MIT
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Review

Navigating a world reshaped by covid-19

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I am writing this on April 10, 2020. Twenty-five days have passed since San Francisco became the first US city to impose a stay-at-home order on its residents. It feels like six months. As the covid-19 pandemic has advanced across the planet at dizzying speed, economies and health-care systems have toppled like dominos. At this moment, a tracker run by Johns Hopkins University shows 1,617,204 confirmed cases of coronavirus infection and 97,039 deaths worldwide. That includes 18,279 deaths in Italy, 16,686 in the US, 15,843 in Spain, 12,210 in France, and 7,978 in the UK. By the time I finish writing, these numbers will all have markedly increased.

In China, by contrast, the death toll hovers at around 3,340. This week, people began emerging from lockdown in Wuhan, the city to which the outbreak was mostly contained. New York City's official toll is now 5,150, and that doesn't count people who were never tested for covid-19. In the first five days of April, 1,125 New Yorkers died on the streets or at home, an eightfold increase over the same period last year. The real toll, in other words, is surely at least double that of Wuhan, which is a larger city, and continues to climb at a terrifying rate.

Even allowing for claims that China is concealing some cases, it now seems incredible, in hindsight, that the rest of the world saw what was happening there and didn't act sooner. It seems even more incredible that more countries didn't shut everything down as soon as they saw the magnitude of the catastrophe in Italy, the first European domino to fall. Expecting the laws of viral spread to be different in New York from what they are in Lombardy or Hubei is like expecting the laws of physics to vary from place to place.

But it is in our nature to discount threats until they become personal. Yesterday I spoke to Craig Spencer, a New York City emergency room doctor who has spent time in China as well as in West Africa, where he worked on (and contracted) Ebola. He told me he knew the novel coronavirus would spread across the world as soon as he heard about the outbreak in Wuhan. Many other public health experts had spent years modeling and planning for similar pandemics. The Trump administration ran its own exercise in the fall of 2019. New York City created a ventilator stockpile in 2006, but stopped maintaining it. For all the planning by experts, the threat of a pandemic never felt real to politicians or to voters—until it became real.

This, then, is the paradox covid-19 has exposed: we are so tightly interconnected that a virus can reach each one of us, yet

Gideon Lichfield is editor in chief of MIT Technology Review.

so insular that we cannot conceive of what happens in one place repeating itself in another. As countries close their borders, hoard supplies, and throw blame at each other, the world risks becoming more insular still, further hampering global efforts to limit climate change (see page 54).

And yet this special issue on covid-19 is not unremittingly bleak. As we scrambled to produce pieces that would not feel dated by the time you read them, we realized that most of them were stories of hope amid the gloom: Herculean efforts to find a drug (page 34); scientists and technologists from all disciplines lending their expertise to the fight (page 40); blueprints for reopening society (page 8), rethinking data privacy (page 16), restarting the economy (page 70), redesigning mental health care (page 76), and safely holding an election (page 58); lessons to be learned from countries that have tackled their outbreaks more successfully (pages 30, 44, and 50); and stories of preparedness (page 64) and resilience in isolation (page 74).

These are grim times. But now that the threat is real to everyone, perhaps we can face it together.

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A blueprint for living in a world with covid-19.

By Gideon Lichfield

Artwork by Rob Sheridan

HOW DOES

HOW DOES WORK?

THE VIRUS

It might have jumped from bats to pangolins, an endangered species sometimes eaten as a delicacy, and then to humans.

How does it get into human cells?

The virus's protein spikes attach to a protein on the surface of cells, called ACE2. Normally, ACE2 plays a role in regulating blood pressure. But when the coronavirus binds to it, it sets off chemical changes that effectively fuse the membranes around the cell and the

*What it is,
where it comes from,
how it hurts us,
and how we fight it*

By Neel V. Patel

What is it?

A SARS-CoV-2 virion (a single virus particle) is about 80 nanometers in diameter. The pathogen is a member of the coronavirus family, which includes the viruses responsible for SARS and MERS infections. Each virion is a sphere of protein protecting a ball of RNA, the virus's genetic code. It's covered by spiky protrusions, which are in turn enveloped in a layer of fat (the reason soap does a good job of destroying the virus).

Where does it come from?

Covid-19, like SARS, MERS, AIDS, and Ebola, is a zoonotic disease—it jumped from another species to human hosts. This probably happened in late 2019 in Wuhan, China. Scientists believe bats are the likeliest reservoir; SARS-CoV-2's closest relative is a bat virus that shares 96% of its genome.

virus together, allowing the virus's RNA to enter the cell.

The virus then hijacks the host cell's protein-making machinery to translate its RNA into new copies of the virus. In just hours, a single cell can be forced to produce tens of thousands of new virions, which then infect other healthy cells.

Part of the virus's RNA also codes for proteins that stay in the host cell. At least three are known. One prevents the host cell from sending out signals to the immune system that it's under attack. Another encourages the host cell to release the newly created virions. And another helps the virus resist the host cell's innate immunity.

How does the immune system fight it off?

As with most viral infections, the body's temperature rises

in an effort to kill off the virus. Additionally, white blood cells pursue the infection: some ingest and destroy infected cells, others create antibodies that prevent virions from infecting host cells, and still others make chemicals that are toxic to infected cells.

