massive hemorrhage, since when the lung collapses down, the vessels don’t bleed as much.

CHAPTER 13

WHERE THE CURE IS NOT

THERE ARE MANY ACRONYMS IN the world of tuberculosis. Global health, like any field, loves to shorten its phrases to make them obvious to experts and inaccessible to neophytes. From BPaLM to PMDT, from GDF to ERP, there's a pretty good chance that if you just string some letters together, it'll mean something in the context of TB. But no acronym has left such a profound imprint on the field as DOTS—Directly Observed Therapy (Short-course), a method of delivering anti-TB drugs to impoverished communities first developed in the 1970s.

Less than fifty years after Thomas Mann published the classic TB novel *The Magic Mountain*, tuberculosis—and the sanatoria that supported TB patients—was mostly a memory in Europe. For one example, take Switzerland, where *The Magic Mountain* is set. By 1970, Switzerland saw fewer than ten TB deaths per hundred thousand residents.

However, in many new nations emerging from colonization—from Vietnam to Sierra Leone to Belize—rates of tuberculosis remained very high, partly because of the poverty and malnutrition that make TB more likely to develop into active disease, and partly because the cure—so well-established in Europe, the U.S., and other rich countries—was almost never available to poor people living with TB.

In her book *An Introduction to Global Healthcare Delivery*, Dr. Joia Mukherjee explains how in the years after formerly colonized nations achieved independence, when the recently created World Bank offered loans to poor countries, the Bank's policies profoundly shaped the

healthcare systems in those countries. Restrictions on how much governments could spend and how they could spend it led to tragic underfunding of healthcare and education systems. “By the late 1980s,” Mukherjee tells us, “health budgets in many African and Asian countries were less than $5 per person per year.”

Before DOTS, efforts to expand access to TB care in low- and middle-income countries were haphazard and inconsistent. Drugs were often out of stock, which is especially dangerous when it comes to tuberculosis because interruptions in care can lead to drug-resistant TB, which is vastly harder to cure. But beginning in the 1970s, the physician Karel Styblo, who was himself a TB survivor, began implementing a strategy, primarily in Tanzania, to offer systematic treatment to those living with tuberculosis. The guidelines were:

1. TB would be diagnosed through smear microscopy. This method of diagnosing TB, essentially identical to the way Robert Koch first identified *M. tuberculosis* in 1882, involves looking for the bacteria in a sputum sample. Smear microscopy misses around 50 percent of cases and is especially likely to miss cases in children (indeed, Henry was smear-negative when he was first tested for TB), but it’s much less expensive than the more sensitive chest X-ray. Styblo’s strategy maintained that by only diagnosing with smear microscopy, health systems would focus on the most infectious and severely ill patients (who are more likely to test positive via microscopy).

2. Treatment would be highly standardized. Everyone would receive the RIPE regimen (or a closely related one) for between six and nine months.

3. Patients would be “directly observed” taking their medication each day by someone other than a family member. Often, this means patients have to make their way to a clinic each day in order to receive their medication and be observed while swallowing the pills to ensure compliance.

4. Standardized reporting would allow better statistics on cure rates and continuous drug supplies would prevent stockouts.

This strategy, which came to be known as DOTS, aimed to solve multiple problems within the setting of impoverished healthcare systems. It would address supply chain and diagnostic challenges by making both diagnosis and treatment standardized and cost-effective, and it would minimize or prevent further drug resistance by ensuring through direct observation that people being treated for TB swallowed their medication.

It’s very common to hear that one of the biggest drivers of drug resistance is patients “failing to take their meds.” This so-called “patient noncompliance” is indeed a central factor driving antibiotic resistance to tuberculosis. (Henry’s original drug resistance from when he was a young child, for example, was most likely caused by his father insisting upon stopping treatment.) For a variety of reasons, many patients struggle to complete their lengthy antibiotic regimens, thereby giving the infection more opportunities to evolve resistance to treatment. As the Ethiopian doctor Girum B. Tefera once explained to me, “There are so many factors. Some factors are they can’t access diagnosis or treatment. If the patient has to travel miles just to be diagnosed and to be treated, this is very expensive. They have to find a place to sleep. They have to find money for a motorbike taxi. This prevents a patient from seeing a doctor. Another factor is inconsistent or frequently interrupted supply of TB commodities, including TB drugs, that force patients not to be compliant to the treatment.”

Of course, this is not only a problem with tuberculosis. Even with much shorter antibiotic regimens, failure to complete therapy is common. In the

U.S., more than a quarter of all prescribed courses of antibiotics go unfinished. I've met with tuberculosis patients who stopped taking, or paused taking, their medications for a huge number of reasons. One young woman I spoke to was nineteen when she was diagnosed with TB. She abandoned treatment after her boyfriend moved to a neighboring community and she followed him. Another patient I spoke to abandoned the pills because they made her terribly sick when taken without food, which she could not afford. Her doctor advised her to try taking the medicine with water that had a bit of sugar poured into it, but even so, her stomach hurt so much after taking the medicine that she just couldn't do it. Others were living with substance use disorders that made it difficult to get to the clinic every day, or they couldn't afford transportation, or they felt better and didn't want to put up with the side effects anymore.

I once visited with a young man at Lakka who struggled to take his medication. He was in his early twenties and wore a dirty T-shirt, jean shorts, and sandals. He suffered from severe depression—his voice barely rose to a whisper and he'd lost both many friends and his romantic partner when he was diagnosed with TB. He told me he felt like he could never return to the world where he had lived; now, he was, as the interpreter put it, "disgraced."

A dirty mask hung limply below his nose. His huge eyes were sunken into his face. He answered my questions with a single word whenever possible: Do you still see your friends? *No.* I would imagine that's really painful. *Yes.* And to be cured of TB, this man had to take medicine every day for months—medicine that made him feel sick, that can make you vomit and render your vision blurry and give you jaundice. You have to take these medications for months even after you feel better, and you have to be watched while taking them, like you are a prisoner.

Of course, patient noncompliance is not only an issue in the world of TB. I have often struggled to take the medication that keeps me alive. Every day, I swallow two pills to treat my OCD and depression, and over the last decade, I've stopped taking them several times, even though I understand intellectually that by doing so, I am putting my life at risk. And so as I

spoke to this young man, I pulled my own bottle of pills out of my backpack and explained to him that I simply don't understand why it is so hard for me to take this medicine. My doctor makes it easy. There is no DOTS for me. I do not have to stay at a hospital or walk to a clinic to receive treatment; I can pick up my medicine at the neighborhood pharmacy once a month. And yet as I told this young man, it is still very difficult for me to take my medicine. *Why?*

The question brought him out a little. He wondered if maybe I did not like the side effects. It is true that I don't like them. I offered that maybe stigma has something to do with it—I feel like I am dependent upon the drugs, like I am not self-sufficient in the way that I am supposed to be. He encouraged me to think of my family, and my future, when taking the medicine. I encouraged him to do the same. It's hard for some people, myself included, to take medicine. I don't fully know why. But I can't very well blame others for not finishing their antibiotics when I know how often I've failed to finish my own.

In his seminal paper "Social Scientists and the New Tuberculosis," Partners In Health cofounder Dr. Paul Farmer outlined the story of one "noncompliant" patient, a nineteen-year-old Haitian man named Robert who got sick in the early 1990s. After being diagnosed with TB, Robert began treatment, but the hospital where he was treated only had two of the four DOTS-approved medications he should've been receiving. (This was—and in some places remains—a problem with DOTS: Stating that all relevant drugs should be available with no stockouts is easier than actually making it happen in poor countries like Haiti.) Robert wasn't allowed to be given more than a day's supply of medicine at a time, so he had to make a two-hour bus trip to the hospital each day, forcing him out of work.

After several months, Robert began walking each day to a better hospital, where three of the four drugs were available. The drugs were expensive—Robert's family had to sell more than half their land to

purchase the medicine. But the drugs were also insufficient, so four years after he initially experienced symptoms, Robert finally entered an in-patient facility in the Haitian capital of Port-au-Prince. There he received six months of the four-drug RIPE cocktail, but by then his TB had mutated to become resistant to standard therapy. Although second-line antibiotics probably would've cured him, they weren't available in the hospital at the time. Robert eventually sought care from an adequately stocked clinic in central Haiti, but by then he was too sick. Robert did everything right—traveling at considerable cost and inconvenience to wherever the best care available was—but he still died in severe pain in December of 1995 at the age of twenty-eight.

And so, as historian Christian McMillen has written, "The terms 'compliance' and 'adherence' or whatever other term might be deployed are all too confining. What does a national TB program's inability to keep track of patients on treatment have, necessarily, to do with *patient* compliance or adherence? When a program loses a large percentage of its patients, is this a compliance problem or a surveillance problem? Is it a patient's fault when he or she cannot afford the food necessary to ward off the hunger brought on by the drugs?"

More broadly, is it a patient's fault if they are too disabled by depression and isolation to follow through on treatment? Is it a patient's fault if they or their children become so hungry that they feel obliged to sell their medication for food? Is it a patient's fault if their living conditions, or concomitant diagnoses, or drug use disorder, or unmanaged side effects, or societal stigma result in them abandoning treatment?

Why must we treat what are obviously systemic problems as failures of individual morality? Many patients have described the experience of receiving their drugs as humiliating—they may be handed their medicine while being told that this only happened because they were unclean or poor or otherwise lesser.[*] This is often not an environment patients are excited to return to—and yet somehow we always seem to blame the patient for noncompliance, rather than blaming the structures of the social order that make compliance more difficult.

The term “compliance” itself reveals what all of this is really about, deep down, which is systems of medical resource distribution exerting the same kind of control over TB patients in the twenty-first century that sanatoria exerted over TB patients in the twentieth.

I want to be clear that DOTS has saved many lives—the UN estimates that over six million lives have been saved since 1995 through people receiving DOTS. It’s cheap and effective—as long as you are in the lucky group who test positive through smear microscopy, who don’t have drug-resistant TB, and who are able to complete treatment. And it’s certainly an improvement on what came before, because before DOTS there was *no* comprehensive global strategy for addressing TB in poor countries.

But randomized control trials have found that directly observed therapy is no more effective than giving patients their pills to take home in two-week or monthlong cycles, provided the patients are adequately supported. DOTS also failed to address the growing crisis of drug-resistant tuberculosis, and failed to identify many cases of TB because smear microscopy is so much less sensitive than chest X-rays. But even in 2025, DOTS remains standard practice in much of the world.

For many people living with TB, daily travel to a clinic proves complicated or impossible, especially when they are very unwell, but such is the fear of antibiotic resistance and the distrust of patients that global health officials have long deemed DOTS necessary. When I asked TB expert Dr. Jennifer Furin about this protocol and forcing people to be visually observed taking their pills each day, she told me, “I know of no other field of medicine where therapy is based so completely on lack of trust toward patients.”

And so DOTS, even as it expanded treatment dramatically, has also continued the longstanding pattern of distrust and stigmatization of tuberculosis patients. It also offered only one solution. As the TB researcher

Dr. Carole Mitnick explained, “Like any one-size-fits-all solution, DOTS will fail a lot of people.” One of those people, of course, was Henry.

Henry is an interesting case study because he received DOTS the first time he became sick with tuberculosis when he was five. At the time, there were no other treatments for TB available in Sierra Leone and most other poor countries. Henry may have failed treatment due to so-called “patient noncompliance” in the sense that his father removed him from treatment prematurely. But will we see Henry as a human individual who wrote lovely paragraphs and poems, who encouraged not just fellow TB survivors, but also his caregivers? Will we see him as a valuable person interwoven into the one human story? Or will we see him as a noncompliant five-year-old?

There is a benefit to systematizing healthcare, to treating everyone like they are everyone else. But there is also a cost.

