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COVID-19

THE EVOLVING VIRUS

Everything you need to know about the new variants

What it means for the roll-out of vaccines

How long before this is just another cold?

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Why it's time to take it seriously

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Time to adapt

As the coronavirus mutates, we will need to adjust our approach to it

JUST one month ago, the world was already struggling to contain the spread of the coronavirus. Now the challenge has become even harder. The emergence of new variants with different properties has changed the rules of engagement.

That the coronavirus should evolve isn't surprising – this is what viruses do. Scientists have been sequencing the genome of the SARS-CoV-2 virus since it began spreading out of Wuhan in China, recording the mutations that naturally accumulate as more and more people become infected and pass it on.

This virus evolves mercifully slowly. Until recently, the genetic changes we saw were of little consequence to us, but that has begun to change.

Now the virus has picked up mutations that allow it to spread more easily and, in some cases, that could help it evade our immune system (see page 8).

A faster-spreading virus leads to more infections, as has been seen

"A virus that can evade our immune system has the potential to reinfect people"

in the UK and several other countries, and thus, inevitably, to more deaths.

An "escape mutant" virus that can evade our immune response, meanwhile, has the potential to reinfect those who have already had covid-19. Such a variant might even lead to the need for tweaks to vaccines or new treatments (see page 10).

The news of these new variants has coincided closely with the widespread and very welcome roll-out of vaccines against covid-19. These vaccines offer us a way out of the pandemic, but we already knew it would be a long road to vaccinating almost the entire adult population of the globe. The recent evolution of the virus shows us just how long and complicated that road could be.

As we try to work out how best to counter these variants, and what tweaks may need to be made to our vaccines, there is really only one thing we know for certain: the only way to stop the virus from evolving is to stop it from spreading. ▮

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Mutant variants

The coronavirus evolves

Several new viral variants pose added threats – how worried should we be?

Graham Lawton

THE rise and spread of new variants of the coronavirus are seen as ushering in a dangerous new phase of the covid-19 pandemic. But from the virus's perspective, nothing has changed. It is just doing what comes naturally to viruses: evolving.

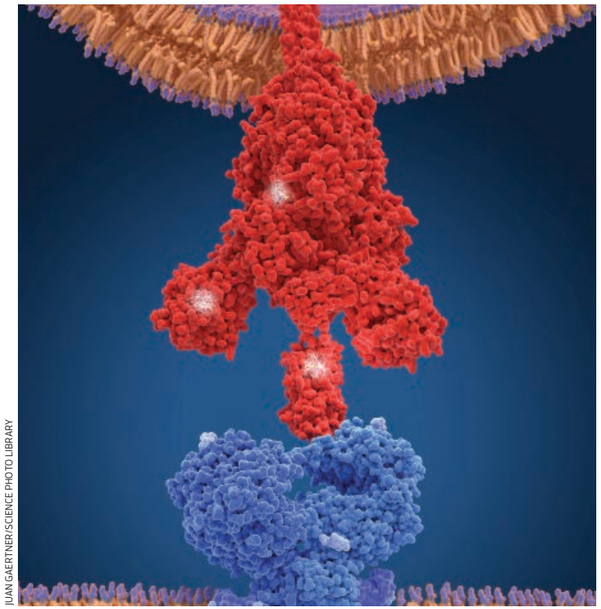
It is now well-established that SARS-CoV-2 is a coronavirus with a large and unusually stable RNA genome, but that doesn't mean it doesn't change at all. Unlike most other RNA viruses, which are among the most mutation-prone biological entities in the world, SARS-CoV-2's genome changes very slowly. This is largely because it has a proofreading function that is efficient at eliminating errors during replication, a major source of the genetic variation that we call evolution.

"There's not masses of evolution occurring, this is a very slow-evolving virus," says David Robertson at the MRC-University of Glasgow Centre for Virus Research in the UK.

A project called Nextstrain, based at the Fred Hutchinson Cancer Research Center in Seattle, compiles all published viral genome sequences and plots them on a family tree. This shows the original virus, called Wuhan-Hu-1, diverging steadily as it spread around the world.

The virus's average mutation rate remains low and steady at about two mutations per lineage per month, but over time this has given rise to thousands of different lineages. For example, there are more than 4000 different versions of the spike protein that the virus uses to break into host cells and which is the target of most vaccines.

Intriguingly, most of the mutations seem to be induced by the human immune system rather than by RNA replication errors.



JUAN GAERTNER/SCIENCE PHOTO LIBRARY

Illustration of the B.1.1.7 coronavirus variant's spike protein (red)

4000

Number of virus variants with different versions of the spike protein for breaking into host cells

10,000

Estimated number of coronavirus genomes sequenced every week in the UK

One arm of our innate immune system is a generalised antiviral weapon that introduces random errors into viral genomes in a bid to neutralise them. It doesn't always succeed.

