

EPIDEMIOLOGY

Why Is Omicron So Contagious?

The new coronavirus variant may be better than other versions at avoiding human immune defenses—but that ability may change in different countries

By Charles Schmidt on December 17, 2021

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A healthcare worker administers a Covid-19 swab test at the Boulder County Fairgrounds testing site in Longmont, Colorado, U.S., on Tuesday, Dec. 14, 2021. Credit: Chet Strange/Bloomberg/Getty Images

The Omicron variant is spreading rapidly worldwide. New travel restrictions and mounting anxiety have followed this heavily mutated version of the coronavirus, which has been reported now in more than 60 countries. In South Africa, where Omicron's 50 or so mutations were first identified, the variant has shown that it can reinfect people who already caught and survived earlier versions of the SARS-CoV-2 virus, as well as people who have been vaccinated against it.

Scientists are now trying to model Omicron's global trajectory, which depends on two factors. One is its innate contagiousness, or transmissibility. The second is its capacity to evade human immune systems. Untangling how much transmissibility and immune evasion each contribute to the variant's spread is "what will allow us to predict how many people Omicron might infect and how fast," says Marc Lipsitch, an epidemiologist at the Harvard T.H. Chan School of Public Health, in Boston.

Transmissibility reflects the virus's ability to replicate in human cells and move from person to person. "It depends on all sorts of biological processes," explains Jeffrey Shaman, an infectious disease modeler at Columbia University's Mailman School of Public Health. "Does it bind more easily to receptors in people's lungs? Do you shed it more efficiently and spew more of it out so you can infect more people?" Immune system evasion, on the other hand, is the capacity of the virus to avoid antibodies that would otherwise mark it for destruction by the body, as well as an ability to dodge various immune system cells.

A key step in gauging a virus's spread is to start with one infected person and estimate how many other people will get the virus from that individual. In an ongoing pandemic, scientists try to capture that estimate with a value called the effective reproduction number, or R_t . The variable "t" represents the number of secondary infections and depends on the effects of other people's immunity, seasonal weather patterns, public health interventions, and other limits on viral transmission. R_t "can change from minute to minute depending on real-world conditions," Lipsitch says. "We use it to determine how fast an outbreak is growing, or shrinking." A value of R_2 , for instance,

means that one person will infect two others while a value of R_5 means the person will spread the virus to five individuals, increasing the number of infected people much faster.

R_t estimates for Omicron are now emerging. On December 9, South Africa's National Institute for Communicable Diseases (NICD) reported that by early November, R_t in that country had stabilized at values below one, signifying cases were actually falling during a period when Delta was the dominant variant and it ran up against widespread immunity in the population. But then R_t shot up suddenly in mid-November. It is now greater than 2 throughout most of the country and exceeds 2.5 in the densely populated province of Gauteng, as well as KwaZulu-Natal and Mpumalanga provinces. NICD scientists calculated the value using laboratory-confirmed cases and hospital admissions data. The R_t in this case includes other variants in addition to Omicron, but the sudden rise indicates that the new variant is in the mix and creating a lot of new infections, according to Carl Pearson, a mathematical modeler at the London School of Hygiene and Tropical Medicine, who works closely with the South African investigators.

Scientists with the United Kingdom's Health Security Agency have since reported an R_t of 3.7 for Omicron itself. That disturbingly high number, presented in a technical briefing released on December 10, is based in part on data showing that Omicron infections in the U.K. are doubling every three days. At that pace, Omicron presents a much larger threat in terms of case counts than Delta, wrote Trevor Bedford, an infectious disease modeler at the Fred Hutchinson Cancer Center, in Seattle, in a detailed series of comments on Twitter.

What's still unknown, Bedford emphasized, is how much of Omicron's rapid rise is because of its intrinsic transmissibility versus its capacity to evade immune defenses. If a given population is broadly immune to other variants, he theorized, then Omicron will spread quickly even if it does not have an inherently superior transmission ability, because people's immune systems will suppress competing variants.

Evidence that Omicron does evade human immune responses is accumulating from different sources. One sign is that it reinfects people who already got the virus. A team led by Juliet Pulliam, an epidemiologist who directs the DST-NRF Center of Excellence in Epidemiological Modelling and Analysis at the University of Stellenbosch, near Cape Town, South Africa, reported on December 2 that more than 35,000 SARS-COV-2 reinfections had occurred in that country among 2.8 million people who tested positive for SARS-COV-2 within the past three months. A different study by a team led by Alex Sigal, a virologist at the Africa Health Research Institute in Durban, South Africa, found that neutralizing antibodies in blood samples from people inoculated with the Pfizer-BioNTech vaccine were roughly 40 times less potent against Omicron than they were against other variants.

But whether Sigal's lab experiments foretell reduced vaccine protection in real life is still uncertain, says Sarah Cobey, an epidemiologist and evolutionary biologist at the University of Chicago. because "immune escape is about much more than neutralizing antibodies." Vaccines also activate specialized immune cells that destroy infected cells, so Omicron would have to avoid them as well. "Immune escape cannot be definitively

measured in a lab,” Cobey says.

During the coming weeks, scientists will assess how Omicron’s R_t values compare in places with different infection histories and vaccination rates. Shaman says it is not clear how much of what’s been observed in South Africa is applicable to other places. For instance, the United States never experienced a wave of infections driven by the Beta variant, while South Africa did, and the exposure to different variants could alter the U.S. population’s immune responses. “We went right from the ancestral Alpha variant to Delta,” he says. “And those sorts of things may change how much of the population is now susceptible to this new Omicron variant. We’ll just have to see how this settles out over time.”