PEOPLE COUNT

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CONTACT-TRACING APPS AND PUBLIC HEALTH

SUSAN LANDAU

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To the memory of Joseph Rotblat, who taught me the responsibilities of being a scientist, and Chiune Sugihara, who saved my grandfather's life

CONTENTS

PREFACE

In mid-February some colleagues and I flew to Brussels to brief European Union policymakers on a report on encryption policy we had prepared as part of a Carnegie Endowment for International Peace project. In our forty-eight hours there, we briefed members of the European Commission and European Parliament and held an evening convening with stakeholders from the government, industry, civil society, and academia. We managed a stop at a famed chocolate shop and a dinner out that included Belgian beer. As I headed home, one of the airline personnel asked me if I had been exposed to the coronavirus. "No," I laughed, "I have only been in Brussels." Little did I, or almost anyone else, know that the disease was already widely circulating in Europe.[1]

I flew home, then two days later went with my husband to visit our son in Austin. It was a tiring trip—as travel often is. Our son and daughter-in-law love to walk. I do too, but I am twice their age. We walked extensively through parks and interesting Austin neighborhoods. That Sunday night, I fell into bed, exhausted. The next morning, my husband flew back home, while I continued on to a planned trip to California. Tuesday morning, my throat was a bit sore. By the end of the day, my

symptoms had blossomed into a cold. That was unfortunate, as I was staying with two sets of friends and I had numerous appointments.

Aside from socializing with my friends, this trip put me in the midst of thousands of strangers. Two of my appointments had been set up by Tufts University, my employer, to advise me on a program we were setting up on cyber security and policy. I was also speaking on a panel on encryption policy at a huge computer security meeting of 36,000 people. Two days later, I spoke at a smaller meeting organized by UC Berkeley's law school on technology and the law. It was no time to be ill, but ill I was, with sneezes, coughs, a runny nose, an upset stomach, and fever.[2]

You know where this is going; I didn't, and neither did anyone else. I was surprised by the upset stomach—I'm always careful about what I eat when I travel—but not so surprised by fever; I can develop a fever at the drop of a hat. I was disconcerted by the fatigue that sent me to bed at 3:30 on Thursday afternoon and 6 pm on Friday. But even though I skipped the Berkeley banquet, I managed to give my two presentations. And because I was sneezing, I kept far away from people. No one on the panels, no one I had coffee with, and no one I visited fell ill—at least as far as I know.

I flew home. I was better the following week, though oddly and frighteningly, I coughed up blood a few times. But I was in Boston and my physician, a new one whom I had not yet met, was in western Massachusetts, where I live. I made an appointment for the following week in case the symptom persisted. This was now the first week in March, and coronavirus disease 2019 (COVID-19) was beginning to claim more of the American press's attention. More than once, I checked the website of the Centers for Disease Control and Prevention (CDC). But with the exception of the blood, all my symptoms pointed to a cold.

I had another busy week: Washington, DC, for an encryption policy briefing and running a two-day student symposium on cybersecurity policy at Tufts. Afterwards, my husband and I managed to squeeze in dinner with two old friends, biostatisticians at the Harvard Medical School and Brown University. They spoke very differently about the potential seriousness and danger of COVID-19 than did the news stories and CDC site. Two days later, I returned home as planned to western Massachusetts. It was March 10, and I was now recovered from my cold. I saw the doctor, who said my slightly painful ear—the only remaining symptom of the dreadful cold—would heal on its own.