But different people's immune systems respond differently. Like the flu or common cold, covid-19 is easy to get over if it infects only the upper respiratory tract—everything above the vocal cords. It can lead to complications like bronchitis or pneumonia if it takes hold further down. People without a history of respiratory illness often have only mild symptoms, but there are many reports of severe infections in young, healthy people, as well as milder infections in people who were expected to be vulnerable.
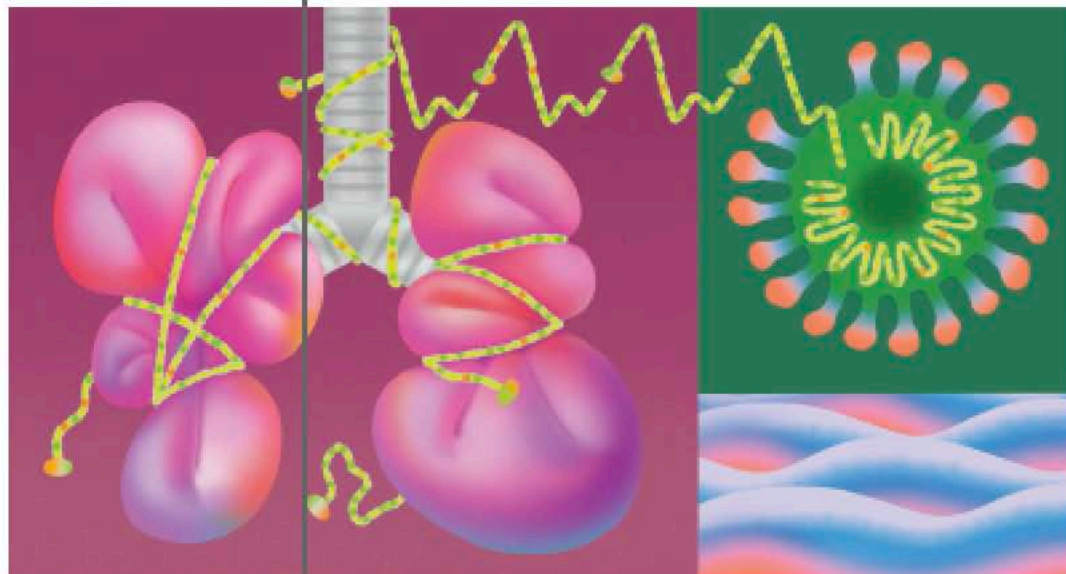
If the virus can infect the lower airway (as its close cousin, SARS, does more aggressively), it creates havoc in the lungs, making it hard to breathe. Anything that weakens the immune system—even heavy drinking, missed meals, or a lack of sleep—could encourage a more severe infection.

How does it make people sick?

Infection is a race between the virus and the immune system. The outcome of that race depends on where it starts: the milder the initial dose, the more chance the immune system has of overcoming the infection before the virus multiplies out of control. The relationship between symptoms and the number of virions in the body, though, remains unclear.

If an infection sufficiently damages the lungs, they will be unable to deliver oxygen to the rest of the body, and a patient will require a ventilator. The CDC estimates that this happens to between 3% and 17% percent of all covid-19 patients. Secondary infections that take advantage of weakened immune systems are another major cause of death.

Sometimes it is the body's response that is most damaging. Fevers are intended to cook the virus to death, but prolonged fevers also degrade the body's own proteins. In addition, the immune system creates small proteins called cytokines that are meant to hinder the virus's ability to replicate. *Overzealous* production of these, in what is called a cytokine storm, can result in deadly hyper-inflammation.

How do treatments and vaccines work?

There are about a half-dozen basic types of vaccines, including killed viruses, weakened viruses, and parts of viruses, or viral proteins. All aim to expose the body to components of the virus so specialized blood cells can make antibodies. Then, if a real infection happens, a person's immune system will be primed to halt it.

In the past it has been difficult to manufacture vaccines for new zoonotic diseases quickly. A lot of trial and error is involved. A new approach being taken by Moderna Pharmaceuticals, which has a vaccine about to enter clinical trials, is to copy genetic material from a virus and add it to artificial nanoparticles. This makes it possible to create a vaccine based purely on the

genetic sequence rather than the virus itself. The idea has been around for a while, but it is unclear if such RNA vaccines are potent enough to provoke a sufficient response from the immune system. That's what clinical trials will establish, if they first prove that the proposed vaccine isn't toxic.

Other antiviral treatments use various tactics to slow down the virus's spread, though it is not yet clear how effective any of these are. Chloroquine and hydroxychloroquine, typically used to fight malaria, might inhibit the release of the viral RNA into host cells. Favipiravir, a drug from Japan, could keep viruses from replicating their genomes. A combination therapy of lopinavir and ritonavir, a common HIV treatment that has been successful against MERS, prevents cells from creating viral proteins. Some believe the ACE2 protein that the coronavirus latches onto could be targeted using hypertension drugs.

Another promising approach is to take blood serum from people who have recovered from the virus and use it—and the antibodies it contains—as a drug. It could be useful either to confer a sort of temporary immunity to healthcare workers or to combat the virus's spread in infected people. This approach has worked against other viral diseases in the past, but it remains unclear how effective it is against SARS-CoV-2.

With additional reporting from Antonio Regalado.

Neel V. Patel is a senior reporter at MIT Technology Review.

WHAT IS SEROLOGICAL TESTING?

The race to develop tests that will tell us how widespread the virus is.

By Antonio Regalado

The US and other countries are scrambling to test hundreds of thousands of people to see if they are infected by the coronavirus. That test, which employs a technique called PCR, looks directly for the genetic material of the virus in a nasal or throat swab. It can tell people with worrisome symptoms what they need to know: Are they infected right now?

But a swab cannot tell you if you've had the disease in the past—which means we may not understand the full extent of its spread, or whether large numbers of people have already been infected and recovered without showing symptoms.

The answer to this is a different kind of test, one that can look at people's blood to find the telltale traces that show if somebody's immune system has been in contact with the virus. This procedure, known as a serological test, asks a different question—not "Does this person have coronavirus?" but "Has this person's body ever seen the germ at all?"

What is a serological test?

Serological tests work on blood samples rather than nasal swabs. These types of test for coronavirus are being developed by a number of labs around the world. The blood of someone who has been exposed should be full of antibodies against the virus. It's the presence, or absence, of such antibodies that the new tests measure. Among those developing such tests are researchers at the Icahn School of Medicine in New York City, led by Florian Krammer.

How does it work?