SKIP NOTES

* One passage from Andrew McDowell’s brilliant book *Breathless* has stuck with me: When asked to take off his shirt for an X-ray, a patient coughed while removing it. “ ‘Don’t cough in here,’ the technician barked. ‘First thing in the morning and they’re already coughing on me.’ ” This technician’s inability to see the humanity of his patient—not even addressing the person directly but lumping them in with all other TB survivors—is of course understandable. The technician did not want to risk contracting TB. But of course the patient also did not want to cough, did not want to be sick, did not want to be shouted at for being ill.

CHAPTER 14

MARCO. POLO.

OVER 90 PERCENT OF PEOPLE who get sick with TB have "drug-susceptible" tuberculosis, which means that the RIPE regimen—whether implemented by DOTS or through any other strategy—can usually cure it.

But about half a million people each year become sick with drug-resistant tuberculosis, which won't respond to one or more of these first-line antibiotics. Such cases, known as DR-TB or MDR-TB or XDR-TB[*1] or by a stunning array of other acronyms, are harder and more expensive to treat, but still usually curable.

Almost immediately after the cure emerged in the 1940s and '50s, a new fear emerged: The cure would be temporary. The bacteriologist Mary Barber showed as early as 1947 that *Staphylococcus aureus* was evolving to select for penicillin resistance, writing the next year that "the present widespread and often indiscriminate use of penicillin, particularly as a preventive measure is seriously menacing its future." (Indeed, when Barber wrote those words, around 40 percent of staph infections were already resistant to penicillin; today, over 98 percent are.)

TB takes a long time to kill because it has an unusually waxy and thick cell coating, which also means the bacteria have a long time to be exposed to their killers and try to develop resistance to them. But working against TB is its extremely slow replication rate.

All four of the drugs involved in the first-line RIPE antibiotic protocol were introduced between 1946 and 1966, and over half a century later the vast majority of TB cases can still be cured with it. So it's not like antibiotic

resistance to TB has overwhelmed us with its speed. *Staphylococcus aureus* replicates up to ninety times faster than TB, which gives staph millions of opportunities each day to evolve toward drug resistance. *Mycobacterium tuberculosis* simply has fewer opportunities, but even so, given enough time, the bacteria will succeed.

Antibiotic resistance is a complex and many-tentacled beast—countless factors from overprescription to antibiotic use in livestock have contributed to it. But in considering the rise of multidrug-resistant tuberculosis in particular, it's important to note that we are in this mess first and foremost because we stopped trying to develop new treatments for tuberculosis. The real issue is not that TB is uncommonly good at selecting for resistance. The real problem is that in the forty-six years between 1966 and 2012, we developed no new drugs to treat tuberculosis.

This strikes me as one of the stranger choices in human history. Humans, a species that simply can never have enough, somehow decided that five or six anti-TB drugs would be plenty.

Why? New classes of drugs to treat bacterial infections are not easy to find, but we know they are *possible* to find. In the last couple decades as economic incentives have shifted, we've been able to develop powerful new medications to treat TB, including bedaquiline and delamanid.

But there is limited profit motive.[*2] The underinvestment in new classes of drugs to fight bacterial illnesses is the central cause of growing antibiotic resistance.[*3] It's easy to blame patients or providers or pharmaceutical companies, but really all of humanity has collectively chosen not to put more of our shared resources toward new treatments for disease. Some of this can be chalked up to our economic systems—the newest antibiotics will not be prescribed as often, meaning they won't be as lucrative as, say, developing a drug that hundreds of millions will take to control blood pressure. This is why when new antibacterial drugs *do* come out, they are often priced very highly.[*4]

But the market need not be the only determinant of human health. Instead, we could invest more public and philanthropic money into research and development of drugs, vaccines, and treatment distribution systems. We

could reimagine the allocation of global healthcare resources to better align them with the burden of global suffering—rewarding treatments that save or improve lives rather than treatments that the rich can afford. When markets tell companies it's more valuable to develop drugs that lengthen eyelashes than to develop drugs that treat malaria or tuberculosis, something is clearly wrong with the incentive structure. And we are not stuck with that incentive structure. I know, because the two most recent drugs developed to treat TB, bedaquiline and delamanid, were both funded primarily by public money.

Different combinations of drugs are used to treat drug-resistant TB. The most effective and least toxic regimens in 2025 consist of five to seven pills taken daily for between six and nine months, but it is still common to receive a cocktail of drugs known—somewhat menacingly—as "the injectables." The injectable cocktail involves a mix of pills and injections of highly toxic drugs.

When I met Henry at Lakka, he had been hospitalized for two months and was receiving the injectables regimen. Henry's treatment included the drug kanamycin, which is known to be ototoxic, causing permanent hearing loss in over 20 percent of people who take it, often resulting in lifelong total deafness. Kidney failure is another common side effect. Other drugs in the cocktail can cause severe liver damage.

Henry should never have received kanamycin. Bedaquiline, which can be taken orally, had been approved by several national health systems (including the U.S.) by 2013. When used in combination with older drugs like linezolid and pretomanid, bedaquiline is not just a powerful drug that helps cure tuberculosis; it also has a far better safety profile than kanamycin. There is no risk of hearing loss with bedaquiline-based treatment regimens. (In 2024, there were at least four treatment regimens involving bedaquiline that were safer and shorter than the injectables-based regimen, and yet still hundreds of thousands of patients around the world

received the injectables, causing tens of thousands of cases annually of hearing loss and/or kidney failure.)

So why wasn't Henry on bedaquiline? Although most of the money that went into developing bedaquiline came from the public (much of it from the U.S. government), the drug was owned and patented by Johnson & Johnson, which had a monopoly and therefore absolute control over the price. A course of bedaquiline can be produced profitably for $130,[*5] but during their monopoly Johnson & Johnson charged much more than that for a course of bedaquiline, making the drug far out of reach for the Sierra Leonean Ministry of Health. As a result, bedaquiline wasn't available to Henry, so he received the injectables.

Shortly after I visited him, he woke up one day only able to hear out of one of his ears. He didn't tell his doctor or any nurses. He knew there was nothing they could do, and anyway, he was scared they would discontinue treatment, which would spell his death.

Johnson & Johnson would later say, "It is false to suggest—as some recently have—that our patents are being used to prevent access to SIRTURO (bedaquiline), our medicine for MDR-TB." But I would challenge them to look into Isatu's eyes and tell her that J&J's price gouging had nothing to do with a medicine funded primarily by the public being unavailable to the most vulnerable members of that public. In fact, many people have died waiting for bedaquiline. Among them was an Indian TB activist named Shreya Tripathi. One of her caregivers, Dr. Jen Furin, would later tell me that Shreya loved a book I'd written called *The Fault in Our Stars*. In a remembrance of Shreya, Dr. Furin wrote, "Shreya's sister had given her the novel after Shreya became too breathless to leave her bed, the result not only of an infectious TB pathogen but of a society's unwillingness to help her survive."

When Shreya first read about bedaquiline while researching her diagnosis of XDR-TB, she knew immediately bedaquiline was "what my body needs." Her doctors agreed, but the national TB program in India at the time denied her doctors' requests for a bedaquiline-based treatment. They argued that 1. Bedaquiline didn't have enough evidence to support

treatment for Shreya's strain of TB (which was untrue; by the time Shreya got sick, thousands of people with XDR-TB had been successfully cured with bedaquiline), 2. It was cost prohibitive (which was only true because J&J chose to make it so), and 3. It was imperative to "protect" bedaquiline for people who may need it in the future. Prescribe too much bedaquiline, the argument goes, and TB will only grow resistant to it as well.[*6]

Shreya sued the Indian government to get access to bedaquiline. She recognized her case might not move quickly enough to matter for her, but as she told her father, she wanted her suffering to have meaning. Shreya won the lawsuit in the high court of New Delhi, forcing the government to make bedaquiline available, but the victory indeed came too late for her. By the time she received her first dose, Dr. Furin would later write, "Shreya's lungs were destroyed. Bedaquiline along with other medications was able to kill the TB germ, but nothing could be done to make the cells of her lungs healthy again: only scars remained."

Shreya died in 2018, six years after being diagnosed with tuberculosis. Dr. Furin would later tell me that Shreya was rereading *The Fault in Our Stars* in the last days of her life, relating to the breathlessness of the novel's narrator, who lives with cancer that has metastasized to her lungs.

When you write a novel, you are alone in it. I wrote that book alone, sitting in airports and coffee shops and lying in bed. But when writing, there is always for me a hope that one day I will not be alone—not in this work and not in this world. It is a bit like that old children's pool game Marco Polo, where one person closes their eyes and swims around the pool trying to tag someone else. "Marco," the person with eyes closed says, and the other pool-goers have to answer, "Polo." "Marco, Marco, Marco," cries one kid, and the others reply: "Polo. Polo. Polo." Writing is like that for me, like I'm typing "Marco, Marco, Marco" for years, and then finally the work is finished and someone reads it and says, "Polo."

And so here is Shreya, saying “Polo” to me from across the great divide. But she is also saying “Marco.” She is also telling me to hear *her* voice, and answer *her* call. People often ask me why I’m obsessed with tuberculosis. I’m a novelist, not a historian of medicine. TB is rare where I live. It doesn’t affect me. And that’s all true. But I hear Shreya, and Henry, and so many others calling to me: Marco. Marco. Marco.

SKIP NOTES

*1 Drug-Resistant TB, MultiDrug-Resistant TB, and eXtensively Drug-Resistant TB, respectively.

*2 Then again, there is limited profit motive in a lot of things we do reasonably well—from space travel to Wikipedia editing.

*3 I am living evidence of the benefit of developing new lines of antibiotics. In March of 2007, my left eye began to swell. I was eventually diagnosed with orbital cellulitis, an infection in the tissue between my eye and brain. Orbital cellulitis can be quite serious—it’s common for patients to lose sight in the affected eye, and the infection sometimes crosses over into the brain, where it is usually fatal. In my case, the bacteria causing the infection was a drug-resistant strain of *Staphylococcus aureus*. By the time I was hospitalized for the disease several months after first seeing a doctor, I was quite sick and had been treated with a number of antibiotics. The infectious disease specialist at the hospital asked me if I’d been treated with this medicine or that one. “Have you taken a yellow pill? A round pill?” The answer to every question was yes. At last he asked, “Have you taken a medicine that costs $700 per pill?” *No*, I said. “Well,” he said, you’re about to.” That drug, which eventually cured my cellulitis, was only a couple of years old.

*4 Hence my seven-hundred-dollars-per-pill cellulitis treatment.

*5 We can be sure of this because J&J now charges $130 for a course of bedaquiline and there have been no notable changes in the manufacture of the drug.

*6 Of course, allowing drug-resistant tuberculosis to go untreated in a patient makes that patient vastly more likely to spread the disease to others, thus furthering the spread of drug-resistant TB.

CHAPTER 15

DR. GIRUM

A FEW MONTHS AFTER I met Henry, a new doctor arrived at Lakka. Dr. Girum Tefera grew up in Ethiopia on the outskirts of Addis Ababa. His father was a schoolteacher, and Girum heard over and over that education was the key to success. He was a good student, although he often had to switch schools as his father's job changed. As a kid, Girum wasn't sure if he wanted to pursue engineering or medicine until seventh grade, when he saw his mother suffer seizures from her uncontrolled epilepsy. "Seeing my mom seizing when the epilepsy wasn't controlled made me think: If I became a medical doctor, I could at least help my mother."

Dr. Girum, as his patients call him, attended university in the far north of Ethiopia and began studying tuberculosis after a mentor helped him see the scope of the global tragedy. When I first met Dr. Girum, he told me, "What makes me want to remain a tuberculosis doctor is I see a lot of helpless people. Most of the people coming in with a diagnosis are from poor and marginalized families. What encourages me is they come in almost dead and then if you find the right regimen, it is complete magic. After a few months, you see a patient who was paralyzed because of bone involvement walk out of the hospital. So these anti-TB drugs are magic." After working on TB in Ethiopia, Dr. Girum got a job thousands of miles away at Lakka. He uprooted his life because he knew about the severity of the TB epidemic in West Africa and felt he could make a difference. He could tell many success stories. "But sometimes," he told me, "it's also discouraging. What's discouraging is you see so many patients come late in

the process. So you still fight to give the care that is needed, but often you see patients with basically no lungs."