Two weeks later, the *New York Times* published an article that caught my attention, titled "Lost Sense of Smell May Be Peculiar Clue to Coronavirus Infection." I'd had that symptom—anosmia—when I arrived in California. Two weeks after that, another *New York Times* story changed my understanding of my illness.[3]

"Google Searches Can Help Us Find Emerging COVID-19 Outbreaks," wrote Seth Stephens-Davidowitz in an opinion column. Stephens-Davidowitz said that searches for "my eyes hurt" had risen in exactly the parts of the United States that were experiencing high COVID-19 rates. One has to be careful about such data—earlier claims that searches for "flu" could be an early indicator of flu outbreaks, even ahead of CDC data, were incorrect—but this hint was later borne out by scientific studies that showed eye symptoms similar to conjunctivitis in a small percentage of COVID-19 patients. Bingo! I had spent the first several days in California looking at my eyes in the mirror, for they had been bothering me and I was concerned that I had conjunctivitis (I didn't).[4]

It was now over five weeks since I had fallen ill; it was impossible to get tested for COVID-19. Indeed, even when I was sick,

it would have been impossible for me to get tested: I didn't fit the criterion of having been to China or of having been in contact with someone who had. And while that is one of the multiple reasons the number of US cases exploded, it's also one of the factors that led to this book.

A second motivation to write this book relates to my research in cybersecurity. For twenty-five years, I've worked on issues of encryption and wiretap policy. This work has brought me to Brussels; Washington, DC; and other places, but it's also led me to ask important questions about the use of communications metadata. At just the moment in the spring of 2020 that the coronavirus was rapidly spreading around the world, I was exploring the types of private information one could discern from communications metadata. Meanwhile, I was hearing smart people propose using GPS data to track individuals with the disease—even though the coronavirus spreads best indoors, where GPS doesn't work well. I wrote a blog post to clarify this issue. As I learned that COVID-19 can spread from people who have no symptoms, I wrote another post on the efficacy of contact-tracing apps. Then, along with Christy Lopez and Laura Moy, I wrote a third on equity issues raised by using mobile apps for contact tracing. At that point, MIT Press sought a book on contact-tracing apps. Here I was.[5]

There was a third reason for this book, too: what was occurring in my hometown. I've lived in the countryside of New England for half my life, but I was born in New York City. That wonderful, dynamic, ethnically diverse city made me who I am. I am a first-generation American, the daughter of two people who never went to college (my father never finished high school). Yet I have a PhD from one of the best educational institutions in the world. Smarts got me there, but the education and

encouragement I received from New York City public schools allowed me to flourish. For that I am forever grateful.

New York City gave me many things—a lifelong love of theater and the New York City Ballet, a wonderful math and science education in high school, enrichment in Saturday science courses for high school students at Columbia University, the 92nd Street Y where I heard Marianne Moore speak, sculpture classes at MOMA, and so much more. The city also gave me a tremendous appreciation for the diversity of its people. I may love living in the country, but I never feel so alive as when I walk down New York streets and ride its subways.

In the spring of 2020, that world was being torn apart. COVID-19 was hitting people of color and poor neighborhoods far worse than it was the wealthy reaches of Manhattan. It was killing people, and it was potentially killing my city. I'm not an epidemiologist or a medical doctor, and I couldn't help cure the people who were ill. But if I, working in security and surveillance, could shed light on what kinds of protection contact-tracing apps can and can't provide, I will have given back a tiny bit to the city that has given me so much. Hence this book.

I am completing this book in early fall 2020; even as I write, the ground underneath contact-tracing apps is shifting. The details of the apps will undoubtedly be different by the time this book appears, and different again six months after its publication. If I focused precisely on what TraceTogether, Aarogya Setu, or any other app does or doesn't do, I will have written a book that is dated by the time the manuscript leaves my hands. Instead, I have sought to write a book that captures the essence of current contact-tracing and exposure-notification apps, discusses their limitations, and addresses the important social and

political issues their use raises. Those issues will not disappear with version 2.0 or 3.1, even though the details of any given app may differ slightly. If the discussion I present provides some insight for the policy choices we must make as we fight this pandemic or the next one—and the next one will come—then I will have succeeded in my mission.

1 INTRODUCTION

We are surrounded by germs. They fill the oceans and the soil, cover the outside of our skin, and inhabit our bodies. They are tiny—microscopic—and they come in a wondrous variety of forms: prions, viruses, bacteria, protozoa, and fungi.