To make their version of a test, the Icahn team produced copies of the telltale "spike" protein on the virus's surface. That protein is highly immunogenic, meaning that people's immune systems see it and start making antibodies that can lock onto it. The test involves exposing a sample of blood to bits of the spike protein. If the test lights up, it means that you have the antibodies.

To check their results, the team inspected blood samples collected before covid-19 came out of China this year, as well as blood from three actual coronavirus cases. According to Krammer, the test can pick up the body's response to infection "as early as three days post symptom onset."

What impact could testing have on treatment?

Krammer believes serological testing could have immediate implications for treatment by helping locate survivors, who could then donate their antibody-rich blood to people in ICUs to help boost their immunity.

What's more, doctors, nurses, and other health-care workers could learn if they've already been exposed. Those who have—assuming they are now immune—could safely rush to the front lines and perform the riskiest tasks, like intubating a person with the virus, without worrying about getting infected or bringing the disease home to their families. But tests could have a bigger impact too.

What else can it tell us?

How widespread is the new coronavirus? How many people get it and don't even know? What is the actual death rate? Those are some of the biggest questions that science doesn't have the answers to yet.

Serological tests, if they are done widely and quickly enough, could give an accurate picture of how many people have ever been infected. And that is the figure disease modelers and governments urgently need to gauge how deep society's shutdown needs to be.

At the time of writing, the coronavirus had killed more than 52,000 people, or about 5% of the confirmed cases: a shocking death rate. But the real fatality rate among everyone infected by the virus is certainly lower, and possibly much lower, than current figures can tell us. The reason epidemiologists can't say for sure is that they don't know how many people are infected but never go to the hospital or even have symptoms. And

that's a huge problem for setting policy.

John Ioannidis of Stanford University argued in the publication Stat that the true death rate could be less than that of the seasonal flu. If so, "draconian countermeasures" are being implemented amid an "evidence fiasco" of "utterly unreliable" data about how many people are infected. Another report, meanwhile, estimated that early in the outbreak only 10% to 20% of the actual infections were being documented. Without more testing, nobody can be truly certain what the next steps should be.

What next?

Other scientific centers, in Singapore and elsewhere, also say they have antibody tests running, as do some US companies selling products to researchers. The US Centers for Disease Control and Prevention says it is developing one; the UK planned to produce millions of at-home testing kits that use finger pricks of blood, but they have run into difficulties with accuracy.

To learn the true extent of infections, the next step for researchers—in New York or elsewhere—is to carry out "serological surveys" in which they'll do the test on blood drawn from large numbers of people in an outbreak area. That may tell them exactly how many cases have gone unnoticed.

But it could be some time before scientists learn the answer. Krammer says the effort to carry out a wider survey is "just starting." T

THE REAL FATALITY RATE AMONG EVERYONE INFECTED IS POSSIBLY MUCH LOWER THAN CURRENT FIGURES TELL US.

Antonio Regalado is a senior editor at MIT Technology Review.

WHAT IS HERD IMMUNITY?

The controversial approach to bringing the pandemic to an end.

By Antonio Regalado

There are basically three ways to stop covid-19 for good. One involves extraordinary restrictions on movement and assembly, as well as aggressive testing, to interrupt its transmission entirely. The second is a vaccine (see "The race for an antibody drug," page 34). A third is potentially effective but horrible to consider: just wait until enough people catch the disease.

If the virus keeps spreading, eventually so many people will have been infected and become immune—as long they survive—that the outbreak will fizzle out on its own as the germ finds it harder and harder to find a susceptible host. This is known as herd immunity.

When experts suggest that a worst-case scenario would lead to 60% of the world's population being infected, they aren't making a guess. The numbers are informed by the point at which epidemiologists say herd immunity should kick in.

Early in the outbreak, UK prime minister Boris Johnson indicated that his country's official strategy might be to put on a stiff upper lip and let the disease run its course. Mark Rutte, prime minister of the Netherlands, struck a similar note, saying, "We can slow down the spread of the virus while at the same time building group immunity in a controlled way."

But many models suggest that shooting for herd immunity right away would be a disastrous strategy. That's because many of the people infected will become severely ill—and a sudden boom in sick people needing hospitalization or intensive care will overwhelm the health-care system.

The UK ultimately decided to enforce strict lockdown rules and try to suppress the virus. But even if this slows the pandemic down, it may still take herd immunity to bring it to an end.

What exactly is herd immunity?

When enough of the population is resistant to a germ, its spread stops naturally because not enough people are able to transmit it. Thus, the "herd" is immune, even though many individuals within it still are not.

Although it is ghastly to contemplate the prospect of billions being infected by the coronavirus—which has an estimated fatality rate somewhere around 1%— we've seen evidence for the emergence of herd immunity in other recent outbreaks.

How does herd immunity work?

If each person who is infected passes the virus along to two more people, there's an exponential increase in infections—but once half the population is infected, the outbreak can no longer grow. The precise point at which herd immunity is achieved, however, changes depending on the disease.

Consider Zika, a mosquito-borne illness that caused panic in 2015. By 2017, Brazilian researchers found that 63% of the population in the northeastern beach city of Salvador had already had exposure to Zika; the researchers speculated that herd immunity had broken that outbreak.

Vaccines create herd immunity too, either when given widely or when administered in a "ring" around a new case of a new infection. That's how diseases like smallpox were eradicated and why polio is close to being erased.

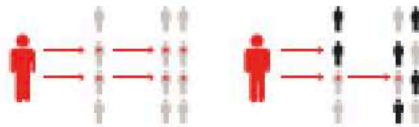## Can people become immune to coronavirus?

With many germs, people who are infected and recover become resistant to getting that disease again, because their immune system is charged with antibodies able to defeat it.

As hundreds of thousands of people recover from the coronavirus, it's likely they are now resistant, although the degree of immunity remains unknown. "I would be surprised, but not totally surprised, if people did not become immune," says Myron Levine, an infectious disease expert at the University of Maryland. Some viruses, like the flu, do find ways to keep changing, which is why immunity against such seasonal germs isn't complete.

When do we reach herd immunity?

