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SOON after vaccination began in many countries, reports of faster-spreading coronavirus variants triggered fears that vaccines might not protect against them. The good news is that initial studies suggest that the existing shots will still work, although they might be slightly less effective against two variants, one that emerged in South Africa and one from Brazil.

“I am optimistic that current vaccines will remain quite useful,” says Jesse Bloom at the Fred Hutchinson Cancer Research Center in Seattle. “But I do expect that eventually it will be necessary to update vaccines to account for viral evolution.”

Antibodies are our main defence against viruses. When we get infected by a new virus, our immune system starts producing a range of antibodies that bind to various parts of viral proteins. Not all antibodies are equal. Studies show that only a few antibodies can “neutralise” viruses and prevent infections. These neutralising antibodies bind to key sites on viral proteins.

For the coronavirus, one such site is the part of the so-called spike protein that binds to receptors on human cells and helps the virus get inside—the receptor binding domain. If this part of the spike protein changes, neutralising antibodies may not bind as well.

A rapidly spreading variant named B.1.1.7, first spotted in the UK, has only one mutation that affects this binding domain. Initial studies of antibodies from those previously infected by the coronavirus or given the Pfizer and BioNTech vaccine show little or no drop in effectiveness against B.1.1.7.

The variant from South Africa, called B.1.351, is of more concern. It has three mutations in the binding domain, including one called E484K. The variant from Brazil, known as P.1, has almost the same three mutations.

According to a computer model, B.1.351’s spread can be explained by this variant being 50 per cent more transmissible or 20 per cent better at evading immunity in previously infected people, when compared with previous variants. Lab studies point to the latter.

Bloom and his team have tested how mutations in the binding domain alter the effectiveness of antibodies from people who have been infected with the coronavirus. Mutations at the E484 site made the biggest difference, with neutralising activity falling as much as tenfold. While that sounds alarming, current vaccines work so well that even a big drop in neutralisation might not substantially reduce protection, says Bloom. The antibodies might not be as effective, but they still get the job done. There were also differences between individuals: antibodies from some worked just as well.

More evidence comes from a study by Rino Rappuoli at GlaxoSmithKline Vaccines in Italy. When his team grew the virus in the presence of antibodies from a previously infected person, E484K was one of three mutations that let the virus become resistant. These findings suggest that the spread of B.1.351 and P.1 is due to the E484K mutation helping the virus evade antibodies and reinfect people who have already had covid-19. “Whether on top of this they are more infectious, I don’t know,” says Rappuoli.

There have been reports of reinfections in South Africa, Salim Abdool Karim, an epidemiologist advising the nation’s government, said in an online presentation. There has also been a report of a woman in Brazil having more severe symptoms the second time round. But such reports are to be expected, said Karim, and in South Africa there is no evidence of a systematic rise in reinfections. This could be because testing how well antibodies neutralise viruses outside the body doesn’t tell the whole story. The so-called T-cell response is also important. T-cells spot an infected cell by detecting viral proteins on its surface, and then destroy it before it releases more viruses.

“T-cells can be incredibly valuable at preventing disease,” says Shane Crotty at the La Jolla Institute for Immunology in California. “They can do it so well that the person never gets sick.” Crucially, an effective T-cell
response only requires the recognition of viral proteins, rather than the blocking of their function. This means it is harder for resistance to evolve because no single site is crucial.

The T-cell response to the coronavirus is broad, involving many parts of the spike protein as well as other proteins. “There is no way these variants are escaping T-cell immunity,” says Crotty. Unfortunately, while T-cells can stop people getting symptoms, they cannot prevent infections.

The bottom line is that existing vaccines should still protect against B.1.351 and P.1, but might be slightly less effective. And there is a danger of these variants or others evolving to be much better at evading vaccine protection.

Escape variants
This means we need to step up surveillance so we can spot any such “escape variants” early and have time to update vaccines, says Angela Rasmussen at Georgetown University in Washington DC.

“It is unlikely that, overnight, a variant is going to emerge that is capable of completely evading the vaccine,” she says. “But if we are not looking, then we might not find them until it’s too late.”

Scientists are already looking at how to update the vaccines and it will be relatively easy to update most of them. The main delay could be getting them approved. New Scientist asked regulators in the UK, US and Europe what manufacturers would need to do.

None has yet decided on the process, but some pointed to the updating of seasonal flu vaccines as a possible precedent. Updated flu vaccines don’t have to undergo clinical trials, so the process could be rapid. “I believe it can be done very quickly,” says Rappuoli.

The more infectious coronavirus variant from the UK has gone global, causing fears that it could lead to a new wave of infections and deaths around the world in coming months if not brought under control. That brings new urgency to vaccination efforts.

The B.1.1.7 variant has so far been reported in 55 countries. There is no evidence that it is more deadly, nor that it is yet spreading locally outside Europe and North America. But initial studies suggest that it is around 50 per cent more transmissible.

This means we need to step up surveillance so we can spot any such “escape variants” early and have time to update vaccines, says Angela Rasmussen at Georgetown University in Washington DC.

A simple calculation illustrates why. Suppose 10,000 people are infected in a city and each infects 1.1 other people on average, the low end for the estimated rate of infection in England now. After a month, 16,000 people would have been infected. If the infection fatality rate is 0.8 per cent, as it was in England at the end of the first wave of infections, it would mean 128 deaths.

With a variant that is 50 per cent more deadly, those 16,000 cases would result in 192 deaths. But with a variant that is 50 per cent more transmissible, though no more deadly, there would be 122,000 cases after a month, leading to 976 deaths.

The number of countries with reported cases of the UK variant

The B.1.1.7 variant is now spreading locally in other nations in Europe and in some US states. Given that the US is already hard hit and unlikely to use lockdown-type measures, Angela Rasmussen at Georgetown University in Washington DC says this is a big worry. “When you already have uncontrolled transmission and then you add another variant that is more transmissible, you are going push the healthcare system past its limit,” she says.

Elsewhere in the world, most reported cases of B.1.1.7 are in travellers, says Áine O’Toole at the University of Edinburgh, UK. That means it may not yet be circulating locally and there might be time to keep it out, she says.

Yet many countries may be finding the variant only in travellers because they aren’t doing genetic sequencing for local cases, says O’Toole. Most countries did little sequencing until recently, so B.1.1.7 could be spreading undetected in places.

The spread of the B.1.351 variant from South Africa appears more limited. Though more than a dozen countries have reported cases, it is only known to be spreading locally in Botswana, Zambia and the UK, says O’Toole. The similar P.1 variant that originated in Brazil has only been found in travellers in Japan so far.

These variants might be dominating in South Africa and Brazil because they seem slightly better at evading the immune response in previously infected people and these countries have had high levels of infections, says Rino Rappuoli at GlaxoSmithKline Vaccines in Italy. If so, the variants will have no transmission advantage in countries with low levels of immunity. But this will alter as vaccination ramps up.