Prions, the most recently discovered type of germs, are twisted forms of proteins that cause such illnesses as mad cow disease. Viruses are not alive; they can't even replicate unless they get inside a host cell and use the cell's replication machinery to reproduce. Bacteria are single-celled organisms that lack a nucleus but contain DNA. Protozoans are also single-celled, but unlike bacteria, they have a nucleus and other internal membranes. Fungi are organisms such as mushrooms that reproduce by spores; they can cause such mundane problems as athlete's foot.

We humans are vastly outnumbered by the microscopic world, at a scale that's difficult to comprehend. As a science writer for *Nature* put it, there are 100 million times more bacteria in the ocean alone than the number of stars known in 2011.[1]

Many of these microscopic organisms are necessary for life. Indeed, plants use bacteria to convert atmospheric nitrogen into a usable form, and we humans depend on the bacteria in

our digestive systems to break down and absorb the proteins, fats, and carbohydrates we eat. The viruses that replicate within and attack bacteria—bacteriophages—prevent certain types of harmful bacteria from entering our bodies through the soft tissues of our nose and mouth. Fungi produce delicious foods we know and love, including beer, bread, blue cheese, and truffles. More importantly, they also produce penicillin. And protozoa, which include amoeba and paramecia, eat bacteria and help the soil decompose; without them, we couldn't produce food.

But some germs kill us. Some target the lungs, kidney, or brain; others launch multipronged attacks against multiple parts of an organism. Their mode of transmission varies, too. Some germs travel through the air or through the exchange of body fluids, while others can be picked up from a surface on which they've landed or from contaminated water or food. Germs can be transmitted from person to person, or they may require an intermediate host. Malaria, for instance, travels between people via the bite of a mosquito. Germs are a complex adversary.

From the bubonic plague to influenza to AIDS, human societies have struggled to understand, fight, and end pandemics caused by germs. The challenges posed by COVID-19 may appear unprecedented, but most pandemics have a few things in common. And humans have learned how to use that in tackling the diseases.

People took on this fight against deadly germs well before they understood anything about viruses, bacteria, or protozoa. In the eleventh century, the Persian physician Ibn Sana observed that tuberculosis spread directly from person to person, even as the source of transmission remained a mystery. In 1668, an Italian physician, Francisco Redi, put fresh meat into three jars—a sealed one, an open one, and one covered with cloth

netting—and showed that maggots appeared only in the open-air jar. His experiment showed that maggots had to come from somewhere—that is, they could not spontaneously generate—but it didn't resolve the mystery of what caused disease.[2]

Less than a decade later, a Dutch shopkeeper studying rainwater and dental plaque provided another clue. While Galileo Galilei was busy training telescopes on the heavens, Antonie van Leeuwenhoek ground lenses to look at the microscopic world. He found dancing "small animals"—bacteria—within the seemingly clear rainwater and plaque. The teeming world of microscopic creatures living in, on, and around us appeared extraordinary—and indeed it was. Leeuwenhoek's microscope showed that we were supporting entire communities of living creatures on and in our bodies. But Leeuwenhoek was a naturalist, not a physician; his studies could reveal microbes' existence, but not their effects.

As the centuries passed, doctors and scientists continued to search for the causes of disease. In 1854, British physician John Snow combined knowledge of both people and place to stop a cholera outbreak in a crowded London neighborhood. The most popular theory at the time was that diseases spread through "miasma"—bad air. Snow didn't buy it, in part because of an observation he had made five years earlier: residents of adjoining houses, who presumably breathed the same air, could experience very different rates of cholera infection.[3]

What Snow had observed was that cholera would appear only after someone came from somewhere where the disease was present. Snow posited that the disease came from swallowing some kind of material that then multiplied inside the body; he became convinced that the disease was transmitted by water. He began studying water samples in the neighborhood of the outbreak.[4]

