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A pandemic like no other

Why has covid-19 been so problematic compared with past pandemics, wonders biologist Jonathan R. Goodman

Was “unprecedented” the most overused word of 2020? There is no doubt that covid-19 has had an extraordinary range of consequences, from turning toilet paper into a treasured commodity and making handshakes taboo to closing schools and putting whole countries in lockdown. But humans have always had to face diseases. Is this one really so different from the others?

As vaccines come into use and we start to see light at the end of the tunnel, it is worth considering this question. There is no doubt that governments, institutions and individuals have made mistakes when trying to deal with covid-19. But perhaps we can be forgiven for some of those failings, because over the past year it has become clear that this disease has unusual attributes. These, combined with certain features of the modern world, may have created the perfect pandemic storm. Whether in our judgements about lockdown and personal risk or in questions about where the virus came from and is going, we really have faced some unprecedented challenges.

The lockdown dilemma

In January 2020, as news emerged that a lockdown had been imposed in Wuhan, China, in an attempt to stop the spread of a new disease, few citizens of other countries could have imagined that their lives would soon be similarly restricted. Quarantine has long been used as a weapon against infectious diseases, from the English village of Eyam’s response to plague in 1665 to action taken in West Africa to curb Ebola outbreaks in the 21st century. However, a year ago, the idea that democratically elected governments would forcibly curtail the freedoms of whole nations seemed unthinkable to many.

A key difference between covid-19 and past outbreaks of infectious diseases is its relatively low mortality rate. No country shuts down its economy when faced with seasonal flu – which is responsible for as many as half a million deaths worldwide every year – but where do you draw the line? SARS-CoV-2, the virus responsible for the
Worldwide, governments that acted early and decisively have generally experienced lower death rates, but the picture isn’t straightforward. Ultimately, we won’t know which strategies worked best until this pandemic is over.

**Varied personal risk**

We can usually identify who might be most at risk from a particular disease and uncover the underlying reasons why. Take the 1918 flu pandemic. Unlike most annual flu outbreaks, it tended to be more deadly in people aged between 20 and 40 than in older people. Two likely reasons have been identified:

- **Circulatory system**
- **Immune system**

Most estimates suggest a mortality rate of about 1 in 100 people, although it has been difficult to pin down, with estimates ranging from 0.5 to 3.5 per cent. Covid-19 is definitely far less deadly than Ebola, though, which without medical intervention kills more than 80 per cent of people who get it. As a result, policymakers haven’t always known how to act.

“It’s very tricky for governments,” says Devi Sridhar, a public health scientist at the University of Edinburgh, UK. “If it were like the MERS or SARS outbreaks of the early 2000s, which killed about 33 per cent and 10 per cent of infected patients respectively, everyone would have pulled out the hammer and shut down. But governments think they’re being judged on their economies, not covid.”

The dilemma is exacerbated by our ability to create models showing what percentage of the population is likely to die if a lockdown isn’t implemented. The question for policymakers then becomes what to do with that information, knowing there will be consequences either way.

“Modelling can be really helpful, but it has no value judgements in it, and the key decisions are political ones, not scientific,” says Sridhar. “New Zealand, under Prime Minister Jacinda Ardern, reacted immediately to treat this like a SARS event. Compare this with Sweden, which attempted to achieve herd immunity by treating coronavirus like flu.”

Unprecedented: Streets in New York, “the city that never sleeps”, were deserted during lockdown
crowded living conditions of young soldiers in the trenches of the first world war, and a level of immunity among older people as a result of contact with previous influenza viruses. The variable severity of covid-19, by contrast, continues to confound us.

Taking a novel approach that uses health analytics to interpret huge English patient data sets, a group called OpenSAFELY has shown that no demographic is at zero risk of dying from covid-19. Many factors have been associated with a higher mortality rate, though. For example, people older than 65 are at a much greater risk than are younger people, men are more likely to die of covid-19 than women and members of ethnic minority groups tend to have much more severe cases. We don’t know why this happens. We also aren’t sure why various underlying conditions, such as lung disorders, coronary heart disease and diabetes, increase an individual’s likelihood of dying from covid-19. And we don’t know why some people get the lasting symptoms of long covid.

