Herd immunity isn't the answer to COVID-19

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As the strain of closed businesses and physical distancing measures continues to mount, everyone is looking for the silver bullet that will put an end to our COVID-19 worries.

Some have suggested letting the disease spread unchecked, until we reach herd immunity: the point at which a disease can't spread through a population because a large enough percentage is immune, either because they've recovered from an infection or received a vaccine. A vaccine for SARS-CoV-2, the virus that causes COVID-19, is still more than a year away, but some individuals, and governments, are hoping that life can return to normal once enough of us have had the disease.

But herd immunity isn't the answer, said Samuel Scarpino, an assistant professor who runs the Emergent Epidemics lab at Northeastern. It won't work.

"They believe that if you just let the wave pass through the population that you'll have 70 or 80 percent of the population infected and you won't have a subsequent wave," Scarpino told an audience of over 200 researchers, students, and others on Thursday in an online seminar as part of a series presented by the University of Maryland's network biology program, in partnership with the University of Vermont's Complex Systems Center.

But those estimates are way too high, Scarpino said. "It's going to be somewhere like 5 to 20 percent, and you're going to have multiple waves of infections because you're still going to have a large fraction of the population susceptible."

The difference between these numbers, Scarpino said, originates with some of the simplifications that epidemiological modelers make to estimate how a disease will spread.

Many models depend heavily on the average number of additional people that an infected person will spread the disease to, a value known as the basic reproductive number or R0. SARS-CoV-2 is estimated to have an R0 of between 2 and 3, meaning each infected person will infect

between two and three others, on average.

This number doesn't account for human behavior. We don't mix randomly with one another—I don't draw a number from a hat to determine my dining companions. We have networks of family members, friends, coworkers we see more frequently, and places where we're more likely to interact with large numbers of people, such as school or church. And these places and patterns are different depending on where we live.

"The social contact networks, the household structures, the demographic patterns, matter immensely when it comes to translating the same reproductive number, the same average number of secondary infections, into an estimate of the total infected population size or percent of the population that's at risk for infection," Scarpino said.

Research has shown that accounting for human behavior lowers the estimated number of infections, Scarpino said. And those predictions seem to do a better job of reflecting reality in areas where large surveys of the infected population have been conducted.

The other problem with the basic reproductive number is that it is an average, Scarpino said. And that average can hide a lot of individual variation, depending on the disease. Both the 1918 flu pandemic and the outbreak of Ebola in West Africa that began in 2013 had an R0 of around two.

"However, 1918 influenza caused one of the most devastating pandemics in history, infecting somewhere around 500 million people, which maybe was around a third of the global population," Scarpino said. Ebola was devastating for West Africa, but did not spread around the world and infected close to 30,000 people.

"They have essentially the same reproductive number, but wildly different outcomes in terms of the public health consequences of these outbreaks," Scarpino said.

The reproductive number is an average. An R0 of two could mean that each person spreads the disease to exactly two people. Or, it could mean that nine out of 10 people don't spread the disease at all, and a tenth person spreads it to 20 people. Researchers call this tenth person a super-spreader.

The 1918 flu pandemic looked like the former situation—each sick person did, in fact, infect about two other people. The Ebola outbreak looked more like the latter—most people didn't spread the disease very far, and a small percentage of the population were super-spreaders. The SARS outbreak in 2003 also relied on super-spreaders.

Diseases that have more variability in the number of infections each sick person causes are more likely to die out on their own by random chance, and are easier to control with public health interventions, Scarpino said.

"One of the biggest pieces of uncertainty, still, for COVID-19 is where it falls in between SARS and Ebola and influenza," Scarpino said. "We know it's not SARS and Ebola, because we were able to stop those diseases with public health measures. We know it's probably not 1918 influenza, because COVID-19 does appear to be more reliant on super-spreading."

If COVID-19 relies on these super-spreading events, then it won't spread evenly through a population, again meaning that there won't be a large enough section of the population infected for herd immunity to work.

It also won't work, Scarpino said, because many people don't want to risk their health or the health of their loved ones. Research has shown that

people started staying home before physical distancing restrictions went into place. And where those measures are being lifted, people have been slow to resume old habits.

"Wuhan is opening up again, but people are still not dining out," Scarpino said. "People aren't just going to go out and get infected. At least, not enough of them to generate herd immunity."

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