This is a huge challenge for tuberculosis treatment in poor communities. In the U.S. in the 1950s, vans containing mobile X-ray machines fanned out across the country offering free chest X-rays to identify tuberculosis earlier and get people on treatment.

This "active case-finding" is key to reducing tuberculosis for three reasons:

1. Early detection stops chains of infection, thereby reducing the future burden of the disease.

2. Identifying patients before they become extremely sick allows for better overall outcomes.

3. The close contacts of infected people can be offered preventive therapy.

If you live with someone who has active TB, you're especially vulnerable to becoming infected yourself. But by taking one of the RIPE drugs, isoniazid, any latent infection can be cleared from the system. Unfortunately, preventive therapy still requires taking pills daily for several months, which can be quite a burden for those who aren't sick, but it's effective and generally well-tolerated. And better, shorter-course preventive therapy solutions have been proven effective but are yet to be rolled out globally.

Preventive therapy was pioneered in Bethel, Alaska, where TB rates in the early 1950s were extraordinarily high (higher, in fact, than they are in Sierra Leone today). But implementing this comprehensive approach to fighting tuberculosis brought down rates of TB in Bethel by 69 percent *in a single year*.

So this strategy can be extraordinarily effective, but in Sierra Leone, there was very limited active case-finding or preventive therapy in the early

2020s, because there was so little money available to fight TB. Instead of X-ray machines fanning out across the country, many patients are still diagnosed via smear microscopy, which misses 50 percent of cases, especially early on in the disease. Many patients are misdiagnosed with malaria or typhoid. Many wait until they are very sick to seek medical attention, because they know it will be expensive and stigmatizing. This combination of factors means that by the time a patient makes their way to Lakka, they are often too sick to survive.

Dr. Girum told me about another great frustration: knowing what needs to be done and being unable to do it. “When you know the drugs they need, but you do not have the drugs available, it is very difficult. In western countries, they can stop a hemorrhage, but we do not have the tools.” And so many times, Dr. Girum has watched patients die from a lung hemorrhage that he knew how to treat, or for want of drugs that he could safely dispense.

Dr. Girum is thin and understated. He embraces complexity—whenever I start ranting, he will always remind me of nuances. It is difficult to imagine him raising his voice. When he first arrived at Lakka, he told me, “I met this boy. Like you, I did not realize he was a patient because he was always helping the other patients. I thought maybe this was his job—being a social supporter. But no! He is a patient! And then I see his record, and I realize. That patient was Henry Reider, of course.”

By then, it was very clear that the injectables regimen was failing, just as RIPE had. The treatment had caused Henry much suffering and left him without hearing in one ear—and it would not cure him. Henry would, Dr. Girum knew, slowly and inexorably decline. When the last line of available drugs fails, he told me, you know the ending of the story. “That’s the point where you put down your stethoscope.”

It’s worth lingering for a moment on the value of early and accurate diagnosis because Henry’s life might have been so different. The rapid molecular test that was not available to Henry, known as GeneXpert, is a wonder of technology manufactured by the Danaher subsidiary Cepheid. Using similar tech to PCR tests for Covid or HIV, one GeneXpert cartridge

can identify not only whether someone has TB, but whether their particular infection will be resistant to certain antibiotics. A second cartridge can test for sensitivity to a broader range of antibiotics, so with these two tests, physicians can know within a few hours of encountering a patient whether the RIPE regimen will cure their tuberculosis.

This would not have been some panacea for Henry, of course, because he still would have had a very difficult time accessing the second- and third-line antibiotics he needed. But his family would have at least *known* what he needed.

Access to molecular testing has been severely limited by cost. As Dr. Muhammad Shoaib of Doctors Without Borders put it, “Not enough people have access to GeneXpert testing and the high price is a major factor.” One lab tech in Sierra Leone told me, “The machines are great. If only we could afford the test cartridges.”

Cepheid uses a business model similar to that of printer ink or razor blades to generate huge profits: They sell the GeneXpert testing machines at a small profit margin and then charge high margins on the test cartridges themselves, just as razor handles are often cheap, but razor blade refills are notoriously expensive. In fact, Danaher CEO Rainer Blair once noted that GeneXpert provided “a razor-blade business model in a mission-critical application,” as if bragging that the company’s profit is built around price gouging the world’s poorest countries and those who serve them.[*1]

A study commissioned by Doctors Without Borders indicated that the cost to manufacture these cartridges was less than $5, but until 2023, the price of a single GeneXpert cartridge was $9.98; the one to test for extensive drug resistance was $14.90.[*2]

Putting aside the costs of the machine itself, maintenance, and lab techs to run the machines, just testing one person with these two cartridges costs $24.88, which is *more than half* of what Sierra Leone spends on healthcare per person per year. And so of course TB was tested in cheaper ways—through microscopy or chest X-ray, neither of which can determine whether a patient’s TB is sensitive to first-line antibiotics.

Combining smear tests and RIPE treatment is, narrowly speaking, the cheapest way to address tuberculosis. Over 90 percent of people with TB will respond well to the RIPE drugs, so it makes a kind of sense to diagnose TB as inexpensively as possible and put everyone on the RIPE drugs. If a person is unlucky enough to have drug-resistant TB, it can be identified through costly molecular tests or culturing after the RIPE protocol fails. But this scarcity mindset was never applied in the U.S., where GeneXpert tests for tuberculosis are routine and where, seventy years ago, we put X-ray machines in buses because even then we recognized that smear microscopy wasn't adequately sensitive. As a result, today we have very little tuberculosis in the United States, and spend very little preventing or treating TB. Analyses of cost-effectiveness often only run skin deep. When looking at the larger costs—the cost of the ineffective pills, the cost of potentially further spreading drug-resistant TB, the cost of hospitalizing a kid who should've been in school, and all the other costs of *not* getting kids access to proper testing—GeneXpert tests should be in every clinic in every country with a high burden of TB. But the obsession with cost-effectiveness often ends at, "Can we get this disease diagnosed more cheaply?" rather than a broader consideration of the human costs.

When we do consider the long-term costs of failing to use all the tools at our disposal, the value calculus changes. From that perspective, investing in tuberculosis diagnosis and treatment begins to look like one of the best bets in global health. A 2024 study commissioned by the WHO found that every dollar spent on tuberculosis care generates around thirty-nine dollars in benefit by reducing the number (and expense) of future TB cases, and through more people being able to work rather than being chronically ill or caring for their chronically ill loved ones. A 2023 paper in the *Journal of Benefit-Cost Analysis* (there's a journal for everything!) calculated an even higher return, finding that every dollar "invested in TB yields US $46 in benefits." The report also found that between 2023 and 2050, there could be "almost one million averted deaths per year on average…. Interventions to address TB represent exceptional value-for-money."

But of course people are not just their economic productivity. We do not exist primarily to be plugged into cost-benefit analyses. We are here to love and be loved, to understand and be understood. TB intervention is an exceptionally good global health investment, but that is not why I care about TB.

I care about TB because of Henry.

Skip Notes

*1 Danaher lowered the cost of their standard TB test cartridge in 2023 and pledged to produce TB tests at cost to low- and middle-income countries, with cost evidence provided by an independent auditor, but as of 2024 the company has not released any audit to back up this promise.

*2 This may not seem like a lot of money, but the Global Fund, one of the major funders of tuberculosis testing and treatment support, has said that when Danaher lowered prices by just two dollars in late 2023, they could afford to purchase millions more tests annually.

CHAPTER 16

HENRY

ISATU BROUGHT EXTRA FOOD TO Henry whenever possible as he endured the rigors of treatment at Lakka. When he lost so much blood via his hemorrhaging lungs that he needed a blood transfusion, Isatu walked from friend to neighbor to relative raising the money needed for the transfusion and seeking a volunteer. She held Henry's hand while he received the transfusion, and held him as he seized on the table, drenched in sweat, his body overwhelmed by the blood it desperately needed.

But as Dr. Girum Tefera already knew, the injectable regimen just wouldn't work well enough to cure Henry. His disease was already worsening. Henry felt he could monitor the efficacy of his own treatment by observing the hugely swollen lymph nodes in his neck and shoulder. When they grew, at times bursting through the skin and leaving open wounds, he knew he was getting worse. When they stabilized or shrank for a time, he felt he was getting better. Dr. Girum told me that Henry was uniquely good at talking himself into optimism, into encouragement. "His way of thinking is amazing," Dr. Girum told me. "He would always tell you, 'I want to go back to school.' He said this even in the middle of a crisis. Failing that regimen, knowing how difficult it was, he was always planning to survive and to go back to school."

But he *was* failing the injectables regimen, and Dr. Girum knew that to give Henry any chance of a cure, he needed to find a different solution. "We were short of other options, so we started talking to colleagues in other countries." He consulted with physicians at Harvard Medical School, and at

other sites around sub-Saharan Africa and Asia. Part of the challenge was Henry's comorbidities—he had other health problems that made him ineligible for some TB drugs, meaning an experimental cocktail would need to be tailored to Henry specifically.

"At that time," Dr. Girum explained, "Sierra Leone's national TB program was trying to introduce new drugs, including delamanid and bedaquiline." But those drugs were not just hugely expensive—costing over $1,000 for an eighteen-month course of treatment. They also weren't yet available in Sierra Leone at all. And they came with their own side effects, which might be a problem for Henry's liver or kidneys because of his preexisting health challenges. It was all a huge mess, one that demanded both a clinician's skill at constructing a cure and an operational genius for finding a way to get the right drugs to Lakka. But Dr. Girum couldn't bear the thought of giving up on this boy, or, for that matter, any of his patients. "In the field of tuberculosis," he told me, "you have to keep trying."

Even though Henry's family struggled to have any income and adequate food, Isatu visited him nearly every day, and his father visited often as well. When Henry's mom brought him food, Henry would share with Thompson, a man in his thirties who also lived on the MDR-TB ward. While Henry made fast friends with the staff—"he was like a son to everyone," as Dr. Girum put it—he was isolated from most other patients because of the risk that he might spread his particularly serious strain of drug-resistant TB. But he was able to spend a fair amount of time with Thompson, and they became close friends. "He always encouraged me," Henry said. "Even when we were both lying in bed with fever and sickness, I shared my food with him, and he shared his encouragement. 'You can do this. You are going to have a good life, Henry.' "

Henry needed that encouragement. While he tried to act happy in public and cheer up everyone he encountered, he was struggling not just with the physical burden of his TB getting worse, but also emotional devastation.

“The hospital room became my entire universe,” he wrote. “A sense of isolation and despair settled in.”

Although his family stayed unusually close, many of his friends disappeared. “I had thousands of friends at school. We spent all our time together. We played together all the time.” He remembered once he hurt his knee and his friends started crying for him in empathy, because they loved him so much. At Lakka, he lay on his side, knowing he couldn’t get enough air even to walk across the room, thinking of those times. “Some of them, when they heard about me having TB, they never talked to me again.”

When Henry learned his treatment of injectables had failed, he became despondent. “Our happy boy,” as one of the nurses called him, turned sullen and angry. “The light that had once shone brightly in my eyes was now dimmed,” he wrote. “As the months went on, the isolation grew more profound.” There was no more rapping and dancing in the hallways, wearing his sunglasses upside down to make the other patients laugh. He knew now. He wasn’t just falling behind his peers; he was saying goodbye to the world at the age of eighteen.

In the small room where Dr. Girum looked through case notes, there was little reason to be encouraged. Lakka at the time didn’t have reliable running water, and the electricity was out for hours each day. They didn’t have enough food to feed the patients properly, let alone the capacity to tailor novel drug combinations to individual patients with complexly drug-resistant tuberculosis.