There are numerous hypotheses to explain the observed patterns and inconsistencies, but, as an article in *Nature* put it, covid-19 poses a “riddle for the immune system”. We know enough to help us prioritise vaccinations, but despite months of intensive research, we still don’t know why one person infected with SARS-CoV-2 becomes severely ill while another has no symptoms at all.

**Hidden transmission**

In 2012, a new disease, caused by a coronavirus, emerged in Saudi Arabia. That disease, MERS, is far deadlier than covid-19, but also less contagious. This is in large part because people catch MERS from camels, an animal most of us rarely encounter. The new coronavirus, SARS-CoV-2, by contrast, spreads from person to person. The density of human populations and our fondness for intercontinental travel explain why covid-19 swept across the world within months of emerging in China.

There are still uncertainties about how the virus is transmitted, however. The main route is via airborne particles. We know people are more likely to be exposed to these in confined indoor spaces than in wide-open, outdoor ones. But we don’t know how far these aerosols travel, or for how long the virus remains infectious when airborne. There is, furthermore, inconclusive evidence about how great the infection risk is from viral particles on surfaces. Some studies suggest that coronaviruses can remain potentially infectious on plastic for over a week. Others suggest that, even when viral particles are present, surfaces are unlikely to infect someone who interacts with them.

Early on in the pandemic, members of the medical community were divided about whether face masks do any good. Now, we know that wearing one protects both you and others nearby, particularly in indoor spaces. In large part, that is because of another anomalous characteristic of covid-19: you can catch it from someone who has no symptoms.

Research from early on in the pandemic found that up to 75 per cent of infected people show no symptoms. A study from December 2020 suggests that the figure may be far lower. Nevertheless, with other contagious
that were sufficiently different to evade their immune memory. What’s more, the discovery of mutations in SARS-CoV-2 viruses circulating among farmed mink in Denmark – which, in November, resulted in the culling of some 17 million animals – seems to support the hypothesis that the virus evolved because of overcrowded conditions in the market in Wuhan. But we don’t yet have a firm grip on how the various mutations are affecting how transmissible and virulent the virus is, or whether they will influence the efficacy of vaccines – although experts agree the risk of mutation shouldn’t dissuade people from getting vaccinated.

Information overload

In past pandemics, there have always been huge knowledge gaps about the origins and spread of the disease. That is true this time, too. We also face another, paradoxical, problem: information overload. Academic journals have published tens of thousands of papers on the covid-19 pandemic. You would need to read several hundred a day to keep up with the output. Coupled with the biological peculiarities of SARS-CoV-2, this surfeit of research makes the science hard to interpret.

When even the scientists disagree, it is easy for people to peddle misinformation – and hard for governments to create coherent public health plans. No wonder conspiracy theories abound and many governments have failed to communicate effectively with their citizens. As a result, it falls on the public to be discerning about the information they take in, and wary of political motivations behind scientific proclamations. Even as vaccines enter mainstream circulation, we must learn to deal with risk and uncertainty if we are to overcome this very peculiar pandemic.

Mysterious evolution

There is no doubt that this pandemic began in or near Wuhan, China. However, misinformation, conspiracy theories and political agendas are preventing us from determining how the virus first infected people. We do know that local bats carry genetically ancestral forms of SARS-CoV-2. One idea is that someone involved in the wildlife trade or deforestation contracted the virus and brought it into the city. Another is that crowded conditions at Wuhan’s enormous Huanan Seafood Wholesale Market – where both wild and domestic animals are traded – created the perfect evolutionary environment for the virus to adapt to new hosts, including humans.

What happened next is puzzling, too. Evolutionary theory predicts that pathogens transmitted from person to person often become less deadly with time because a disease that kills too rapidly will soon run out of hosts to infect and so peter out. The SARS-CoV-2 genome contains 30,000 bases – the letters of the genetic code – and there have been just a handful of pervasive mutations since the pandemic began, according to Nextstrain, an open-source project tracking genetic changes in the virus as it moves through human populations.

Although SARS-CoV-2 has been exceptionally stable until now, that may be changing. Two recent mutations – one increasingly dominant in the UK, one known as the South African variant – are raising concerns. In addition to those two new variants, a few purported cases of reinfection among humans indicate that people had encountered mutant versions of the virus.