Most of the surviving mutations are of no medical significance. Up until now, the virus has been circulating unhindered in a large host population with little immunity, and hence has encountered minimal resistance, or selection pressure as evolutionary biologists call it. The evolution that has occurred is therefore mostly just random genetic drift rather than being the virus adapting.

But not entirely. In May 2020, a new variant with a mutation called D614G started circulating. It seems to be slightly more transmissible than the original virus because of an alteration to its spike protein. About 90 per cent of the viruses now circulating worldwide carry this mutation.

More recently, three other mutants, known as the UK, South African and Brazilian variants, have also started spreading rapidly. All are also thought to have mutations that make them more transmissible, and some might be able to outsmart parts

"We are rolling out vaccines to high-risk groups. We may well see a rapid rise in mutations as a result"

of the immune system, although they don't seem to be more deadly.

The sudden appearance of these three new variants doesn't suggest that the virus has upped its mutation rate, says Sudhir Kumar at Temple University in Pennsylvania. They are an inevitable product of time and lots of transmission events between people. Under such circumstances, new variants are bound to arise by chance. Highly transmissible ones have a biological advantage and so will outcompete their more sluggish rivals.

More variants are inevitable. "As the virus mutates, this story will keep repeating itself," says Sharon Peacock, head of the COVID-19 Genomics UK Consortium. The big worry is the emergence of "escape mutations" that enable the virus to dodge the immune system or render vaccines or drugs useless (see page 10).

Such an escape becomes even more likely as we begin to exert selection pressure on the virus in the form of vaccines, natural immunity and drugs. Mutants that can evade these interventions could slip through the net and start circulating wildly, potentially pushing us back towards square one in our efforts to beat the pandemic.

"We are now rolling out vaccination to high-risk groups and this is going to provide a very strong selection pressure," says Emma Thomson at the University of Glasgow. "We may well see a rapid rise in mutations as a result."

We will also have to keep an eye out for viruses that can evade natural immunity, she says. Virologists have already discovered variants that are able to partially evade antibodies.

These are a wake-up call. Even though the UK variant, known as B.1.1.7, doesn't seem to have an escape mutation, the fact that its spike protein is 17 mutations away from the original is "a little bit terrifying", says Robertson.

"It is a concern that a large number of spike mutations are found in the same strain," says Kumar.

One potential danger that we can probably stop worrying about is recombination, which occurs when two related coronaviruses mash their genomes together to create a hybrid. Two studies scouring thousands of viral genomes have found no evidence that this has occurred.

But escape mutation is a real and present danger. A recent case study highlights what could happen once we put the virus under heavy selection pressure. In May 2020, an immunocompromised patient was admitted to a UK hospital with covid-19. He died of the disease in August. Over the 101-day course of his illness, a team led by Ravindra Gupta at the University of Cambridge repeatedly sampled and sequenced viruses from the patient's respiratory tract.

The virus strikes back

The patient was given infusions of an antiviral therapy called convalescent plasma – an antibody-rich blood extract from another person infected with the virus.

Days later, Gupta's team saw a dramatic rise in a mutant version of the coronavirus and later confirmed that it had partially escaped the therapeutic effects of the plasma. This mutant virus eventually killed the patient.

We mustn't draw too many conclusions from this single case, says Gupta. The patient was also being treated for cancer and couldn't mount an effective immune response of his own. But the study shows how quickly and viciously the virus can mutate and escape under selection pressure.

The answer to these threats is surveillance, to flag up and isolate escape mutants before they spiral out of control. The UK's world-class surveillance system relies on a combination of monitoring and sequencing. Red flags are raised if something unusual happens clinically or epidemiologically, and then geneticists search for mutant viruses that could be responsible.

The new UK variant, for example, was spotted because lockdown restrictions were reducing viral spread everywhere but Kent. Surveillance would also

"Even though this virus is evolving slowly, we have to take surveillance very, very seriously"

be triggered if vaccinated people or those who had recovered started falling ill, says Kumar.

About 10,000 genomes a week are sequenced in the UK and there are plans to up that to 20,000 by March. The country also has a new body called the G2P-UK National Virology Consortium to keep track of new mutations and warn about potentially dangerous ones.

"Even though this virus is evolving slowly, we do really have to take surveillance very, very seriously," says Robertson.

What are the new coronavirus variants?

THERE are tens of thousands of variants of the SARS-CoV-2 virus that differ from each other by at least one mutation, according to sequencing studies that track its spread and monitor how it is evolving.

Many of these variants die out, but others spread and acquire further mutations. Overall, though, the coronavirus hasn't changed much. Any two SARS-CoV-2 coronaviruses from anywhere in the world will usually differ by fewer than 30 mutations, and they are all still regarded as one strain.

In early December, scientists looking for reasons for a rapid growth of case numbers in Kent in south-east England, noticed that one variant, now known as B.1.1.7, was spreading faster than others. The evidence that it is more transmissible is growing ever stronger.