The point at which we reach herd immunity is mathematically related to the germ's propensity to spread. This is expressed as its basic reproduction number, or R_0—the average number of people to whom each infected person passes the germ when everybody is susceptible. As more people become immune, and it becomes harder for the virus to find new hosts, the effective reproduction number (denoted as just R) falls. If R_0 is 2, as in our earlier example, half the population has to become immune for R to reach 1, the point at which the outbreak stops growing. If R_0 is higher, so is the proportion needed to reach herd immunity. Measles, one of the most easily transmitted diseases, has an R_0 over 12, and requires about 90% of people to be resistant for unprotected people to get a free ride from the herd. That's why new outbreaks can start when even small numbers of people opt out of the measles vaccine.

In a simple model, each case infects two more. Once half the population is immune, an outbreak no longer grows wider.

● Infected
○ Susceptible
● Immune

Estimates made in early March suggested an R_0 for the coronavirus between 2 and 2.5. That rate of spread is higher than for ordinary flu, but similar to the rate for emergent influenzas that have occasionally swept the globe before. "That is similar to pandemic flu of 1918, and it implies that the end of this epidemic is going to require nearly 50% of the population to be immune," Harvard University epidemiologist Marc Lipsitch told a gathering of experts.

But in late March researchers at London's Imperial College, using data from 11 European countries, estimated R_0 for coronavirus at 3.87. That would mean nearly three-quarters of the population would have to be immune before the effect kicked in, according to the simplest model.

What are the costs of getting there?

Whether it happens at 50% or 80%, the implication is that billions will be infected and millions killed around the world before herd immunity takes over. Many epidemiological models recommend aggressive "suppression" of the virus: isolating sick people, trying to reduce social contacts by at least 75%, and closing schools.

"Suppressing transmission means that we won't build up herd immunity," says Azra Ghani, an epidemiologist who is one of the leaders of the team modeling the outbreak at Imperial College. The trade-off of success is "that we are driving it down to such a low level that we have to keep those [measures] in place." T

Antonio Regalado is a senior editor at MIT Technology Review.

Why some countries have faced far better in the battle against covid-19 than others—and what we can learn from them.

By James Crabtree
Illustrations by Franciska Gorczyk

HOW TO MANAGE A PANDEMIC

My first taste of coronavirus panic came early one morning in January. An email with the heading IMPORTANT INFORMATION PLEASE READ arrived from our son's elementary school, just minutes before we put him on the bus. The parents of one of his teachers, who had recently returned from China, had been infected—Singapore's cases 8 and 9, as it turned out—and the teacher in question was being quarantined.

Singapore was among the first countries to suffer an outbreak. In the months since, it has been at once reassuring and unnerving to watch its journey from an early hot spot to a kind of haven state, holding out doggedly against an invader that has infiltrated so many others.

Early commentary in the West focused on the failings of China's autocratic system, which hid the severity of Wuhan's outbreak—at what we now know to be catastrophic cost. The more the epidemic has spread, the more it has become clear that Western liberal democracies have badly mishandled it too, ending up with severe outbreaks that could—perhaps—have been avoided.

Yet it makes a little sense to view the coronavirus as some kind of perverse vitality test for liberal and authoritarian regimes. Instead we should learn from the countries that responded more effectively—namely, Asia's advanced technocratic democracies, the group once known as the "Asian Tigers." In the West the virus exposed creaking public
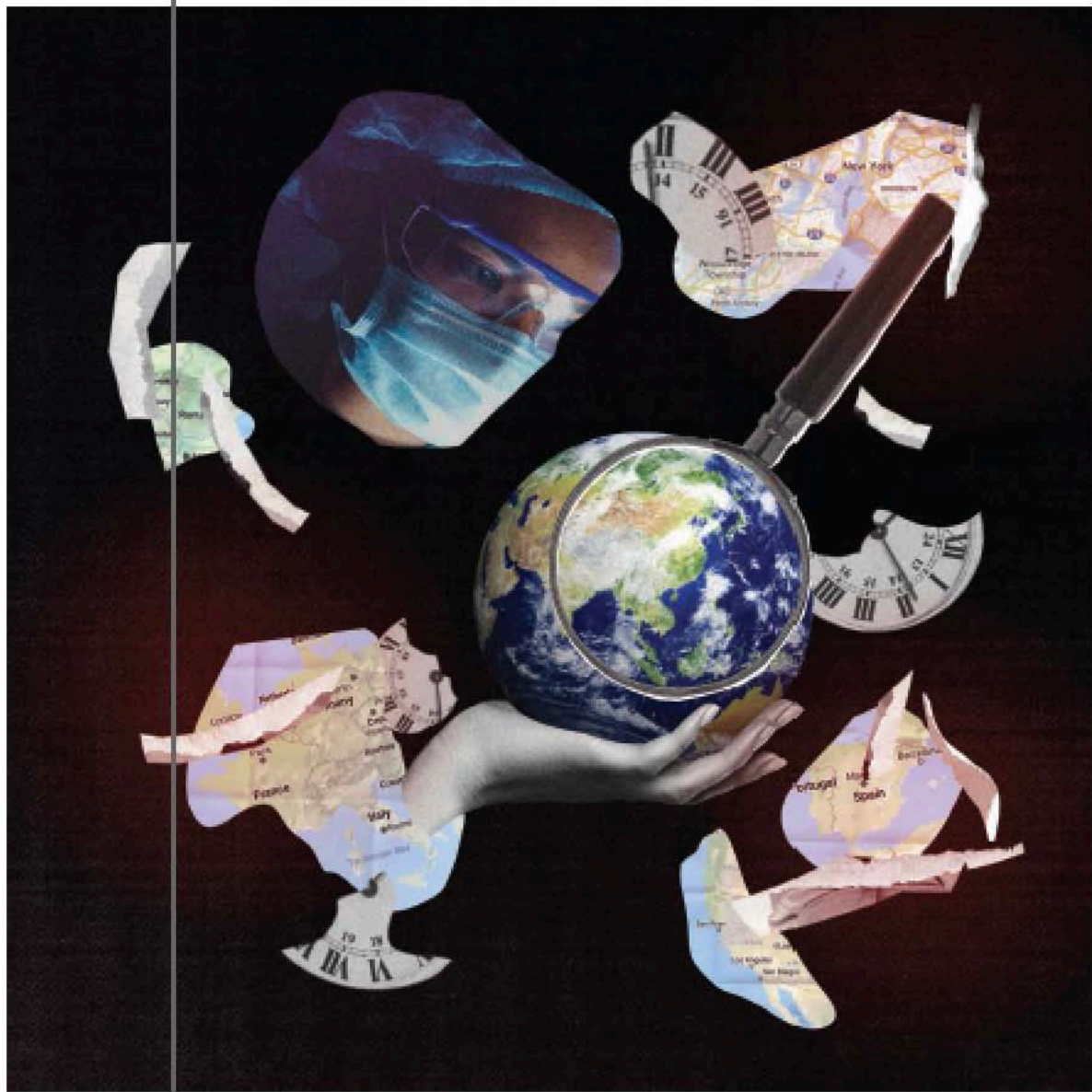
THE RACE FOR AN ANTIBODY DRUG

