

By Invitation | Responding to covid-19


Nicholas Christakis on fighting covid-19 by truly understanding the virus

The latest coronavirus is different from past ones, so requires a different response



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By Nicholas Christakis

SEVEN VARIETIES of coronavirus

infect humans: four give us the sniffles; one causes a deadly disease smouldering in the Middle East since 2012; and two erupted into full-on pandemics. The first caused SARS and it petered out quickly. The other causes covid-19 and it has hobbled the global economy. Why the difference?

It is not only a matter of the public-health response or governmental incompetence (though that has certainly made things worse). It also has to do with the underlying epidemiology of the pathogen, which we are coming to appreciate, after little more than a half-year of experience. Viewing covid-19 through the lens of SARS can lead to flawed responses. Understanding how the novel coronavirus is different is essential for identifying how society can best confront it.

SARS, caused by the virus known as SARS-CoV-1, appeared in 2002 and spread to 30 countries. But it infected only 8,422 people and killed just 916 before being declared “contained” by the World Health

contained by the World Health Organisation eight months later. On the other hand, the virus behind covid-19, SARS-CoV-2, has infected more than 18m people and killed more than 700,000 so far.

On the surface, the pathogens have several aspects in common, beyond belonging to the same family of coronaviruses (and having genetic sequences that are 79% identical). Both emerged in China in late autumn and were noticed in a place where wild animals and people were in close proximity: a seafood market in Guangdong for SARS, and a similar market in Wuhan for covid-19. Both produced respiratory ailments and could be lethal. Their transmissibility or “reproduction number” (the now famous R_0) was also roughly the same, with each victim infecting about three other people, on average.

But that is where similarities end. The virus behind SARS had intrinsic qualities that made it harder to spread and easier to control, compared to the one behind covid-19, which has overwhelmed the

world. This is because of their respective fatality rates, symptoms, infectious periods and a nuance in their reproduction number. Consider these features in turn, for they explain why covid-19's virus has been so destructive—and how we can best fight it.

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First, fatalities. One way that epidemiologists quantify lethality is the case fatality rate, which is the probability a person will die if they come to medical attention. SARS's case fatality rate was around 11%. Covid-19's is estimated to be in the range of 0.5-1.2%, making it one-tenth as deadly as SARS. This makes it harder to control because there are more walking wounded. SARS did not spread as far because it was, paradoxically, too deadly. (This also

helps explain why the Ebola epidemics that can rapidly claim a terrifying 80-90% of infected people in some African outbreaks eventually wane.)

However, there is another brutal aspect to these numbers. Although the virus behind covid-19 is less deadly than the one that causes SARS in a given single case, that does not mean that it is less dangerous on the whole. On the contrary, it harms many more people. To understand why, consider two pathogens. Imagine that for every 1,000 people, the first pathogen makes 20 seriously ill and

kills two. The case fatality rate is 10%.

Now imagine a second pathogen that again makes 20 seriously ill and kills two for every 1,000 people, but also infects a further 180 people making them mildly or moderately ill, but not killing them. Perhaps some of them are left seriously disabled, too. But the case fatality rate gets calculated as two deaths out of 200, for a mere 1%. The second disease seems much milder. In reality it is far worse: no one would prefer to be in a group facing the second pathogen rather than the first—with ten times as many people infected.

The second situation partly characterises the current pandemic. Covid-19 encompasses a range of severity and a variety of symptoms (affecting not just our respiratory system, but also our gastrointestinal and neurological systems in some cases). Perhaps half of those infected are asymptomatic. Some who fall ill indeed suffer serious long-term health problems. But

because, for many, the symptoms resemble a mild cold, there is a tendency for the public and politicians to take it less seriously. Thus, its protean nature makes controlling it harder.

Making matters worse is that covid-19's virus is transmissible before symptoms appear. In February and March, many governments, offices, and schools in the West advised people to stay home only if they showed signs of obvious illness. These meek instructions were in spite of the fact that public health authorities were warning that asymptomatic carriers were a problem. The head of America's Centres for Disease Control, for instance, said this publicly in mid-February.

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The period between becoming infected with a pathogen and showing symptoms is called the “incubation period.” This ranges from 2 to 14 days for covid-19 (hence the recommended 14-day quarantine period) and is typically 6-7 days. For SARS, the incubation period is 2-7 days.

But the crucial difference between the two pathogens is a related metric called the “latent period”. This is the time between becoming infected and being able to spread the disease to others. The incubation and latent periods are not always the same, a difference known as the “mismatch period”.

When the incubation period is longer than the latent period, asymptomatic carriers abound, as with HIV: an infected person is unaware of it without a blood test. When the latent period is longer, as with smallpox, a person shows symptoms before (or at the same time as) they are infectious: the illness is clearly visible to all. Because the incubation period of

covid-19's virus is generally longer than the latent period, it has been far more devastating than the virus behind SARS. Covid-19 patients take about seven days from infection to show symptoms, but they can spread the disease for 2-4 days before they are symptomatic. In fact, the 1-2 days before symptoms may be when they are most contagious.