The outbreak had begun the night of August 31. Snow quickly realized that deaths were concentrated in the area surrounding the Broad Street water pump. He collected drinking water samples on September 3, but these contained relatively little organic matter. Later samples had much more. When he reported this to the neighborhood Board of Guardians—essentially the district's government—they immediately shut down the pump. Snow did not discover the bacterium responsible for the epidemic, but the connection he drew between the pump and the deaths stopped the cholera outbreak and changed the practice of public health.[5]

But what had caused the cholera? Snow didn't figure that out, but an Italian researcher did. In 1854, a year before Snow's investigation into the Broad Street pump, Filippo Pacini had found the comma-shaped bacteria that caused cholera (though he didn't show that it did so). But Pacini's paper lay ignored for decades, not the first or last time that correct scientific answers are overlooked. The bacteria's more famous discoverer was Robert Koch, the founder of bacteriology.

Koch, a German physician, was obsessed with finding the causes of diseases. He started with anthrax, a disease that could kill a healthy flock of animals in a matter of days—and then kill the farmer, too. Looking at the blood of dead sheep and cows, Koch noticed that the blood of infected animals contained microscopic rods. When he infected live mice with blood that contained these rods, the mice got infected too.[6]

Koch had discovered the anthrax bacillus. In time, he would also identify the bacterium that caused tuberculosis, and the one for cholera. But far more important than those discoveries were Koch's contributions to the germ theory of disease. Koch postulated that the link between bacterial organisms and diseases was

both specific and necessary. The anthrax bacillus could no more cause TB than could bad air, but unless the anthrax bacillus was present, there was no anthrax. Koch's work remains fundamental for how we treat infectious diseases.

Ending a plague requires more than medication: we need to stop spread. And for that, as John Snow demonstrated, we need data. Although the type of evidence differs, history offers ample data on the nature of pandemics: how they start, how they spread, how they end. Each disease begins as a mystery: what it is and how it spreads.

The history of the flu, which may be the world's oldest pandemic, is instructive. Some historians believe that flu epidemics occurred in India as early as four millennia ago. Long before airplanes and globalization enabled the global spread of disease, flu pandemics affected Asia, Europe, Africa, and the Americas. Relatively accurate records since an outbreak in Asia in 1580 indicate that flu has caused at least forty-six pandemics or serious epidemics in the past five centuries.[7]

Flu is an upper respiratory disease, which means that we spread it to each other by coughing and sneezing. Once you've had a particular strain of flu, you develop immunity to it—but there's a catch. The flu is caused by a virus, and like all viruses, its genes mutate. Most mutations are minor, enabling anyone who has developed immunity to a strain to retain it against a close version of the disease. But over the space of a year, a flu changes enough that a person doesn't necessarily have immunity to the most common strain in circulation.[8]

Flu is zoonotic; that is, it can infect multiple animals as well as people. This is one of the reasons the disease has proved so dangerous for humans. The virus can mutate within different

species—birds, pigs, horses—then come back to infect us. This ability to jump back and forth between humans and other animals increases the virus's capacity for mutation. When new versions make the jump from animal to human, they spread quickly around the world. Indeed, avian and swine flus have repeatedly done so.[9]

Flu normally kills 0.1 percent of those infected, but mortality rates vary dramatically according to the strain. The Spanish flu, a particularly deadly variety, killed approximately 10 percent of those infected when it raged across the globe between 1918 and 1920. Both death rates are extremely low compared to those of the Black Death, the fourteenth-century outbreak of bubonic plague that killed between a third and a half of the population of Europe.

Bubonic plague is caused by *Yersinia pestis*, a bacterium transmitted by fleas. The bacteria are present around the world; rodents, especially rats, carry the fleas that transmit the disease.

Although the *Yersinia pestis* bacterium still circulates in rodents and fleas, bubonic plague no longer represents a serious threat to humans. For one thing, the infection can be cured through quick antibiotic treatment (rapid response is critical; 60 percent of untreated sufferers die). And unless someone develops pneumonic plague—plague in the lungs—people don't transmit the disease to one other. Eliminating the vector of disease—the fleas or the rats—ends its spread.[10]

Similar tales of surprise and fear could be told about any number of epidemics, but let's focus instead on their most salient common features. Lethal diseases emerge from time to time; they can produce dramatic social effects in addition to grief and suffering. Over time, one of two things happens. Either the disease becomes endemic and we learn to live with it, or we figure out how to stop its spread.