One of the most promising approaches to making a treatment for covid-19 is to find a powerful antibody in the blood of survivors and produce enough for the rest of us.

By Antonio Regalado
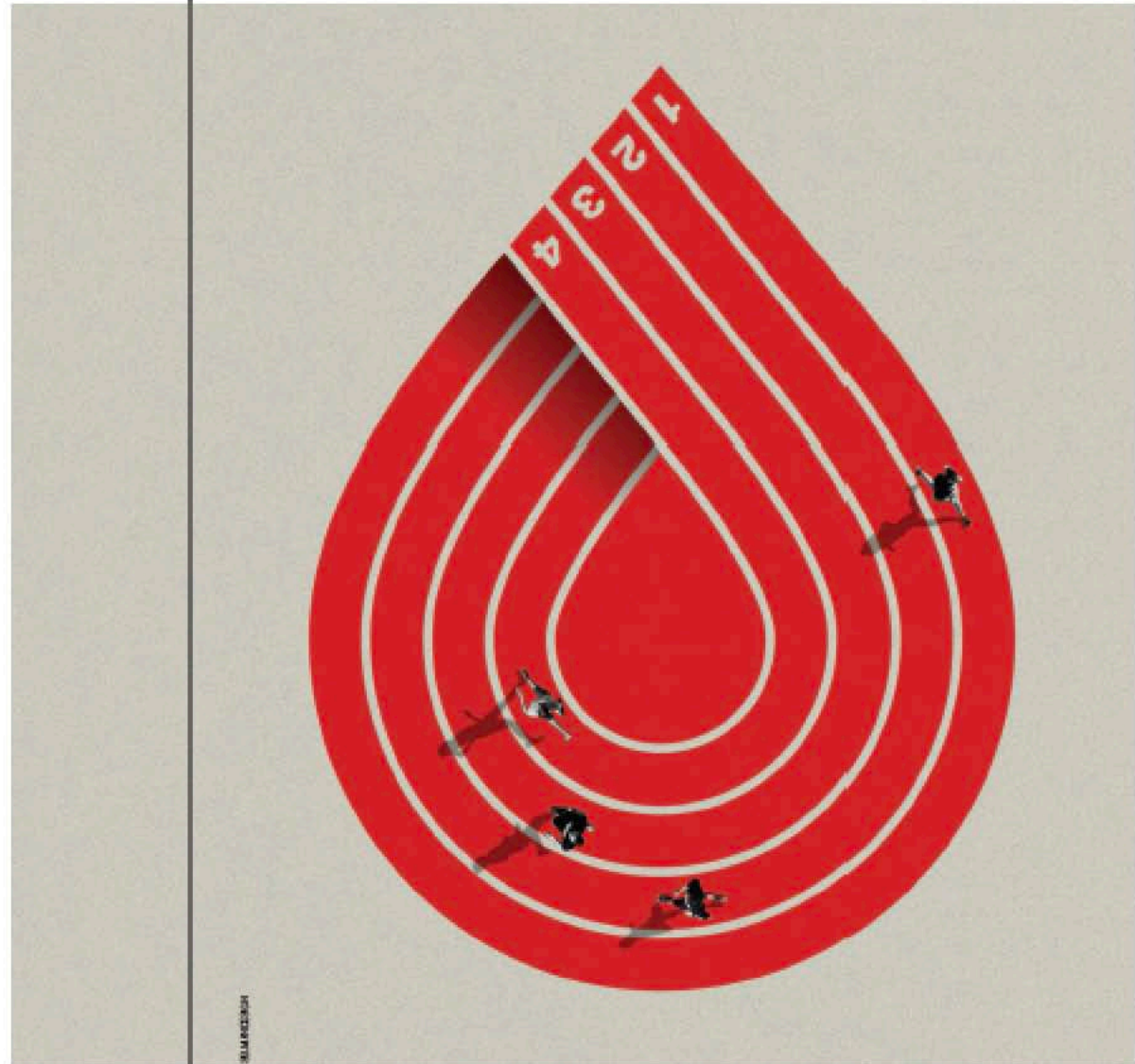
By Sonia Faleiro

THE TRACE RACE

Even with a national government asleep at the wheel, one Indian state showed the world the right way to tackle coronavirus.

A government health worker in Kerala checks a boy's temperature.

THE SUN HAD ALREADY SET ON March 7 when Nooh Pallichalil Bava received the call. "I have bad news," his boss warned. On February 29, a family of three had arrived in the Indian state of Kerala from Italy, where they lived. The trio skipped a voluntary screening for covid-19 at the airport and took a taxi 125 miles (200 kilometers) to their home in the town of Ranni. When they started developing symptoms soon afterward, they didn't alert the hospital. Now, a whole week after taking off from Venice, all three—a middle-aged man and woman and their adult son—had tested positive for the virus, and so had two of their elderly relatives.

