

New model looks at what might happen if SARS-CoV-2 is here to stay



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Most of the optimistic ideas about what to do about SARS-CoV-2 involve engineering the virus' extinction. We could ramp up testing and isolate anyone who's been in contact with an infected individual. We could carefully manage infections to build up herd immunity without exceeding our hospital capacity. Or, in an ideal world, we could develop herd

immunity using an effective vaccine.

Unfortunately, there are reasons to be worried that none of these will work. Tracing the contacts of infected individuals may be impossible with a virus that spreads as easily as SARS-CoV-2. And some of the virus' closest relatives don't build up the long-lasting immune response that's needed for persistent herd immunity. All of which raises a disturbing question: what happens then?

A group of Harvard epidemiologists attempted to answer the question by trying out models that tested the impacts of different assumptions about the virus' behavior and the immune system's response to it. The researchers find that there's a risk that it could become a seasonal menace, and we might have to be socially isolating every winter.

Unwanted family

The study is based on what we know about SARS-CoV-2's closest evolutionary relatives. Coronaviruses are a family-level designation, two steps up from species. One step up from species is genus, and there are four coronavirus genera (alpha, beta, gamma, and delta). SARS-CoV-2 is a member of the beta coronaviruses, a genus that includes subjects of prior pandemic fears like SARS-CoV-1 and MERS. But it also includes two species that are less threatening and more annoying: HCoV-OC43 and HCoV-HKU1, which are collectively the second-most common cause of cold-like symptoms.

The reason these cold viruses cause so much annoyance is because they fail to generate long-term immunity. By a year after infection, people's immune systems seem to have forgotten they've ever seen the virus.

But there are complicated relationships among the responses generated by these betacoronaviruses. SARS-CoV-1 generates a long-lasting immune response, which can include antibodies

that block HCoV-OC43 and HCoV-HKU1, the cold viruses. And, while the immune response to the cold viruses is weak, the antibodies generated against them do react to SARS-CoV-1.

To find out what these interactions might mean, the researchers took test results for these two viruses and built an epidemiological model that matched their prevalence. As expected, the model showed a seasonal pattern of infections, with rates peaking between October and May. The model also suggests that the arrival of fall causes the start of this peak, but its decline is largely driven by the lack of susceptible individuals, as a large chunk of the population has already been infected by spring. In addition, the results indicate that infection by one of the viruses provides a degree of protection against the second, leading to only one being common in most years.

Now, add a pandemic

Layered on top of that this year, we have SARS-CoV-2. The basic properties of the virus—how long it takes to become infections, how long it's infectious for, and so on—were based on the properties we're seeing in various countries. But the key questions here can't be extracted from known data: how long-lasting the immune response is, and whether it provides protection against related coronaviruses. Another open question is whether the virus might display seasonal behavior like we see in its relatives.

So, the researchers simply tried different values for these properties to see what would happen.

In any case where immunity to SARS-CoV-2 isn't permanent, the virus is capable of producing sporadic outbreaks. If the duration of the immunity is less than a year, the outbreaks will be annual. If it's longer, we could see bi-annual outbreaks of COVID-19 cases. The model indicates that we'd need to develop long-term immunity to actually have a chance

of suppressing future outbreaks.

Cross-immunity has some interesting effects. If the cold viruses provide even a weak immunity to SARS-CoV-2 (in the area of 30 percent), it's enough to delay future outbreaks of COVID-19; for example, if SARS-CoV-2 would have its next outbreak in 2022, this cross immunity would push that back to 2023. If SARS-CoV-2 induces immunity to the cold viruses, the impact could be dramatic. A 70 percent cross immunity would be enough to effectively eliminate the circulation of the cold viruses.

Doing the distance

Of course, we're not simply allowing SARS-CoV-2 to circulate unimpeded. When the researchers added social-distancing efforts, they saw what had been seen in other models: infections were suppressed, but the virus returned with a vengeance after they were lifted. And, because the isolation is so effective at suppressing the virus' circulation, there was little immunity built up in the population. As a result, the ensuing outbreak is roughly as large as one in which no social distancing is ever attempted. Adding a seasonal influence on the virus' behavior would simply ensure that the post-distancing outbreak would occur in winter.

If they assumed that distancing rules were put in place once infections reached a certain level, the authors model suggested that the current outbreak could last until 2022, with social-distancing rules in place for anywhere between 25 and 75 percent of the time. During these years, we'd see increasing gaps between the times when distancing is enforced, as the percentage of the population with some immunity would steadily rise.

The researchers also examined two things that could reduce the societal impact of the ongoing outbreak: increasing critical care capacity and a partially effective therapy. Either could

have a large impact, as they'd allow us to avoid social distancing for longer without the health care system being overloaded. And that, in turn, would mean we'd tolerate more infected individuals, and the immunity that would ensue would also be a benefit.

While there's a lot of information here, there's a couple of key takeaways. The key ideas for controlling SARS-CoV-2 involves generating herd immunity, either by controlled infections or through a vaccine. But these suppose a long-lasting immunity that is anything but guaranteed. This doesn't mean a vaccine won't work, but it does mean that we may have to plan on annual boosters—maybe it could be rolled into a flu vaccine. In fact, even without a vaccine, the model suggests that SARS-CoV-2 could settle into behavior that resembles our annual flu outbreaks.

The other big takeaway is that, to really understand what's coming next, we need to know how long-lasting the immunity is and whether there's any cross-immunity with the other betacoronavirus strains. Differences in these properties lead to very different behavior on a population level. And that means figuring out the actual values of those properties should be a priority if we're to do intelligent planning.

Of course, like any other model, there are limits to this one. Like all other models in operation now, it depends on our imperfect knowledge of things like the infectivity and frequency of asymptomatic cases. This particular model is also limited by its treatment of the population, which doesn't include any details of the geographic distribution of that population or its modes of interaction.

And, more generally, it's important to emphasize that no single model is ever going to be an exact recapitulation of reality. Instead, these models simply show the rough outlines of what we should expect if a given list of assumptions turns out to be accurate. And, ideally, as more models tackle an

overlapping set of questions, we'll get a stronger consensus about what our near-term future is going to look like.

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