Different factors affect whether a disease leads to an outbreak, an epidemic, or worse. One crucial aspect of spread is a disease's contagiousness. Epidemiologists use R_0, pronounced "R naught," to represent the "reproduction number" of a disease. An R_0 less than one means that, on average, each infection will result in less than one additional infection. The disease, in other words, will die out. If R_0 equals one, then each infection causes, on average, an additional one. The disease will not die out, but neither will it cause an epidemic. If R_0 is greater than one, however, each infection causes, on average, more than one infection. These are ripe conditions for an outbreak, an epidemic, or possibly even a pandemic.

Even for a given disease, R_0 varies with environment and human behavior. Respiratory illnesses are affected by weather; smallpox and flu, for example, spread less during damp periods. An infectious disease spreads more easily in the crowded immigrant dormitories of Singapore than on the sparsely populated pampas of Patagonia. For HIV/AIDS, R_0 varies with people's sexual behavior and drug use. And when there's "herd immunity," when most of the population has immunity to the disease, R_0 is lower.

R_0 estimates for COVID-19 were all over the map, from a low of 2.2 to a high of 5.7. The size of this range shouldn't be surprising. There's much we don't yet understand about COVID-19. Epidemiologists determine R_0 based on accurate data. At the beginning of the pandemic, cases were often misdiagnosed—and often not tested—due to stringent testing criteria and an incomplete list of symptoms. The fact that the disease can be spread by people who are asymptomatic only complicated epidemiologists' ability to pin down COVID-19's R_0. With better data, epidemiologists now believe R_0 to be between 2 and 3 in the absence of any types of controls such as masks and social distancing.[11]

High R_0s are a reason for concern, but they do not convey the full story of an epidemic's impact. The R_0 for measles is astounding, ranging from somewhere between 12 and 18 depending on community immunity. Smallpox has a much lower R_0—somewhere between 3.5 and 6—but is more dangerous than measles because its death rate is 30 percent compared to measles's 0.2 percent. The legendary Spanish flu of 1918 had a relatively mild—at least in comparison to the numbers we've mentioned so far—R_0 of 1.7–2. Yet the Spanish flu, with its 10 percent mortality rate, killed at least 50 million people, or around 3 percent of the entire world population.[12]

The way that a disease spreads matters nearly as much as its level of contagion. To cause a pandemic, a disease must spread efficiently as well as directly between people. A disease that kills its host too quickly—before the host has managed to infect others—will itself die out. And the disease can't kill everyone; it needs hosts to survive to attack another day.

Measles checks all the boxes for a potential pandemic. Its high R_0 is explained by the ease of which it is transmitted, either through airborne droplets containing the virus or through rash secretions. Up to 90 percent of people exposed to measles develop the disease. The disease can produce serious complications, including death. That's why the measles vaccine has been so important in eliminating this childhood disease in much of the world.

The habits of modern human life greatly increase the risk of global pandemics. By concentrating people, urbanization provides easy paths for diseases to spread from human to human. Poor sanitation and crowded conditions create spaces for disease to thrive. A classic example is tuberculosis, a curable disease that flourished in the early twentieth-century slums of New York City and continues to do so in those of India, China, Indonesia, the

Philippines, Pakistan, Nigeria, Bangladesh, and South Africa. And with air routes crisscrossing the planet, we have reduced the time in which a disease can spread from years to hours.[13]

The recent spread of cities into previously wild areas raises the risk of new infections. Zoonotic diseases are not new, but large concentrations of people now live in close proximity to wild animals. These creatures provide a rich pool of new pathogens.