PB Nooh, as he is known, is the civil servant in charge of the district of Pathanamthitta, where Ranni is located; his boss is the state health secretary. He'd been expecting a call like this for days. Kerala has a long history of migration and a constant flow of international travelers, and the new coronavirus was spreading everywhere. The first Indian to test positive for covid-19 was a medical student

THE LESSONS OF EBOLA

TR :
Q + A

Smarter leadership makes a big difference, says Christopher Kirchhoff, who worked on US Ebola response.

By Konstantin Kakaes

Q: What steps did the US government take after the 2014 Ebola outbreak?

A: An emergency spending bill that was passed by Congress in December 2014 included $1 billion that the administration used to address some crucial weaknesses. Many nations around the world didn't have testing capabilities to be able to notice when a novel or really lethal pathogen emerges. Using that money, we partnered with more than 60 countries to introduce much more widespread testing capability to detect pathogens when they first emerge. Then we conducted a country-by-country assessment of how strong their emergency response and public health systems are and worked with each country to strengthen their preparedness and response capabilities.

We also established a network of Ebola treatment centers: 35 hospitals across the United States, plus a number of labs that were designated by the federal government. If somebody were to come down with Ebola or another highly lethal pathogen, they wouldn't be more than two hours away from a hospital that was designed to treat them.

Another thing that was really important was the creation, toward the end of the Obama administration, of a new office in the White House called the Global Health Security Directorate.

This new office within the National Security Council had two functions. The first was to coordinate the response in the event of a future crisis. The second purpose was that it would be responsible for seeing through substantial structural changes in many departments and agencies. These were the kinds of reforms that wouldn't happen on their own, without organized follow-through from the White House.

Q: Can you go into a little bit more detail on what those structural changes were?

A: On the domestic side, the very small number of Ebola cases that we had in the US showed major gaps in how federal, state, and local authorities responded together. Because the US has a federal system where most public health authorities are actually at the local levels, but most capability is at the federal level, we had to have tighter coordination in the future to respond.
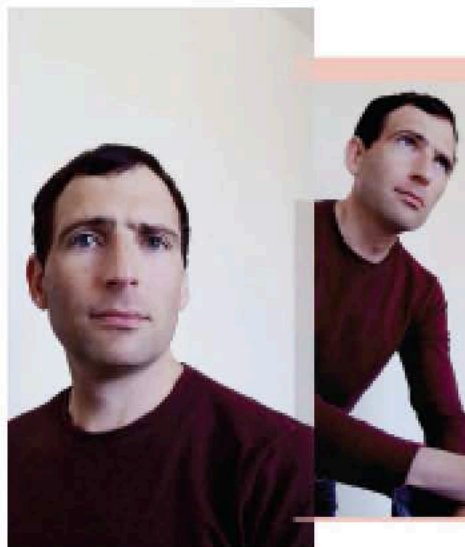
On the international side, we discovered whole new doctrines for how to respond to an outbreak abroad by using different capacities in government. Never before had the military been used to support civilian health responders in the way it was in West Africa.

Q: Do you think the existence of an office like that would have made a substantial difference to the prevalence of the novel coronavirus in the US today?

A: Yes. The office was dissolved in May 2018. But Ebola taught us that there's an incredible penalty for inaction, because epidemics grow exponentially: every day you delay responding, you end up facing a steeper exponential curve that makes the situation quickly transition from what would have been manageable to something that's unmanageable. This is when we are today. You have to imagine that the presence of an office well staffed with professionals in emerging infectious diseases would have been able to help the US government be more nimble in those crucial early days, when more capabilities could have been brought online and could have been ready to help us get ahead of the curve.

Q: What do you think might have happened had the Obama administration not sent nearly 3,000 military personnel to West Africa?

A: I think you would have seen the epidemic continue the way that it was growing in August 2014, when it was doubling in size every three weeks. Although the three countries where the outbreak was concentrated don't themselves have high rates of international travel, there are land routes to other African nations. One scenario that people worried about tremendously was Nigeria — not only having the mega-city of Lagos, but in the north of Nigeria having insecure conditions with an Islamic insurgency that might well have prevented international health responders from accessing those who needed care, which could have resulted in Ebola becoming endemic in Africa.

Q: Has the role the current administration has given to scientific research limited the effectiveness of the US government response?

A: I think it's unavoidable to talk about the fact that the CDC [Centers for Disease Control] budget has been significantly decreased, that administration budgets have continually advocated for dramatic cuts to research and development. Programs oriented to delivering therapeutics and vaccines have been affected in this process. And in an emergency like this, you want to have more of them.

Q: What role did the private sector play in 2014, and what can be done today?

A: There were tremendous contributions during the Ebola outbreak from both the private sector and the philanthropic sector. Paul Allen pledged $100 million to fight Ebola, and his foundation developed an ability to safely transport people infected with Ebola on airplanes so they could be medically evacuated. This was a capability that the US military didn't even have. We're seeing the same thing today, where the Gates Foundation stepped up in Seattle and rolled out test kits before the government was able to.

At Schmidt Futures, the philanthropy I work for, we're doing a lot of thinking about the role technology can play. One of the efforts we've already funded is using online education tools to train people to use ventilators. It turns out that we have very few ventilators, but we have even fewer people able to operate them.

Another example: there's a great race among technologists in Western countries to be able to do location-based, smartphone-enabled contact tracing in a privacy-protected way. There are several different architectural approaches to this. It could be an enormously powerful tool — particularly toward the latter stages of an outbreak, when you revert from a situation with widespread community transmission to just a few carriers who nevertheless still infect others. Just like in Ebola, contact tracing is the only way, at the tail end of an outbreak, to ensure that an outbreak is stopped in its tracks. This gives technologists an important window to experiment with different capabilities that could be enormously important if they were to come online two to four to six months from now.