By the spring of 2020, Henry and his friend Thompson were both in decline. To Henry, Thompson was not only a friend and mentor but also, as Henry once called him, “a ghost of my future.” And then one morning, Thompson was gone. He died starved for air, an experience one person

described to me as “breathing through a straw, or into a pillow, all the time.”

“My friend lost his life. And after he died, something told me: ‘You are next, Henry. You are next.’ ” Henry felt certain that his death was imminent. He found himself crying more, and leaving his room less. When Isatu visited with food, he wouldn’t eat it. He had no appetite—although the doctors couldn’t say whether it was due to depression or tuberculosis. To Henry, they were inseparable. Just as he probably never would have developed TB if he hadn’t been malnourished because his family couldn’t afford food, he probably never would have developed depression if he hadn’t seen his best friend die, and then known for certain that he was next.

At night, he thought of his mother. He would miss her so much, but even more, she would miss him so much. His mother loved him so deeply, so consistently. She’d already lost a daughter. He prayed to God for deliverance, for healing—not for himself, although Henry desperately wanted to live, but for Isatu, so she would not be alone in the world.

With his father, it was a more complicated relationship. They’d been only intermittently close. Henry’s dad was prone to rage, and Henry often felt that his dad had abandoned their family—providing nothing in the way of financial support while living apart from them. He *had* visited Henry frequently at the hospital, and clearly cared for his son, but Henry’s dad still felt strongly that Henry shouldn’t be in the hospital at all, and that his tuberculosis could be cured by prayer.

Henry was inclined to trust Dr. Girum, but especially after Thompson’s death, he began to despair. “I was so scared,” he said. “Thompson and I, we would encourage each other. He was my only great friend. So to see him die, it gave me different thoughts. It gave me thoughts about death.”

And these thoughts could not easily be quelled by Dr. Girum or Henry’s other caregivers. They could use phrases like “We’ll take the best care of you we can,” or “We’ll be with you whatever comes,” and they could even say, “There’s still hope.” But they couldn’t say, *You will get better*. They couldn’t say, *You will be cured*.

CHAPTER 17

"BEAT ME LATER"

STILL, DR. GIRUM KEPT WORKING on finding a new drug regimen that might help Henry. He consulted with the government's TB program: What if we *could* get the right drugs into the country? Perhaps Henry could be the first to receive them, and we could use his case to prove that in Sierra Leone, it is fair and appropriate for patients to receive the kind of personalized, tailored treatments that people in the rich world can expect.

As Dr. Girum worked to make the case that Henry needed and deserved tailored treatment to survive, Henry's father felt increasingly despondent and angry with a system that kept his son locked away in a hospital. And when Henry's father learned the injectables treatment was failing, he was furious. His son had been cooped up in this hospital, which had been known for decades as a place one goes to die, for two hundred days, and now the medicine promised to be a cure had in fact done nothing—or maybe worse than nothing, since his son was now sicker than ever.

Now Henry's dad was sitting across a desk from Dr. Girum, whose patient demeanor did nothing to calm Henry's father. Henry's dad stood up in Dr. Girum's stuffy consultation room, shouting at this foreign doctor that his son needed to return to school, to home, to life. The three previous treatments had failed. As far as Henry's dad could tell, this medicine had failed Henry just like the formal healthcare system always failed the sick. After all, the boy was in bed all day now, unable to get up for anything but the bathroom.

Dr. Girum was afraid—he was new to the country and didn't yet speak fluent Krio. He didn't even fully understand *what* Henry's father was yelling, only *that* he was yelling. The doctor tried to explain that Henry *couldn't* go back to school—he wasn't well enough, and anyway he would be exposing other students to an extremely dangerous strain of drug-resistant tuberculosis. And he couldn't go home, either—he would only die there.

Then let him die at home, his father responded. The man was heartbroken and enraged. Henry's father announced he would come back tomorrow to take his son so that Henry could spend his final days at home surrounded by those who loved him, instead of at this hospital that only delivered false hopes. And if he wasn't allowed to take his son, Henry's father promised to beat Dr. Girum.

Henry, meanwhile, languished in his room. He knew of his father's wish to bring him home, and of Dr. Girum's promise that there might still be some hope if he stayed at Lakka. He didn't know whom or what to believe. He felt hopeless and terrified. He didn't trust his father, but he wasn't sure if he should trust the doctors and nurses, either. After all, they hadn't saved Thompson.

Henry wanted to be someone—to contribute to his country, to travel the world and see how others lived. He remembered the businessmen on the streets of Freetown, how they had new shoes. He knew it was a silly thing to think about, especially if he was dying, but he'd never had a pair of new shoes. His feet had always been in shoes already molded to someone else's gait.

When Henry listened to music, he imagined the people around the world who made those songs. What must their lives be like, in London or Los Angeles or Lagos? He would never know now. He tried to calm himself down, to reassure himself, but then he would cough up more blood. He could feel the ruptured sores on his neck and shoulder getting larger, his

skin no longer able to contain the bacteria's growth. His misery was compounded by loneliness. He was too sick to leave his room often, but also he wasn't allowed to. "The medical team," he wrote, "recognizing the gravity of my condition, made the difficult decision to limit my interactions with other patients. I was confined to my own space, cut off from the camaraderie and shared experiences that had once been a source of comfort."

Maybe he should go home. But he wanted so desperately to live, and he had no more faith in traditional treatment like exorcism than in biomedical treatment. It had all failed him. Maybe God needed him. But he was scared to go to God. Maybe he had not been good. He thought of the way people treated him when they knew he had TB—how they scowled, how they stood across the room from him even when he was masked. How they pitied him or reviled him.

That night, Dr. Girum struggled to sleep. He was two thousand miles away from his wife and baby, working so far from home because it was the best available opportunity, but also because he knew how desperately Sierra Leone needed tuberculosis care. In his home region of Ethiopia, TB was also a huge problem, and chronically underfunded—but in Sierra Leone the situation was much more desperate. So many people couldn't even access diagnostic tests, and when they finally made it to Lakka, their lungs were devastated. Dr. Girum knew that even if he could get better treatment to Henry, the boy still had a high chance of dying. His lungs looked terrible in his most recent X-rays.

Also, he feared Henry's father and his threats. "I tried to remember," Dr. Girum would later tell me, "that this man, he just wants his son at home. He does not want his son to die alone. I understand this. I am also a father." At last, Dr. Girum came up with a plan, and only then could he fall asleep.

The next morning, Henry's father returned to the hospital. He was angry that the nurses would not allow him to see his son. He walked around the

doctor’s desk, ready to punch Dr. Girum, who calmly told Henry’s father, “If you take your boy away now, all the work we have done is meaningless. I know you are a dad. I am also a dad. But I am his doctor, and I can promise you that if this boy is not doing well on a new drug regimen, come and beat me. Don’t hit me today. Beat me later if this fails.”

Henry’s father stomped out of the room to get to his son, but the nurses turned him away, and ultimately, so did Henry. He was eighteen now. Henry told his father he would stay at Lakka, and wait for the new drugs.

The next morning, Isatu came to the hospital and apologized. “My husband already gave up,” she explained. “He wants to stay with his son. But I am not giving up on you, Dr. Girum. I am giving you my faith, my life, my son.”

Dr. Girum later told me, “Yes, I know, it’s just one patient. There are so many patients, and Henry is just one. Why should we move mountains to save one patient? Because he is one person. A person, you understand? And anyway, what if he can be the first of many?”

CHAPTER 18

SUPERBUG

TO THE EXTENT THAT ONE hears about TB at all in the rich world, it's usually in the context of a looming crisis: Given enough time, a strain of tuberculosis may evolve that is resistant to *all* available antibiotics, a superbug that is perhaps even more aggressive and deadly than previous iterations of the disease. The fearmongering around superbugs can serve a purpose—it is one strategy for getting people in wealthy communities to care about TB. It may not be at your doorstep yet, but by the time it is, it'll be too late.

And to be clear: That's very possible. *M. tuberculosis* has shown an ability to select for variants that evade antibiotics, and because we haven't done a good job creating a wide array of TB treatments, a highly infectious and totally resistant strain could emerge. It probably wouldn't sweep through Earth in a matter of weeks or months—we must remember that TB divides (and sickens) quite slowly relative to most other pathogens, and that it primarily sickens those with immune systems compromised by malnutrition, concomitant illness, or poor living conditions. But TB could once again become the fully global crisis it was until seventy years ago.[*1]

But I'm suspicious of focusing on the superbug argument for two reasons: First, we should not need to personally fear TB in order to understand and respond to the crisis. For billions of people, the superbug era is here: Tuberculosis is a powerful bacterial infection that billions of people lack any effective tools to fight, not because those tools don't exist, but because we've done such a poor job of getting the cure to the disease.

But also, as we saw with Covid, when an incurable illness spreads widely among the rich and powerful, we dramatically increase our investments in tools to treat and prevent that disease. Within eighteen months of Covid's emergence, we had excellent vaccines and effective antiviral medication to combat the pandemic. Covid remains a serious public health threat in 2025, and a major driver of death and disability, but the situation is different from 2020 because of the research money poured into responding to the disease. If TB became a problem in the rich world, attention and resources would rain down upon the illness until it ceased to be a problem for the rich, powerful, and able-bodied.

And that brings us back to our old frenemy, cost-effectiveness. To understand how cost-effectiveness distorts the TB treatment landscape, let's look at the rifamycin class of antibiotics, the R in the RIPE protocol. The first rifamycin drug was synthesized in an Italian lab by Maria Teresa Timbal, Pinhas Margalith, and Piero Sensi.[*2] As one 1969 analysis put it, "The major attraction of rifampicin [a variant of rifampin] is its very low toxicity and ease of administration." This seems like a very major attraction, indeed! But there was a downside: "The major disadvantage is its cost. It is a highly expensive drug and seems likely to remain so. In many countries the cost will be prohibitive. It will be far more important to put the available resources to better use by improving primary treatment with the cheaper regimens."

There's a lot to unpack in those three sentences. Let's begin with "it is a highly expensive drug." Why is it a highly expensive drug? Well, in the 1960s, it was challenging to produce in large quantities. But often, our cost analyses assume, incorrectly, that "a highly expensive drug…seems likely to remain so." Today, rifampin costs less than half of what it did forty years ago, and is absolutely essential to our ability to cure most cases of tuberculosis.

At the time of its introduction, rifampin was tested in a 600-mg once daily dose—not because that was believed to be the most effective dose, but because it was believed to be the cheapest dose that was still effective. And even today, we *still* prescribe 600 mg of rifampin, even though the drug costs less to produce and it now seems likely that higher doses would be more effective. “We’ve been underdosing with this drug for fifty years,” Dr. Carole Mitnick told me, because we still base our cost analysis on how expensive the drug was to synthesize in 1969. This increases the likelihood of patients developing resistance to rifampin and other drugs in its class, because the bacteria has longer to evolve immunity to a needlessly low dose, and it also means that people are sick (and contagious) longer than they need to be.

All of this happens because of what Dr. Mitnick describes as “a failure of imagination.” “There is this continued mentality of scarcity in TB,” she explained. I think of this in the context of my brother Hank and his cancer care. Cancer care even within the U.S. remains wildly inequitable and littered with all manner of price gouging, but no one questioned whether treating my brother’s lymphoma was “cost-effective,” even though it cost a hundred times more than it would’ve to cure Henry’s tuberculosis. My brother is my oldest friend, my closest collaborator, and his work has been transformative in many lives. I would never accept a world where Hank might be told, “I’m sorry, but while your cancer has a 92 percent cure rate when treated properly, there just aren’t adequate resources in the world to make that treatment available to you.” That world would be so obviously and unacceptably unjust. So how can I live in a world where Henry and his family are told that? How can I accept a world where over a million people will die this year for want of a cure that has existed for nearly a century?

Skip Notes

*1 Indeed, TB is already rising—albeit slowly—in the United States, which in 2023 saw nearly ten thousand cases of active TB infection.

*2 The lab named its drugs after pop culture phenomena, and they named their antibiotic after a French gangster movie, *Du Rififi Chez les Hommes*, directed by Jules Dassin, an

American who’d been blacklisted from Hollywood due to Communist Party membership.