Through trade, travel, and geographic expansion, humans have turned epidemics into pandemics. Germs need carriers. Bubonic plague traveled along the Silk Route from Asia to Europe in animal hosts, while West Nile virus made its way from Africa to Europe through migrating birds. In early 2020, foreign travelers fleeing Wuhan spread severe acute respiratory syndrome 2 (SARS-CoV-2), the coronavirus that causes COVID-19, across the globe.

From its first appearance in December 2019, COVID-19 has stretched our ability to cope with infectious disease. It is thought to have emerged from an animal host, in a crowded city and transportation hub, with confusing or even no symptoms, and is highly infectious. The leading US expert on infectious disease, Dr. Anthony Fauci, ticked off the timeline: "First notice at the end of December [2019], hit China in January, hit the rest of the world in February, March, April, May, early June." COVID spread like wildfire. Even for someone who spent his career imagining the dangers of a new pandemic, this disease turned out to be Fauci's "worst nightmare."[14]

Sometimes, in the battle of humans versus germs, the humans win. The elimination of smallpox, a disease that had caused epidemics for millennia, represents a victory of medicine and international cooperation. We don't know when humankind first suffered smallpox; there is "tantalizing speculation" that it was

as early as the tenth century BCE. The symptoms of smallpox include characteristic red spots that become small blisters that first fill with clear fluid, later changing over to pus. The lucky two out of three who survive are left with scars on their faces, arms, and legs.[15]

Highly contagious and thriving in densely populated areas, smallpox was endemic in European cities by the fifteenth century. Those who survived were immune, a fact that changed the course of history. When sixteenth-century European explorers brought smallpox and measles to the New World, Native Americans had never been exposed to either disease. The epidemics that followed ravaged the native population.[16]

Thanks to several centuries of vaccination and an ambitious eradication program, smallpox no longer poses a threat in ordinary life. Vaccination against smallpox was practiced in the Ottoman Empire in the sixteenth century, using a technique that appears to have been first developed in China or India. This method, variolation, which involves inoculating someone with live virus from smallpox patients, had a death rate of 1–2 percent. In the late eighteenth century, the English physician Edward Jenner developed a vaccine based on cowpox, a milder form of the illness that infected milkmaids and left them immune to smallpox. His vaccine inoculated against the disease without variolation's relatively high death rate.[17]

The possibility of vaccination reduced the number of smallpox fatalities, but the disease did not disappear. Instead, by the end of the nineteenth century, the disease was everywhere, endemic in most nations of the world. Half a century later, public health authorities floated an audacious plan to eradicate it.[18]

That this even seemed possible was the result of the disease's disappearance in the 1940s and 1950s from Europe and the Americas. In Europe, Canada, Mexico, and South America this

occurred through mass vaccination, while in the UK and US, it was done through controlling known outbreaks and selective vaccination. In 1959 the World Health Organization (WHO) launched a global eradication program that would attempt to vaccinate at least 80 percent of people in places with endemic smallpox. The program failed for a variety of reasons, ranging from a lack of funding to Cold War tensions to the politics of decolonization. But when WHO took up a second approach in 1967, it succeeded.[19]

Studies in Pakistan and elsewhere showed that the spread of smallpox could be stopped by focusing on case surveillance and containment (yes, "surveil" is the word health professionals use). With smallpox, it is easy to tell when someone is sick: infected people develop pocks on their face or other extremities. Instead of vaccinating everyone, the idea was to eliminate the spread of disease from one person to another. Such a technique can work with diseases that are spread through interpersonal contact and have no animal reservoirs.[20]

This public health approach, typically referred to as "surveil and contain," requires accurate reporting of the appearance of the disease. Before WHO could do anything else, it had to assure that reporting was accurate. Prior to the campaign, clinics and hospitals only documented the cases that came to them, which meant that they were missing ninety-nine cases out of a hundred. The patients hadn't been turning up because the health centers couldn't help them.[21]