Q: How optimistic are you that we will learn lessons from what's going on now that will enable us to be much more effective in fighting epidemics in the future?

A: What we are living through now will be hard to forget. So I think there will be an intense focus on how to prevent an outcome like this in the future, but there will be no substitute for leadership to see through the very significant changes that are necessary if we want to grow our capacity on all fronts for outbreaks.

I think there's an enormous opportunity for Congress to lead on making investments that not only will help us respond today but will help us grow the capacity of our response systems in the future. Investments in helping hospitals all across the United States be able to surge capacity in the event of an emergency; investments in our ability to rapidly produce diagnostic testing; investments in our public health infrastructure at the state and local level; investments around the world, particularly in nations that are not themselves well equipped to confront the outbreak of novel diseases. And that work can begin now.

This interview has been condensed and edited for clarity.

Konstantin Kakaes is an editor at MIT Technology Review.

Christopher Kirchhoff was a member of the White House Ebola Task Force. He is a senior fellow at Schmidt Futures.

THE CLIMATE IS ALSO A CASUALTY

Global cooperation on climate change was already faltering.
Things just got much worse.

By James Temple

On the early afternoon of December 15, the gavel fell at the UN COP25 conference in Madrid. The weeks of negotiations over crucial pieces of the Paris climate agreement reached four years earlier had ended in failure. Despite spending nearly two days longer than scheduled, thousands of delegates departed the convention halls deadlocked on the basic rules required to move forward.

There's plenty of blame to go around. But by most accounts, Australia, Brazil, and the United States—each now run by nationalist leaders who rose to power in part on promises to defy global demands for greater climate action—took special pains to thwart progress.

Brazil immediately backed out of hosting the convention after the election of Jair Bolsonaro, and its delegates spent their time in Madrid arguing for the need to open up the Amazon for farming and mining. The US, on track to exit the accords altogether under President Donald Trump, stonewalled efforts to establish a process for providing funding and support to poor nations hit by climate disasters.

In the end, nearly every major decision at COP25 was punted to the next conference, originally scheduled for this November in Glasgow. "The can-do spirit that birthed the Paris agreement feels like a distant memory today," Helen Mountford, vice president for climate and economics at the World Resources Institute, said at the close of the talks.

Two weeks later, researchers in China identified a deadly new coronavirus strain that had infected dozens of people, marking the start of the global pandemic. Borders slammed shut. Global trade stalled and markets crashed. Countries traded accusations and insults. In a matter of weeks, any lingering momentum behind efforts to jointly confront climate change essentially vanished.

As the worldwide death toll accelerated, countries locked down cities, banned international travel, and all but shut down their economies in a desperate effort to slow the outbreak. Under the demands of social distancing, the teenage activist Greta Thunberg shifted her swelling climate movement online—where it effectively dropped out of public sight. The UN ultimately canceled this year's COP, killing any last hopes that nations would, as originally intended, adopt more ambitious emissions targets on the fifth anniversary of the deal.

The Paris accords had lifted hopes that after decades of dithering, the world might finally pull together to confront climate change. Nearly every nation signed on, each agreeing to take specific steps to rein in emissions. But what if, in retrospect, Paris was not the start of an era of cooperation, but its high point?

THE NATIONALIST NARRATIVE

As the covid-19 outbreak rages across the world, it's easy to forget about the climate crisis. The priorities right now are, and should be, slowing the pandemic, saving lives, and then restarting economies left
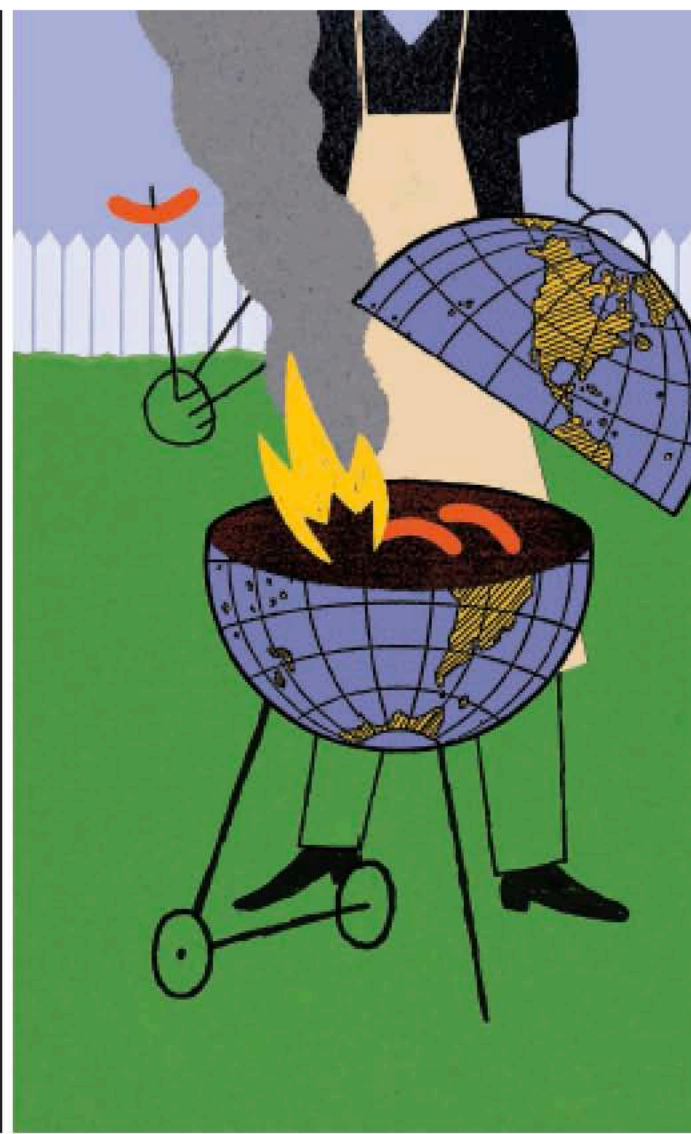
in shambles. But by that point few countries are likely to be able or especially eager to sacrifice near-term growth to help slow global warming.