CHAPTER 19

VICIOUS CYCLES

BEGINNING IN THE EARLY 1980S, physicians and activists in the Global South began sounding the alarm about an explosion in uncommonly swift and severe cases of tuberculosis. Young patients were dying within weeks instead of over years, often with TB disseminating throughout their lungs with terrifying speed, choking patients to death. TB had always been a creeping killer, a slow strangling. Now patients were dying hours after entering the hospital.

These deaths seemed to be associated with the emerging pandemic now known as HIV/AIDS. In 1985, physicians noted high rates of active tuberculosis disease among HIV-positive patients in Zaire and Zambia. Because untreated HIV lowers resistance to infection, TB infections are far more likely to progress to active disease as the immune system weakens, and that weakened immune system allows TB to kill quickly. As early as 1986, Gnana Sunderam et al. wrote, “It is possible that AIDS reflects and magnifies diseases that are endemic.” And Annik Rouillon noted, “By 1986-87 we had begun to note that some patients, young patients, were dying.”

Even though many were pointing out this connection, far too little was done to expand access either to TB or HIV medication in low- and middle-income countries. By the mid-1990s, antiretroviral cocktails made HIV a treatable and survivable disease in rich communities. While taking these medications, viral levels generally become so low as to be undetectable, making it impossible to transmit the virus.

But people in poor countries still died by the millions every year. TB treatment, too, often remained out of reach, especially because multidrug-resistant tuberculosis was common among those coinfected with HIV. And so these intertwining epidemics led to an explosion of death—so much so that overall life expectancy dramatically declined in many poor countries. In Lesotho, for example, overall human life expectancy dropped by about ten years between 1985 and 2002.

In denying HIV treatment to the poor, the reasons cited—patients couldn't be trusted to take their medication on time, better to focus on prevention and control—were the same as we've seen with TB. In 2001, the head of USAID—the U.S. government's arm devoted to international aid—had this to say about making antiretroviral treatment accessible to the poor: "If we had [HIV medicines for Africa] today, we could not distribute them. We could not administer the program because we do not have the doctors, we do not have the roads…[Africans] do not know what watches and clocks are. They do not use western means for telling time. They use the sun. These drugs have to be administered during a certain sequence of time during the day and when you say take it at 10:00, people will say, 'What do you mean by 10:00?' "

We see here that the racist dehumanization of African people is not only part of nineteenth and twentieth century history. Racism continues to distort our policies and practices. And just as with previous examples of racism, it proved to be totally false. In point of fact, a 2007 study found that Africans were *more likely* to adhere to HIV/AIDS treatment regimens than North Americans.

It wasn't until the mid-2000s that, through programs like PEPFAR and the Global Fund, millions of people living with HIV in poor countries were at last able to access antiretroviral therapy. Again, cost-effectiveness proved a moving target. It was impossibly expensive to treat HIV in poor communities…until drug companies were pressured to lower prices by 95 percent, at which point it suddenly became affordable.

As for roads and clinics, it transpired that strengthening healthcare systems and transportation systems as part of a comprehensive plan to

deliver better HIV care was not just possible, but an extraordinarily good investment. Millions of lives have been saved—and tuberculosis deaths among those living with HIV have declined dramatically in the decades since. But so many were lost between the mid-1980s, when activists first began shouting that the commingling of HIV and TB would lead to catastrophe, and the mid-2000s, when HIV treatment finally became widely (but not universally) available. Tens of millions of people died of tuberculosis in those years. In fact, between 1985 and 2005, roughly as many people died of tuberculosis as in World Wars I and II combined.

Tuberculosis is so often, and in so many ways, a disease of vicious cycles: It's an illness of poverty that worsens poverty. It's an illness that worsens other illnesses—from HIV to diabetes. It's an illness of weak healthcare systems that weakens healthcare systems. It's an illness of malnutrition that worsens malnutrition. And it's an illness of the stigmatized that worsens stigmatization. In the face of all this, it's easy to despair. TB doesn't just flow through the meandering river of injustice; TB broadens and deepens that river.

CHAPTER 20

HAIL MARY

FOR A TIME, HENRY RECEIVED no treatment at all, which was hugely discouraging. At least when he suffered through the painful and toxic injections, he was *doing* something about his tuberculosis. Now he lay on a bed at Lakka, the hot, still, damp air weighing down on his chest. His breathing grew shallower. When people are scared, they are often told to take slow, deep breaths. But Henry couldn't take a deep breath.

PIH found the money needed to treat Henry. Henry would be the first Sierra Leonean to receive this kind of highly personalized, tailored cocktail, and Dr. Girum knew how much that meant—not only to Henry but to the country as a whole. If Henry survived, he would be living evidence that even complex drug-resistant TB can be cured in Sierra Leone. If he didn't, his story might be used by global health authorities as further evidence that it simply doesn't make sense to treat people like Henry in places like Lakka. Some of the drugs Dr. Girum sought were available in neighboring Liberia. Others would have to be flown in from further afield—Lesotho, perhaps, where Partners In Health had established a robust center for treating tuberculosis and HIV.

For weeks he'd been asked to wait, to do nothing, to lie silently and alone in a single room because he was too sick to have a roommate. He thought of Thompson's death. He felt himself going next. He thought of his childhood friends growing up, being normal. And he wrote poems. His poetry was

strange, impressionistic, arresting, and lovely. One, called “Golden Axe,” begins:

Golden Axe a mysterious battle
An Axe which need to be found
Warriors without determination
Gentleness dashes their toes.
They pump held their booze throw in obedient
Moving in the place of Gold
Lion tortured their back
Warriors went down the hole, without finding where to hold

He prayed—Henry was a committed Christian. He thought of Isatu, thought of trying to live for her. But survival is not primarily an act of individual will, of course. It’s an act of collective will. Henry had only contracted TB because of choices humans made together to deny treatment to people in poor countries. A child born in Sierra Leone is over one hundred times as likely to die of tuberculosis than a child born in the United States. This difference, as Dr. Joia Mukherjee writes, is “not caused by genetics, biology, or culture. Health inequities are caused by poverty, racism, lack of medical care, and other social forces.”

Henry was sick not really because of Koch’s bacillus, but because of historical forces, the ones we’ve encountered here. Henry embodied spes phthisica—he was sensitive and poetic. And yet he wasn’t treated as a luminous, beautiful poet condemned to die by the same wonderful forces that gave him his creative powers. His illness was a product of Sierra Leone’s centuries-long impoverishment, of a healthcare system hollowed out by colonization and war and Ebola, of a world that stopped caring about TB when it ceased to be a threat to the rich.

We live in between what we choose and what is chosen for us. Henry was acted upon by historical forces, but he was also a historical force unto himself—as we all are. He made choices. And as one of those choices, he

chose to stay at Lakka, to believe in Dr. Girum, to trust a medical system that had done so little to earn his trust.

On the day Henry started the new treatment, he was, as he put it, “in a state of confusion.” The medicine had arrived—some of it by car from Liberia, some in the suitcases of folks visiting Sierra Leone. Dr. Girum began Henry’s course of treatment immediately. By this time, Henry had spent over three years of his life in either Connaught Hospital or Lakka. He’d watched his best friend die. He did not know whether the new regimen would work. Nor did Dr. Girum. Even if it did, Henry’s lung damage was serious. Many patients who get treatment too late die, like Shreya Tripathi did, even if they respond well to the antibiotics. Henry’s lungs were heavily scarred from years of living with the disease, years of it crawling further into his lungs, years of having the antibiotics work well enough to ease his breathing and shrink his scrofula—but only temporarily.

The question was not whether he would feel better. The question was whether he would get better. And so when he started to feel better, he still could not relax.

He kept taking the medicine, praying the warriors down the hole would find where to hold.

CHAPTER 21

LIKE MAGIC

WITHIN A WEEK OF THE new medication regimen, Dr. Girum noticed progress, especially with the open sores from Henry's ruptured lymph nodes. "It was like magic," he told me. For over a month, Henry had lived with open wounds in his neck and shoulder from lymph nodes so swollen they burst through the skin. But now, the nodes were retreating, and the open sores began to heal. "I could see the wound drying," Dr. Girum said. "I told myself, 'This is an early sign.' In a week, the boy started to eat well."

Henry feared another false dawn. For the first two weeks of taking the medication, he felt certain it was failing, and he was overwhelmed with loneliness, especially because he couldn't see his mother. "I had to be in one room, in isolation. So I could not see anyone. I was discouraged thinking the sickness is so bad that they don't want me to be with anyone else." But after three weeks, he was starting to stand up and walk around. He too noticed the lesions in his neck and shoulder improving. He felt stronger, and he was hungry again. Within a month, he gained ten pounds.

After a few months of effective treatment, no bacteria was detectable in Henry's sputum for the first time in years. Although the infection still lived in his lungs and lymph, he was far less infectious, and so he was able to receive visitors again. Isatu returned to her daily visits. Henry's father came to Lakka a month into Henry's treatment. "He was a little ashamed" of his threats, Dr. Girum recalled. But Dr. Girum reassured Henry's dad. "I know the social impact of a child sleeping in the hospital every night for over a

year. I understand this anger, this lack of trust in medicine. I understand. Maybe if it was my child, I would do the same thing."

As Henry began to feel better, he wanted to go home to his mom in Freetown. His dad was living elsewhere by then, and Henry felt disenchanted with his father's outbursts and inconsistency. But he desperately missed being home with his mom, "the one who stands closer" when others ran away. He wanted to go back to school, too. He was eighteen now, and had been unable to attend school since before his freshman year of high school. He wasn't sure that he *could* go back to school—he knew how far behind he would be. He wasn't in close touch with any of his former classmates, but he knew they were in university now, or else working. Would any school accept an eighteen-year-old high school freshman?

After over a year at Lakka, and three total years of hospitalization, Henry got to go home. He wasn't finished with treatment—that would be twelve more grueling months of a dozen pills a day, taken each morning, often with little food to help the antibiotics settle in his stomach. But he was home, and more importantly, he was with Isatu, the mother who stayed. "It was the happiest moment of my life," Isatu told me in 2023.

There's no playground at Lakka Government Hospital, or really much of anything to do besides rest on your cot or walk around the dusty grounds or sit on the long wooden benches in the shade of the immense mango tree. At Lakka, you can hear the world—the hospital backs up to a busy road and a line of market stalls—but you are not of that world. Henry desperately wanted to return to that world, but he was also frightened of it.

Isatu and Henry were profoundly impoverished by the experience of tuberculosis. Isatu lost her business selling goods at the local market during the ordeal, and the two-room cinder-block home where they had lived. Now they stayed in a smaller apartment with a rusty metal roof that leaked when it rained—which it does over one hundred days a year in Freetown. No

matter where Isatu and Henry put their mattresses, water found its way to them, and they often shivered wet through the long nights of the rainy season. The apartment was dark—it was not connected to the electrical grid—and in a crowded area of Freetown that many called a slum. Some days, Henry and Isatu struggled to eat enough. Life, as Henry told me, had many difficulties. “There are so many constraints,” he said.

Henry had grown into a strong young man, with a defined jawline and a muscular build. It was impossible to imagine that I had once thought him the same age as my now thirteen-year-old son. But Isatu could still quiet him with the slightest touch. “Constraints,” she agreed, then added: “But I see you alive again. I look at you, and you are alive. My son Henry is alive.”

CHAPTER 22

VIRTUOUS CYCLES

MERE DESPAIR NEVER TELLS THE whole human story, as much as despair would like to insist otherwise. Hopelessness has the insidious talent of explaining everything: the reason X or Y sucks is that everything sucks, the reason you're miserable is because misery is the correct response to the world as we find it, and so on. I am prone to despair, and so I know its powerful voice; it just doesn't happen to be true. Here's the truth as I see it: Vicious cycles are common. Injustice and unfairness permeate every aspect of human life. But virtuous cycles are also possible. In fact, it is because of one that Henry is alive, and that others will live because of Henry.