Surveil and contain relies on contact tracing, that is, identifying people who have been in contact with an infected person. But in the smallpox campaign, public health authorities took surveil and contain a step further, to something called "ring vaccination." Everyone in close contact with a smallpox patient was to be vaccinated, as was everyone in close contact with the

contacts. The strategy worked, with the last case of endemic smallpox occurring in October 1977.[22]

Eradicating an infectious disease is the exception, not the rule. The disease can't have animal reservoirs that would allow it to reappear in mutated form. There must be obvious ways to discern when someone is infected, and cases must be accurately reported. There must be a vaccine. And there must be either a high rate of vaccination in the population or, as was the case with the end of smallpox, a sufficiently low spread that ring vaccination can work.

The success story of smallpox contrasts sharply with the world's failure to end tuberculosis (TB). In this case, the stars did not align, despite the fact that the disease can be cured by antibiotics. TB, a disease that has been with us since at least the time of the pharaohs, is widespread; a quarter of the world's population is infected, although only 5–10 percent of infected people will go on to develop the disease. Once a disease is present in so many people, the usual methods of public health—test, trace, isolate—become harder to implement. And over time, treatment has become less easy than it was; the disease has developed strains that are resistant to multiple antibiotics. Today, TB kills more people annually than any other infectious disease; people with weakened immune systems, including those with HIV/AIDS, are especially vulnerable.[23]

Diseases like TB and COVID-19 in which asymptomatic people can spread infection are particularly challenging to control. Another of these diseases is typhoid, a bacterial infection spread by eating or drinking food contaminated by fecal traces from the carrier. In the early twentieth century, public health authorities imprisoned an asymptomatic typhoid carrier and cook named Mary Mallon. Based on contact tracing and stool samples, public health authorities in New York accused

Mallon, an Irish immigrant, of infecting at least fifty-two people and killing at least two. Since there was no way to cure her infection, and since Mallon kept returning to work as a cook, public health authorities had her arrested and quarantined on North Brother Island twice—the second time for life. When there is no treatment for a disease, surveil and contain presents social and ethical dilemmas that public health authorities have yet to resolve.[24]

Aside from the question of vaccines, general prevalence, and the availability of treatment, public health authorities face a different problem when attempting to control sexually transmitted diseases like syphilis, gonorrhea, and HIV/AIDS (which is also transmitted through the blood). Surveil and contain only works when those infected are willing to share how they might have been infected and whom they might have passed the disease along to. Stigma, discrimination, gender expectations, the risk of arrest, and just plain personal privacy issues limit people's willingness to disclose information about their sexual partners, and this in turn limits authorities' ability to stop the spread of sexually transmitted diseases.

Pandemics end in various ways. The London cholera outbreak ended because John Snow traced the problem to water from the Broad Street pump—and got it shut down. Smallpox was eradicated by combining surveil and contain with ring vaccination. A pandemic can end because we find a cure, because we provide treatment even if we can't cure the underlying disease, or because we can vaccinate against it. But sometimes we are unable to contain the disease through any of these techniques. And then, unless you quarantine everyone—which is what Wuhan did in early 2020—you must find some way of locating those who have been exposed to the disease. That's contact tracing.

Contact tracing involves feet on the ground. Contact tracers interview people to find out the intimate details of their lives, including whom they spent time with or, in some cases, whom they had sex with. Contact tracers seek to learn where people spend their days. A tracer might have to tell someone to upend their lives, and their incomes, for a period of quarantine. The job is part detective, part social worker, and part medical investigator. It's labor-intensive and highly privacy-invasive. And it's never been tried before in a situation of wide community spread of a highly contagious respiratory disease.

Contact tracing also necessarily infringes upon the privacy and autonomy of individuals, albeit in order to end an epidemic. The questions a contact tracer asks constitute an invasion of privacy—and not for the purpose of protecting the individual, but to protect the public's health. Under the circumstances, it's not surprising that contact tracers have sometimes struggled to convince the public to participate. Yet we also know that, when it works right, contact tracing is one of the most effective tools of public health there is.[25]

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