In the short term, global emissions are falling, as they did during steep economic declines in the past. But carbon dioxide can stay in the atmosphere for centuries, meaning the total concentration will continue to rise even if we're producing less of it. And emissions will bounce back as soon as economies do. They're already nearly within normal ranges in China again.

So the threat of rapidly accelerating climate change will remain. And we'll be living in a much poorer world, with fewer job opportunities, less money to invest in cleaner systems, and deeper fears about our health, our financial futures, and other lurking dangers.

These are ripe conditions to further inflame nationalist instincts, making our global challenges even harder to solve. Indeed, the breakdowns in international (and even intra-national) cooperation as countries race to understand and tackle the covid-19 outbreak offer a stark warning for our climate future.

By its very nature, climate change is a global problem: every country needs to nearly eliminate emissions. But they don't all have the same incentive to do so. Regions like Europe that pumped out huge shares of historic emissions have less to lose by curbing them than nations like India that need faster economic growth to reduce poverty. Those rich countries also

MIGUEL PORLAN

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America might survive coronavirus.
But will the election?

By Patrick Howell O'Neill

VOTE

THEY WERE WAITING FOR THE BIG ONE.

THEN CORONAVIRUS ARRIVED.

LINDA KOZLOWSKI'S NEIGHBOR wanted to know if she needed anything from Walmart. It wasn't a quick trip into town; the drive from the Oregon coast to Portland took two hours. But because of her age, Kozlowski, a 77-year-old retiree, might be at risk from covid-19. Perhaps there would be hard-to-find goods, like hand sanitizer. She thought for a moment and asked for bread, pasta, and toilet paper.

Helping senior citizens is a neighborly thing to do, especially in the middle of a pandemic. But in Manzanita, where

Can being ready for one kind of disaster prepare you for another?

Kozlowski lives, joint grocery runs are part of a detailed disaster preparedness plan that Kozlowski herself introduced to the town 13 years ago. Back then, it wasn't a disease they were concerned about, but a storm that helped locals realize exactly how vulnerable they were to power outages, floods, and landslides.

The Oregon coast is a harsh, unforgiving place where mundane outings can quickly turn deadly. This past January, Jeremy Stiles and his two young children, Lola and William, were swept out to sea

By Britta Lokting

THE VALUE OF A SAVED LIFE

How do we choose between stopping covid and reviving the economy? Fortunately, it doesn't have to be a choice: we can do both.

By David Rotman

In the first employment report after social distancing measures had taken hold in many US states, the Department of Labor announced that 3.3 million people had filed jobless claims. A week later, in the first week in April, an additional 6.6 million claims came in—almost unfathomable compared with the previous record of 695,000, which was set in 1982.

As bad as those numbers are, though, they greatly understate the crisis, since they don't take into account many part-time, self-employed, and gig workers who are also losing their livelihoods. Financial experts predict that US GDP will drop as much as 30% to 50% by summer.

In late March, President Donald Trump warned against letting "the cure be worse than the problem itself" and talked of getting the country back to business by Easter, then just two weeks away. Casey Mulligan, a University of Chicago economist and former member of the president's Council of Economic Advisers, warned that "an optimistic projection" for the cost of closing nonessential businesses until July was almost $10,000 per American household. He told the New York Times that shutting down economic activity to slow the virus would be more damaging than doing nothing at all.

Eventually the White House released models suggesting that letting the virus spread unchecked could kill as many as 2.2 million Americans, in line with the projections of other epidemiologists. Trump backed off his calls for an early reopening, extending guidelines on social distancing through the end of April. But his essential argument remained: that in the coronavirus pandemic, there is an agonizing trade-off between saving the economy and saving lives.

Evidence from research, however, shows that this is a false dichotomy. The best way to limit the economic damage will be to save as many lives as possible.

A novel recession

Part of the difficulty with setting policy now is that

Pandemics through the decades

A viral disease has a way of reminding us that technology can't help us if it's not paired with human cooperation.

June 1955

October 1986

July/August 2003

From "The First Great Epidemic of History": Since the beginning of recorded history the people of this world have been molested by a long series of awesome epidemics, several of which have brought mankind dangerously close to extinction. The worst of them all is generally thought to have been the so-called Black Death, which ravaged the known world during most of the second half of the Fourteenth Century. Even more extensive in scope than the Black Death was an epidemic, or pandemic, which occurred nearly 600 years later. This was the influenza outbreak of the Twentieth Century, which began in Europe in May or June of 1918 and in three waves traveled literally throughout the world. It is probable, in fact, that the total carnage from this recent epidemic exceeded that of the Black Death.

From "Controlling Infectious Diseases": These are global problems transcending political and national boundaries. An infection may come to light anywhere in the world and span continents within days or weeks. Recognizing as much, several expert groups have concluded that a surveillance system to spot emerging infections—an "early warning system"—is an essential first line of defense. But so far we aren't even close to having such a system .... As of now, humanity remains vulnerable to a staggering array of infections. We have no unified system for global surveillance, let alone one for response. Some of us have been paralyzed by complacency—thinking, wrongly, that the threat of infectious diseases is past. Others have become equally paralyzed by defeatism, perhaps feeling that it is too difficult to build the systems needed to protect us. But even imperfect systems are better than none at all.

From "Thinking Like a Virus": Why did it take less than two weeks to find the mutant coronavirus responsible for Severe Acute Respiratory Syndrome, or SARS, while it took the better part of three years to find HIV? There are many reasons—including better technology and a less elusive viral target—but don't discount the unprecedented level of worldwide communication among SARS researchers.

The success of a global research network in identifying the pathogen is an example of the huge payoff that can result when researchers put aside visions of patents and glory for their individual laboratories and let their work behave more like, well, a virus. After all, the hallmark of an opportunistic virus like the one that causes SARS is its ability to spread quickly. Those mounting a response need to disseminate their information and innovation just as rapidly.