One virtuous cycle began in the early 1990s, when Peru became one of the first countries in South America to implement a comprehensive DOTS program aligned with WHO guidelines. As Tracy Kidder writes in *Mountains Beyond Mountains*, Peru's TB program came into being "largely because of protests staged by residents…by their nuns and priests." The government agreed to fund DOTS. But WHO guidelines at the time did not call for any treatment for those living with drug-resistant TB. If patients were failed by the first line of RIPE antibiotics, they were administered those same antibiotics again. When those drugs inevitably failed, the standard of care was so-called "supportive therapy." I didn't understand this term, and so I asked Dr. Carole Mitnick about it in one of our first meetings. "It basically meant, 'Put sick people in a hut on the side of the road and wait for them to die,' " she told me.

In low- and middle-income countries, no second- or third-line antibiotics were available through the public health system—and so people with drug-resistant TB were essentially left to die (and not only left to die, of course—they were also left to further spread drug-resistant TB before dying). The WHO maintained "MDR-TB is too expensive to treat in poor countries."

At the time, treating just one patient with a two-year course of drugs cost between $15,000 and $20,000. From a cost-effectiveness perspective, treating the sick seemed to make no sense—not only because it was so much money to save one person's life, but also because of what the WHO called "the limited prospect for cure of those cases." Curing MDR-TB was challenging even in the best hospitals. It was, they argued, basically impossible in middle-income countries like Peru. So why bother?

And yet, this approach was failing in multiple ways. As the legendary Dr. Paul Farmer pointed out, everyone emphasized the cost of treating drug-resistant tuberculosis, but "failure to diagnose and treat MDR-TB is what is really costly." Every uncured case of MDR-TB was an opportunity for the disease to spread further and to develop further resistance, an opportunity for the disease to cause yet more suffering. And failing to invest public money in MDR-TB treatment didn't keep people from seeking it on their own. Often, desperate families would mortgage their homes or sell their belongings to buy second-line antibiotics from private doctors, but then those families often couldn't afford enough of the drugs to achieve a cure, which led to more resistance and more suffering and more death.

In the late 1990s, Partners In Health (known as Socios en Salud in Peru) began to treat MDR-TB patients in an impoverished neighborhood of Lima where the disease had become endemic. PIH cofounder Dr. Jim Kim led the project but would be quick to point out that it was really led by a diverse group of partners including researchers like Dr. Carole Mitnick, epidemiologists like Dr. Meche Becerra, nurses, and community health workers. They individually tailored treatment regimens to patients and provided comprehensive support via direct financial payments so that patients could eat enough to fuel their recovery. The project also provided

regular visits from community health workers who lived in the affected communities and served as a bridge between the healthcare system and neighborhoods that had long been overlooked by that system.

The idea was simple. “We should treat people if we have the technology,” as Dr. Farmer put it. But it was not straightforward or cheap—PIH spent up to $20,000 per case, and they were keenly aware of the tradeoffs involved. As Dr. Jim Kim told a group of tuberculosis experts, “We actually had to make a choice that we would not feed four thousand more children in Haiti…and if any of you have been to Haiti, there’s hardly anything more morally compelling than the situation of landless peasants in the central plateau.” But Jim Kim and others felt certain that if they could prove that MDR-TB was in fact curable in poor countries, then the global health community and governments would begin to invest more in its treatment. They believed they could spur a virtuous cycle.

In April 1998, Partners In Health announced its extraordinary results: Over 85 percent of the Peruvian MDR-TB patients achieved cure with comprehensive support. One expert called it “astonishing.” These cure rates were comparable to, or even better than, those seen in the world’s best-funded hospitals. The global health community did indeed take notice—and within two years, the WHO had begun recommending a “DOTS-plus” strategy that incorporated some access to care for MDR-TB patients.

There was still the old enemy of cost-effectiveness, of course. But as Kidder points out, “Experts in TB control had declared MDR treatment inordinately costly, but no one had tried to reduce the main expense, which was high-priced drugs.” It was discovered that the patents on most of those drugs had expired, but no one had sought to create inexpensive generic versions of them, because there was “no market.” But of course there *was* a market—many people living with MDR-TB were desperate for treatment. The “market” just wasn’t generally wealthy. Soon, Partners In Health and others worked to kickstart generic manufacturing of these antibiotics.

Survivors and civil society groups pressured drugmakers as well, and the price of a two-year course of curative treatment quickly dropped from $15,000 to $1,500.

In part because of that price drop and the shifting guidelines around drug-resistant TB, more people began to survive and become advocates for further progress. At a TB conference not long ago, I met a young South African woman named Phumeza Tisile, who was diagnosed with TB in 2010 when she was a freshman in college. Phumeza was born in the Eastern Cape but moved to Cape Town as a young child so her mother could work as a domestic worker in the city. As a kid, Phumeza ran track—her specialty was the 800-meter race—and was such a good student that she received a full scholarship to university. But from the start of her freshman year, something felt off. She lost weight, and found herself frequently short of breath. Not only did she abandon running, she soon found it difficult even to walk up a flight of stairs. “So I went to the clinic and coughed into a cup,” she explained to me.

A lab tech looked at her sputum for the squirming rods of TB, but, as we’ve learned, microscopy misses about 50 percent of cases—and indeed, Phumeza was told she was negative for tuberculosis. Still, she kept getting sicker. Within a couple months, she had to drop out of school. Her weight plummeted. Two months after school began, she weighed less than seventy pounds. “I was really struggling to breathe and walk,” she told me. Finally, she received a chest X-ray, whereupon it was obvious that tuberculosis had choked both of her lungs. She started treatment immediately, but she did not respond to the antibiotics, and so was soon hospitalized.

Like Henry, Phumeza received months of treatment that didn’t work, followed by months of second-line antibiotics that also didn’t work. After being diagnosed with drug-resistant TB, she told me, “I was searching stuff online, and it was really, really scary to see because on Google so many of the people on image search were already dead. Their ribs were exposed and

I thought I was gonna be like that, too. I thought I was likely going to die." Like Henry, Phumeza lost hearing due to the injectables—but in her case the hearing loss was complete, and for five years she could hear nothing at all before finally receiving a $40,000 cochlear implant.

Ultimately, she was in treatment for tuberculosis for three years and eight months—during which time she took between twenty thousand and thirty thousand pills. The treatment cost her years of life and her hearing, but she was finally cured.

It is here that we start to see virtuous cycles in action. Today, Phumeza Tisile is a college graduate, a sociologist, and a leading voice in the fight against tuberculosis. Along with her friend and fellow TB survivor Nandita Venkatesan, Tisile filed a patent challenge in an Indian court asking the government to reject efforts by the pharmaceutical company Johnson & Johnson to extend their patent on the drug bedaquiline.

As we've seen, bedaquiline is a powerful medicine in the fight against MDR-TB, but since its first release in 2013, it was far out of reach for most people living with the disease, because J&J charged $900 for a single course of treatment in poor countries and $3,000 in middle-income countries like South Africa. And so most kids like Phumeza with drug-resistant tuberculosis could never receive appropriate treatment—not because it was unavailable, but because it wasn't "cost-effective."

J&J's patent was scheduled to expire in 2023, but the company attempted to file and enforce secondary patents in order to extend the life of its intellectual property. Patent "evergreening" is a common strategy among pharmaceutical companies to block generic competition in order to protect their prices and profits. In J&J's case, they claimed that while the patent on the bedaquiline drug compound itself was expiring, an adjuvant compound that increased the drug's effectiveness had been patented much later, and that patent should apply to the entirety of bedaquiline.

But Tisile, Venkatesan, and their lawyers successfully argued in Indian courts that J&J's secondary patents did not involve meaningful innovation and were merely an attempt at profiteering. As a result, the Indian government determined they would not honor J&J's secondary patents, and bedaquiline became available for generic production in mid-2023.

While this finding meant that bedaquiline would become less expensive in India, J&J *had* succeeded in filing secondary patents in most low- and middle-income countries, meaning that, for much of the world, affordable bedaquiline would remain out of reach. After negotiations with global health organizations and loud protests from anti-TB activists, J&J backed down, allowing generic bedaquiline in most countries, and then eventually abandoning all efforts to enforce their secondary patents on the drug. As a direct result, the price of bedaquiline dropped by over 60 percent almost overnight.

And so the work of TB caregivers, survivors, and activists in Peru in the 1990s helped Phumeza Tisile survive TB, and her work in turn lowered the price of bedaquiline, which will help many others survive TB. This virtuous cycle has dramatically expanded access to treatment by lowering its cost: MDR-TB was labeled as too expensive to treat in the 1990s, when it cost over $15,000 per patient. Organizations like PIH were able to drive that cost down to $1,500 by the late 1990s. Thanks to the efforts to lower the price of bedaquiline, that price has dropped further. In 2023, the endTB trials—funded by Unitaid, Doctors Without Borders, and PIH—found that around 90 percent of MDR-TB cases could be cured for about $300 per course, a 98 percent reduction in price from the 1990s. As Christian McMillen reminds us, when it comes to TB treatment, "cost-effectiveness is a moving target."

There is still a long way to go when it comes to making TB treatment plentiful, affordable, and universally accessible. But it is only because PIH and others proved that MDR-TB could be cured in poor countries, and only because MDR-TB survivors like Venkatesan and Tisile lived to fight patent evergreening, that we are seeing progress at all.

Still, many drugs that effectively treat multidrug-resistant strains of tuberculosis remain very expensive, and not because they are made of gold or platinum, or because we have to fly to the moon to find them. They are expensive because 1. Prices are kept artificially high by pharmaceutical companies, and 2. We are afraid that making these drugs less rare will lead to further antibiotic resistance. But as Dr. Carole Mitnick said to me once, “This is a human-manufactured problem that needs a human solution. If medications were a public good, the burden of disease would drive the priorities of the industry and TB treatment would be varied and plentiful.” And so we must fight not just for reform within the system but also for better systems that understand human health not primarily as a market, but primarily as a shared priority for our species.

We do not only see these virtuous cycles within the world of TB, of course. Surviving TB does not only mean the opportunity to support others with TB. It also means the opportunity to support one’s family, to pursue educational opportunities, to live.

Which brings us back to Henry. Although Henry worried he’d aged out of school, with support from Partners In Health he found a place at a secondary school where he excelled. He made friends easily and enjoyed the academic work. He wasn’t just able to catch up to his peers; he earned admittance to the University of Sierra Leone, one of the nation’s most prestigious institutes of higher learning, where he is now a second-year student studying Human Resources and Management. “Education is the most important thing,” he told me once. “Not just for me, you know, but also for the nation.”

Henry continued to live in difficult circumstances in Freetown until more people began to hear his story. A GoFundMe set up to help rebuild Isatu’s business quickly surpassed its initial goal of $11,000 and raised over $60,000 to support Henry and Isatu. As a result, they were able to buy a small home in Freetown. Isatu is just happy to have him home, happy to

have opportunities to work again, using the capital from the GoFundMe to buy items in bulk that she can sell at a modest profit at the local market, thereby generating enough money for living expenses and the next round of bulk purchases.

I found it extremely heartening to see all those donations pour in to this young man who has too often been neglected and ignored by society. It reminded me that when we know about suffering, when we are proximal to it, we are capable of extraordinary generosity. We can do and be so much for each other—but only when we see one another in our full humanity, not as statistics or problems, but as people who deserve to be alive in the world.

Henry not only managed to return to school; he also started making online videos on YouTube. I've been a YouTuber since 2007 and sent Henry some equipment so he could start his own channel because I felt it was important for people to be able to hear directly from Sierra Leone about the challenges, opportunities, and joys of life there. He turned his smartphone into a kind of window on his life in Freetown. Some days, he makes videos interviewing businesspeople in Sierra Leone—folks running print shops or selling phone cases from market stalls or popcorn in the streets. Other days, he'll make videos of him dancing with friends. He also runs a channel for Isatu where she shares traditional Sierra Leonean recipes and explains how she runs her shop.