Last, the two viruses differ in their transmissibility. Return to the reproduction number of the virus, known as R_0 (pronounced "R-nought"). It quantifies how many new cases spring from each existing case. For example, measles is one of the most infectious diseases known, with an R_0 around 12-18, whereas the seasonal flu ranges from 0.9 to 2.1. The R_0 for SARS-CoV-1 was computed to be in the range of 2.2 to 3.6, and that for SARS-CoV-2 is roughly similar.

Yet the transmissibility of a pathogen need not be the same for every person. The extent of the variation in R_0 across individuals in a population, if any, can be

quantified. And this has subtle but important effects on the course of an epidemic. The greater the variation or dispersion (sometimes quantified with something called the kappa parameter, or K), the more likely an epidemic is to feature both super-spreading events and also dead-end transmission chains. That is, an epidemic where the R_0 is a steady 3 for every person follows a different course from when the R_0 ranges from 0 to 10, even if the average is again 3.

Because the incubation period of covid-19 is generally longer than the latent period, it has been far more devastating than SARS”

If the variation is wide, the risk of an outbreak from a given person is low because most people will not spread it. To illustrate this, say there is a group of 100 people with one super-

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spreader who could pass the disease to 300 people, and the other 99 people are not infectious at all. The average R_0 is 3, but with a wide variation. Allowing a random person from such a group to travel to another place will mean that, 99 out of 100 times, the pathogen will not spread in the new location.

On the other hand, if there is another group of 100 people, each of whom can spread the disease to three people, the average R_0 is again 3, but now there is no variation in infectiousness. Allowing a random person to travel to another location means that the infection will surely commence there too, and continue. Although in both cases the pathogen has the same average R_0 , the fact that the variation of the R_0 is smaller for the second case means that the pathogen is much more likely to seed new infections elsewhere. This makes efforts to stop importation more important.

An epidemic with large variation in the R_0 manifests itself with many super-spreaders and super-spreading events. This is what

happened with SARS. It was estimated that four importations were necessary for one transmission chain to be initiated (the other three would fail to start epidemics and die out). For covid-19, it appears that the variation in R_0 is lower than for SARS, so super-spreading events, though they do occur, are less important than the more frequent, humdrum chains of transmission. Yet again, this makes covid-19 harder to control. Its transmission chains are less likely to be dead ends, which makes the virus easier to spread.

What do the differences between yesteryear's SARS-CoV-1 and the current SARS-CoV-2 tell us about how to respond today? There are critical insights that must be heeded if countries are to protect their people and economies.

The larger fraction of individuals surviving serious infection means that health services should prepare for long-term care, not just build pop-up, refrigerated morgues. The prevalence of mild, flu-like symptoms means that governments, media, businesses and the public

media, businesses and the public should redouble their efforts to promote safety precautions because there will be a natural tendency towards complacency. It's harder to mount a consistent public-health message when the virus itself is so inconsistent in whom it infects, harms and kills.

The fact that infected people can transmit it before they develop symptoms (the mismatch period) means that detection is difficult, so widespread voluntary self-isolation and mandatory quarantine both make sense. The low variance of the reproduction number means that focusing just on super-spreading events is unlikely to be effective in stopping the epidemic. A wider dragnet is required, with more testing and rigorous contact tracing.

Testing needs to be widespread. A thin cotton swab shoved up your nostril beats a thick plastic tube jammed down your trachea”

Most important is the notion of asymptomatic carriers: the huge share of infected people who show no symptoms but are contagious. A consequence is that a testing programme appropriate for SARS, where people come to medical attention when they feel ill and are infectious is unsuited to the circumstances of covid-19. Since we cannot rely on symptoms to identify cases, testing needs to be widespread and the results returned rapidly if not immediately.

After all, a thin cotton swab shoved up your nostril beats a thick plastic tube jammed down your trachea. And though some people resist face masks, perhaps they will come to see that they're better than closing the economy or counting body-bags. Given the epidemiology of the virus, the best response is to do what has been voiced by health officials but not always adhered to in our communities. Until an effective vaccine is developed and becomes widely available, minimise social interactions, keep our physical

distance, implement widespread testing and yes, wear masks.

The distinctive characteristics of the virus behind covid-19 mean it will inexorably infect a large percentage of the world before the pandemic has run its course—an epidemiological parameter known as the “attack rate.” For SARS, the attack rate was infinitesimal: only 8,422 people out of a global population of 6.3bn in 2003, just 0.00013%. For covid-19, at least 40% of the world’s 7.6bn people will probably become infected, with millions of deaths. We have a long and sorrowful way to go. So we had better respond wisely.

Nicholas A. Christakis is a doctor and sociologist at Yale University, researching social networks and epidemiology. He is the author of several books, notably “Apollo’s Arrow: The Profound and Enduring Impact of Coronavirus on the Way We Live”, to be published in October by Little, Brown Spark.