His videos show the "constraints" of his community—people working with handheld tools to break down rocks or wooden pallets for a dollar or two a day, the challenges of running a market stand in a high-inflation economy, and so on. And he also makes videos showing the joy and connection of his community—sharing church services, stories of young students overcoming adversity, and his beautiful poems that embrace hope without ever being saccharine or sentimental. And he uses his platform to raise money. He has fundraised for clean water solutions in Freetown and for a lifesaving operation for a young boy.

In the years since his recovery, Henry has also become a TB activist with a special focus on raising money and attention for Lakka. He frequently makes videos there and encourages deeper global support for the hospital. “Lakka Hospital is developing gradually,” he noted in a recent video where he visited the hospital, highlighting the crumbling cinder block buildings and overgrown courtyards. He was there on a day when patients were being discharged after successful treatments, a joyous occasion. But he also knew that there was an area near Lakka where fresh graves were always being dug.

Henry seeks to fight the stigma of tuberculosis by openly identifying as a survivor and seeking to educate the public about the illness, and especially about Lakka, which still has a reputation as “a place you go to die.” In one video, he says, “You know, guys, I am one of the TB patients. I got discharged, and I am healthy and strong…. If you have the disease, you can overcome it. This place is not a bad place.” He encourages other survivors to “go out and preach that TB can be cured.”

How fortunate we are to have Henry among our number. Henry’s life and work today, both as a student and as an activist, embodies what can happen when people survive serious illness. He loves and is loved by family and friends. He learns and teaches. Either by text or phone, Henry and I talk a couple times a week, sometimes to strategize about YouTube, but also just to chat about life. Often in these calls, Isatu will appear in the background, cajoling and laughing with Henry. She will speak to him, and then Henry will translate: “My mother says she is still praying for you and Sarah and Alice and Henry.” Henry has also become a mentor and friend to my son Henry. They call each other “the namesakes.”

CHAPTER 23

THE CAUSE AND THE CURE

WE CAN UNDERSTAND THE HISTORY of tuberculosis as a story of competing paradigms: These days, we primarily see tuberculosis through a biomedical lens—as an infection caused by a bacterium and cured by drugs designed to kill or otherwise inhibit that bacterium. Others view TB through a religious paradigm—an illness caused by spirits or demon possession and cured by religious rituals or holy tinctures. In some communities, the illness continues to be, as it long was in Europe, viewed through a hereditary paradigm, where certain families or personality types are especially vulnerable to the disease. Still others view TB through a sociological lens, as an illness caused by poverty and marginalization.

The biomedical paradigm has become so powerful in my imagination that it's easy to forget how inadequate mere medicine can be. Yes, illness is a breakdown, failure, or invasion of the body treated by medical professionals with drugs, surgeries, and other interventions. But it is also a breakdown and failure of our social order, an invasion of injustice. The "social determinants of health"—food insecurity, systemic marginalization based on race or other identities, unequal access to education, inadequate supplies of clean water, and so on—cannot be viewed independently of the "healthcare system," because they are essential facets of healthcare. When someone living in Haiti contracts cholera, is the resulting illness *really* caused by a bacteria called *Vibrio cholerae*, or is it also caused by dirty water, by poverty, and by the reintroduction of cholera to the nation by aid workers after a 2010 earthquake? We cannot view "health" absent the

“social determinants of health,” or else we end up in situations seen all the time with TB, wherein people are, to cite just one example, unable to take their medicine because they don’t have enough food in their stomach.

I often think of these interdependent systems in the context of my own healthcare. Not long ago, I was walking in the backyard, staring up at the night sky, when I happened to step on a nail that went right through my shoe and an inch into my foot. The next morning, I drove on a good road to a clinic a few minutes from my house, where I received a booster shot to eliminate the already small chance that my mishap with the nail might result in tetanus. But in order for this minor medical intervention to occur, so many systems had to work in my favor: I needed healthcare access, of course—in my case, a health insurance program that pays for basic preventative care like vaccines. I needed to live in a community with twenty-four-hour electricity, so that the tetanus shot could remain cold and not lose its efficacy. I needed a system that could efficiently and reliably transport not just the shot itself, but also the gloves worn by the nurse who did my injection. I needed to live in a community with an education system strong enough to train nurses and doctors. Ultimately, what I needed was not just a tetanus shot but an entire set of robust systems to work perfectly in concert with each other—a phenomenon that ought not be a luxury in our world of abundance, and yet still somehow is.

I once asked a tuberculosis doctor, KJ Seung: Of the 1,300,000 people who will die of TB this year, how many would survive if they had access to the kind of healthcare I have? After all, while TB is often curable now, it remains a very difficult disease to treat, especially in cases of extensive drug resistance. And people in wealthy countries do continue to die of TB, albeit rarely—in the U.S., around five hundred people will die of TB this year. In Japan, over a thousand.

“How many would die if everyone could access good healthcare?” he asked me, as if he seemed confused by my question.

“Yes,” I said.

“None. Zero. Zero people should die of TB.”

It is difficult to imagine eliminating tuberculosis entirely. The disease has many animal reservoirs, and because a quarter of all people living are infected with it, the total elimination of TB is a distant dream. But we could live in a world where no one dies of TB. That choice would require sacrifices, as most choices do. We would need to reform our systems to include the impoverished as well as the rich, offering what Catholic liberation theologians called “a preferential option for the poor.” We would need to improve not just healthcare systems but also the social determinants of health—access to safe housing and adequate nutrition and reliable public transportation and so on. But this can be done—and in fact it *has* been done, which is why TB death is already rare in much of the rich world, although not as rare as it would be if everyone in rich countries could access good healthcare.

And this is why I would submit that TB in the twenty-first century is not *really* caused by a bacteria that we know how to kill. TB in the twenty-first century is really caused by those social determinants of health, which at their core are about human-built systems for extracting and allocating resources. The real cause of contemporary tuberculosis is, for lack of a better term, us.

That is bad news. But it is also good news. In 1804, there was nothing James Watt could do to save his son Gregory. In 1930, there was nothing my great-grandfather Charles could do to save his son Stokes. But we no longer live in that world, thanks to the accumulation and dissemination of knowledge about the illness and how to treat it. And so we have entered a strange era of human history: A preventable, curable infectious disease remains our deadliest. That’s the world we are currently choosing.

But we can choose a different world. In fact, we *will* choose a different world. The world *will* be different a generation from now. The question is whether we will look back in gratitude at the virtuous cycles, or in horror at the vicious ones.

TB activists and researchers have developed a comprehensive plan—and yes, of course it has an acronym: STP (Search, Treat, Prevent). The STP initiative would hire healthcare workers to Search for cases household by household around the world, diagnosing cases of TB before they become so serious or disabling as to require hospitalization. It would then Treat those diagnosed with a four-month course of antibiotics for most patients, and a six-month course for those with multidrug-resistant TB. And lastly, this program would Prevent further lines of infection by offering one month of preventive therapy to all those living in the same household as a person diagnosed with TB, because that preventive therapy helps to end the chain of transmission. If we spent twenty-five billion dollars on comprehensive care per year, we could drive tuberculosis toward elimination. We'd also save a lot of money in the long run—over forty dollars for each of those twenty-five billion dollars. Cutting the overall burden of TB means fewer future cases, and less expense to care for them.

We've already seen the benefits of STP programs in smaller communities, from Karachi to Lesotho. Within a few years, we could implement STP programs in select nations to prove their efficacy and efficiency in larger settings, and then move on to a global program. Combined with better vaccines and new approaches to treating TB, we could see an end to TB, or at least an end to TB's long reign as "the captain of all these men of death." That's one future for tuberculosis.

But that is not the only possible future. One can also imagine tuberculosis continuing to kill over a million people every year for another century, or another ten centuries. And yes, one can even imagine that if we continue to neglect research and treatment, someday soon a strain of tuberculosis could emerge that storms the world as the disease has so many times in the past,

and we return to the days when tuberculosis kills the rich and poor alike, even if never equitably.

We cannot address TB only with vaccines and medications. We cannot address it only with comprehensive STP programs. We must also address the root cause of tuberculosis, which is injustice. In a world where everyone can eat, and access healthcare, and be treated humanely, tuberculosis has no chance. Ultimately, we are the cause.

We must also be the cure.

POSTSCRIPT

Before I was obsessed with tuberculosis, I had a very different job. My books, including novels like *Looking for Alaska* and *Paper Towns*, tended to focus on grief and forgiveness and how we imagine each other. My characters experienced first love and heartbreak. Then I wrote a book called *The Fault in Our Stars* that, strangely, became a huge bestseller. As a result of that book's success, and the strong community that has grown around the YouTube videos I make with my brother, Hank, I have been given a loud, if somewhat fickle, megaphone. This particular megaphone is unusually dependent upon algorithms, and so I'm not always sure what or when it is going to project, or indeed whether it will seek the people I'm trying to reach or some entirely different group of folks.

The megaphone comes with other risks, too: When much of what you say is amplified, it is easy to drown out voices that ought to be heard. Also, the megaphone can hurt people's ears. You may just be trying to speak, but to others it can seem like shouting. I have tried, and often failed, to use this megaphone thoughtfully. For a while, I tried to get rid of the megaphone. But the megaphone comes with my job, and I love my job, and so eventually I found something useful to do with it.

At the urging of friends who worked at Doctors Without Borders, Treatment Action Group, and Partners In Health, I began talking about the TB crisis and the importance of lowering barriers to diagnostics and treatment. Together with thousands of TB Fighters around the world, we have advocated for the End TB Now Act before the U.S. Congress and pushed for more funding for antituberculosis initiatives in low- and middle-income countries. We have fought to convince drug companies to stop seeking secondary patents on lifesaving TB medication, and for the

corporate conglomerate Danaher to lower their test prices. We've met with some success—Danaher has lowered the price of their standard TB test, for example—but also many setbacks.

If you'd told me when *The Fault in Our Stars* was published that a decade later, I'd be writing and thinking almost exclusively about tuberculosis, I would have responded, "Is that still a thing?" It's only because I met Henry Reider in 2019 that this book exists, and that I've found a hopefully good use for the curious megaphone I lucked into. TB has become the organizing principle of my professional life over the last five years. It's nice to have something to think about before bed, and in the morning while brushing one's teeth, and while walking in the woods—and what I think about is tuberculosis. I think about the strange fact that we could end the TB pandemic, but haven't. I think about the people I've known who had TB, and how many of them aren't here anymore. I think about Shreya Tripathi reading my book before I knew that TB was still a thing. I think, "What if I had used my megaphone better back then?" I think, "Am I using it correctly now?" I think about the caregivers and patients who don't have megaphones, who often feel like they're screaming into the void.

In some ways, the landscape of tuberculosis care has never looked more promising. High-quality vaccine candidates are (very belatedly) now in late-stage trials, and shorter preventive care solutions are on the horizon. Mobile digital chest X-rays can mean faster diagnosis with the assistance of artificial intelligence. A quick, inexpensive TB test using tongue swabs is hopefully on its way. New antibiotic compounds are being tested. And innovation is driving improvements in outcomes. For example, Dr. Melino Ndayizigiye and his team have developed the TB Hunters app, which tracks infections and outbreaks in villages around Lesotho. But none of these new tools will matter if we don't make them broadly available through the open exchange of expertise and technology.

Dr. Jen Furin recently emailed me about the growing scourge of bedaquiline-resistant strains of tuberculosis. TB will always eventually learn how to evade the drugs we throw in its path, which is why it's so important that we continue to invest in new compounds. If I developed bedaquiline-resistant TB, it would be hard to cure but I'd still likely survive thanks to individually tailored treatment and access to the newest antibiotics. But for Dr. Furin's patient population, the outcome is almost always death. "There are so few options for them," she wrote to me, "and no access to the new drugs through compassionate use." This is the gut-wrenching, heartrending injustice of living with tuberculosis in the twenty-first century: You live if you're rich. And if you're not, then you hope to get lucky.

It is strange to call my friend Henry lucky when so many historical forces have pressed against his life, impoverishing him, his family, and his country. But he *was* lucky. His friend Thompson, as worthy of life as Henry or any of us, died. His roommate at Connaught Hospital died. But Henry, somehow, is still here with us.

The problem with statistics is that I cannot take in what it means to lose 1,250,000 people each year to a curable illness. That's more than a hundred thousand people a month. But how do I conceptualize such statistics? I've been in a stadium with a hundred thousand people, but I didn't know each of their families. I didn't know about the people they've loved, the heartbreaks they've endured, their constraints and encouragements, their frailty and resilience. I simply cannot fathom what 1,250,000 means.

But I can, just barely, fathom Henry. He and I talk all the time now. He likes to call me "Dad," and tells me that in Sierra Leone, *Dad* is a title you can be born into or one that you can earn.

He studies so hard. When he gets malaria, like he did most recently in 2024, he becomes sicker than most people because of the damage to his lungs, but still he studies. He was named the best TikToker at his college

last year. He wore a suit to the awards ceremony and brought his mom. He is proud of the small home he shares with her, but wishes he could afford art and decorations to put on the walls. He has a big, toothy smile. He is one of those young people who texts one word per text rather than all at once. “Dad,” he will send. And then, “Hello.” And then, “How is my namesake?”

He is excited about the publication of this book. He wants everyone to know about his YouTube channel, which you can find at https://www.youtube.com/@Tuberculosis-l1jSurvivorHenry/.

Henry is a human being, just as you are a human being. Consider yourself for a moment—everything you've overcome, everything you've survived. Think of the people who loved you up into your now. Think of how hard school is or was, how you were lucky or blessed to meet people you could love and who could love you. Think about how rare and precious humans are, and how many of them you get to worry for and care about. Then, if you can, find a way to multiply that times 1,250,000.

That is why we must work together to end tuberculosis and all other diseases of injustice.

ACKNOWLEDGMENTS

I must first thank Henry and Isatu Reider for generously sharing their time and stories with me. I am grateful for their friendship, their guidance, and the laughs we have shared. The list of folks in Sierra Leone I ought to thank would turn into its own book, but I especially want to express my gratitude to Dr. Girum Tefera, Dr. Bailor Barrie, Jon Lascher, Ashley Kappeler, Dr. Micheal Mazzi, Isata Dumbuya, and Sierra Leone's Minister of Health Dr. Austin Demby for their expertise and openness.

One of the great joys of this book has been the friendships that emerged from writing it, especially with Dr. Carole Mitnick at Harvard Medical School. At every turn, Carole has generously and patiently educated me about tuberculosis and the systems that underlie it, while encouraging this work. When I first became interested in tuberculosis, Carole and her community introduced me to a host of other experts I've been fortunate to learn from—including Jennifer Furin, Christophe Perrin, Salmaan Keshavjee, KJ Seung, Mercedes Becerra, Lucica Ditiu, Atul Gawande, and so many others. Each of these experts and caregivers has welcomed me into their work with enthusiasm and kindness.

I am so grateful to the TB survivors I've been blessed to meet and learn from, especially Phumeza Tisile and Handaa Enkh-Amgalan. Survivors and civil society groups are the heartbeat of the anti-TB movement, and their courage in standing up for others living with TB despite the stigma attached to the disease inspires me deeply. I'm also grateful to Shreya Tripathi, without whose efforts Henry might not be here—and I might not have written this book.

At my publisher, I'm perpetually grateful to Julie Strauss-Gabel, who when she signed up to publish my first YA novel in 2003 could never have

imagined that I would one day force her to become an expert in tuberculosis. At Penguin Random House, my publisher for twenty years now, I'm grateful to Elyse Marshall, Kaitlin Kneafsey, Anna Booth, Rob Farren, Natalie Vielkind, Grace Han, Vanessa Robles, Jen Loja, Helen Boomer, and Kim Ryan. Thanks also to my indomitable agent Jodi Reamer. This book was indefatigably fact-checked by Will Fraker, and I'm so grateful to all the early readers (especially KJ Seung, Carole Mitnick, and Jen Furin). All errors are my own.

Parts of this book started out as a video made with our production company, Complexly. Karim Hajj, Meghan Modafferi, and Stan Muller all contributed to this project in important ways. Rosianna Halse Rojas read every draft. Heather South, the lead archivist at the Western Regional Archives of North Carolina, helped solve mysteries about my great-uncle Stokes. Dr. Andy Bridge gave me helpful historical texts.

At Partners In Health, I'm grateful to Ophelia Dahl and Amy Huizing, who traveled with me to Sierra Leone in 2023, as well as to Gabi Palmi, Leslie Friday, Garrett Wilkinson, Jim Kim, Joia Mukherjee, Todd McCormack, Lindsay Palazuelos, Sheila Davis, Max and Deb Stone, and the entire PIH family. In some ways, this book is a love letter to our late friend Paul Farmer, who showed us the way.

I'm also thankful for other TB-fighting organizations whose passion has shaped my own, especially Stijn Deborggraeve, Saloni Fruehauf, and Shailly Gupta at Doctors Without Borders and Lindsay McKenna, Mike Frick, and David Branigan at Treatment Action Group. Thanks as well to other writers who share my fascination with TB, especially Maria Smilios and Vidya Krishnan.

In the field of fashion and health, I am indebted to Nicole Rudolph and especially Abby Cox, who invited me to her home to check out her collection of eighteenth- and nineteenth-century corsetry.

Thanks also to my family. Much of this book emerged from long conversations with friends, including Chris and Marina Waters. To my brother, Hank, who told me not to let his cancer get in the way of my tuberculosis activism: I love you so much. What a privilege it is to be the

tail to your extraordinary comet. My parents, Sydney and Mike Green, supported our family throughout this project. Thanks as well to my wonderful in-laws, Connie and Marshall Urist. My kids, Alice and Henry, have shown extraordinary patience and only occasional eye rolls as Dad related everything in their lives to tuberculosis. And this book would not exist without the support and editorial guidance of Sarah Urist Green, my first reader and favorite person, and my partner in the journey to understand health inequity and TB.

Finally, thank you to our online community of nerdfighteria, and especially to the TB Fighters, whose collective action has brought down the price of TB treatments and diagnostics and also reminded me in difficult days of what humanity can be when we work together in real solidarity. You are truly made of awesome.

FURTHER READING

When I began reading and writing about tuberculosis, I was very fortunate to come across Vidya Krishnan's arresting and brilliant book *Phantom Plague: How Tuberculosis Shaped History*, which I recommend to anyone interested in learning more about TB and its endless connections to our history and shared present. In the years since first reading the book, I've been fortunate to become friendly with Vidya, and to join her in the fight to lower barriers to TB diagnosis and treatment.

The most comprehensive history of tuberculosis that I know of is René and Jean Dubos's *The White Plague: Tuberculosis, Man, and Society*. Although published way back in 1952 at the dawn of the antibiotic era, it remains a fascinating read.

For a story of TB written from within, I heartily recommend Handaa Enkh-Amgalan's *Stigmatized: A Mongolian Girl's Journal from Stigma & Illness to Empowerment*, a moving exploration of surviving TB that doubles as a beautiful memoir following Handaa's journey from Mongolia to global work in human rights and refugee advocacy.

I also love Maria Smilios's history *The Black Angels: The Untold Story of the Nurses Who Helped Cure Tuberculosis*, which explores life in sanatoria while also telling the astonishing story of the Black American nurses who helped establish the cure for tuberculosis.

If you're interested in learning more about how microbes have shaped history, I recommend Frank M. Snowden's *Epidemics and Society: From the Black Death to the Present*, where I first learned about the romanticization of consumption. The book explores everything from the Black Death to the AIDS pandemic, and I find myself returning to it often.

To learn more about the history of Sierra Leone, I recommend Joe A. D. Alie's *A New History of Sierra Leone*, which explores the history of the country from a Sierra Leonean perspective.

Much of the demographic data—life expectancy, poverty rates, and so on—comes from the indispensable online tool *Our World in Data* at http://ourworldindata.org. This book, and so many like it, are deeply enriched by the work done by OWiD, but I also relied heavily on WHO statistics gathered by a wide variety of professional statisticians and epidemiologists. Their annual "Global TB Reports" are especially helpful for data nerds.

Those seeking an understanding of TB and the social determinants of health will find Paul Farmer's "Social Scientists and the New TB" to be essential reading. You can find it online or in the 1999 book *Infections and Inequalities: The Modern Plagues*.

To understand the sanatorium age, I recommend two memoirs: *The Baby's Cross: A Tuberculosis Survivor's Memoir* by C. Gale Perkins, which is quoted here extensively, and also *A Child of Sanitariums: A Memoir of Tuberculosis Survival and Lifelong Disability* by Gloria Paris. Both are visceral survivor accounts of growing up with TB and lives that straddled the line between an incurable and curable illness. I also really enjoyed the short history *Well Diary…I Have Tuberculosis: Researching a Teenager's 1918 Sanatorium Experience*, by Shirley Morgan, which tells the life story of Evelyn Bellak through the lens of her sanatorium diary.

But the most critical book for me in understanding the TB era remains Sheila M. Rothman's brilliant *Living in the Shadow of Death: Tuberculosis and the Social Experience of Illness in American History*. The sheer length of that subtitle might make you think it's a dreary read but in fact it bursts with life and insight.

To understand the rivalry between Louis Pasteur and Robert Koch, and their respective empires, I relied heavily on the wonderfully told and deeply researched book *The Remedy: Robert Koch, Arthur Conan Doyle, and the Quest to Cure Tuberculosis* by Thomas Goetz.

If you're looking to learn more about the relationship between consumption and fashion, I recommend Carolyn A. Day's *Consumptive Chic: A History of Beauty, Fashion, and Disease* but also two fashion historians who make video essays on YouTube: Nicole Rudolph (https://www.youtube.com/@NicoleRudolph) and Abby Cox (https://www.youtube.com/@AbbyCox).

For my understanding of eighteenth-century medicine and the relationship between the patient and the physician, I am deeply indebted to Barbara Duden's wonderful *The Woman Beneath the Skin: A Doctor's Patients in Eighteenth-Century Germany*, which is worth reading if you are interested in the history of medicine or if you just want a jarringly brilliant read.

To better understand New Mexico's quest for statehood and consumptives' quest for a cure, I recommend Nancy Owen Lewis's *Chasing the Cure in New Mexico: Tuberculosis and the Quest for Health*.

For a much deeper dive into the relationship between TB and HIV, among many other fascinating threads, I recommend *Discovering Tuberculosis: A Global History, 1900 to the Present* by Christian W. McMillen.

In seeking to understand how we've imagined good deaths through the last several centuries, I read and greatly enjoyed Philippe Aries's *The Hour of Our Death*.

To understand global health and the inequities built into our current systems for allocating health resources, I encourage everyone to read Dr. Joia Mukherjee's *An Introduction to Global Health Delivery: Practice, Equity, Human Rights*.

For more on the history of Partners In Health and their work on drug-resistant tuberculosis, check out *Mountains Beyond Mountains: The Quest of Dr. Paul Farmer, a Man Who Would Cure the World* by Tracy Kidder, one of the most important and deeply moving books I've ever read.

ABOUT THE AUTHOR

John Green is the award-winning, #1 bestselling author of books including *Looking for Alaska*, *The Fault in Our Stars*, *Turtles All the Way Down*, and *The Anthropocene Reviewed*. With his brother, Hank, John has co-created many online video projects, including Vlogbrothers and the educational channel Crash Course. John serves on the board of trustees for the global health nonprofit Partners In Health and spoke at the United Nations High-Level Meeting on the Fight to End Tuberculosis.

John lives with his family in Indianapolis. You can visit him online at johngreenbooks.com or join the TB Fighters working to end tuberculosis at tbfighters.org

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