This variant is spreading faster than different variants in other regions of the UK and in at least three other countries: Ireland, Denmark and Switzerland. It has reached many other countries, too, but because most countries sequence far fewer samples than the UK or Denmark do, it isn't yet clear whether it is outcompeting other variants in these countries as well.

Initial studies suggest that B.1.1.7 is about 50 per cent more transmissible than other variants. This might not sound like much, but it makes a huge difference over time.

Another new variant, known as B.1.351, was discovered in South Africa after an unusual surge in coronavirus cases beginning in October. It is thought to spread faster too, but there is less evidence than for B.1.1.7.

Why these variants spread faster is unclear (see page 11). B.1.1.7 has 17 defining mutations, and B.1.351 has nine. The overall number of mutations isn't unusual and many of them have been found before.

There has been much focus on the only mutation common to both viruses, known as N501Y. However, this was first seen last April, in Brazil, and a variant with it circulated in Wales for a while, so this alone cannot explain the higher transmissibility.

With many countries now looking for the new variants, reports are emerging of other versions with similar changes. In particular, the P.1 variant found in Brazil has nearly the same three mutations in the spike protein as B.1.351.

Reports of two new variants have also emerged in the US, one of which also has the N501Y mutation, as well as another mutation seen on B.1.1.7. However, it remains unclear if any of these other variants also spread faster.

B.1.1.7 and its ilk will continue to change, so there is a risk they could become even more dangerous. The more people they infect, the more chances there are for these viruses to evolve further.

Michael Le Page

Immunisation

Will vaccines work on new variants?

Some coronavirus variants seem able to partly dodge the immune system, but there is still hope for our vaccines, reports **Michael Le Page**

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 its 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 named E484K as it occurs at a site called E484. The variant from Brazil, known as P.1, has almost the same three mutations.

NELSON ALMEIDA/AFP VIA GETTY IMAGES

Production of the Sinovac vaccine in Brazil, with roll-out imminent

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.

"A mutation in the variants from Brazil and South Africa may help the virus evade antibodies"

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. ▮

International spread

The global threat of the coronavirus variants

Michael Le Page

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.

That is actually a bigger problem than if it were more deadly, says Adam Kucharski at the London School of Hygiene & Tropical Medicine.

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.

55

The number of countries with reported cases of the UK variant

To halt a surge in UK cases partly due to B.1.1.7, England and Scotland this month joined Wales and Northern Ireland in strict lockdown. By the start of this week, all parts of the UK had brought in tougher travel rules.

Last month, Ireland began a strict lockdown after reporting the fastest growth rate of any country in coronavirus cases.

One reason was relaxed restrictions in early December, with pubs and restaurants reopening, says Kingston Mills at Trinity College Dublin. But by last week, nearly half of all new cases were due to B.1.1.7. "I think it was a combination of both," he says.

Efforts are under way to contain a new variant in Pretoria, South Africa

PHILL MAGAKOE/AFP VIA GETTY IMAGES

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 transmitting 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. ▮

The long view

The coronavirus could end up mild like a common cold

Anthony King

POLICY-MAKERS are scrabbling to contain the spread of the coronavirus, as more highly transmissible variants travel around the world. Yet the evolution of SARS-CoV-2 in this way comes as no surprise to virologists. In fact, it is probably just one step on a much longer evolutionary trajectory. In time, virologists predict, the virus will become more benign, following an evolutionary pathway previously taken by four other human coronaviruses that today cause nothing more than the "common cold". How could this happen, and how will our actions play a part?

Coronaviruses tend to evolve slowly compared with other RNA viruses because they proofread their genetic material as they replicate, so can filter out mutations. What's more, SARS-CoV-2 isn't currently under much pressure to change, says virologist Ralph Baric at the University of North Carolina at Chapel Hill. It is successfully colonising a new species – with an open banquet of hosts – and variants that spread faster are outcompeting others.

But evolutionary pressures are starting to kick in. As the virus encounters increasing resistance from antibodies among people who have been infected or vaccinated, new mutations become more likely to take hold. Indeed, some experts suggest that the new variants we currently see arose inside the bodies of people with long-lasting infections.

Lab studies back up this idea. "Some of these variants emerged in vitro when the virus was cultured for several days in the presence of convalescent plasma," says Manuela Sironi, an evolutionary virologist at the Scientific Institute IRCCS Eugenio Medea in Italy.

We don't know exactly what mutations might increase the speed at which the virus can spread. SARS-CoV-2 has four main structural proteins, including the spike protein that sticks out from its surface and helps it attach to cells in the body, as well as non-structural proteins that hijack the machinery inside host cells.

Changes in transmission would probably involve mutations in the spike, which is targeted by the vaccines, says Sironi.

It is impossible to say which mutations would make SARS-CoV-2 more or less deadly. "That is more casino than science at the moment," says Marc Van Ranst at KU Leuven in Belgium. "There are a gazillion possible mutations."

LUIS ALVAREZ/GETTY IMAGES

People at a supermarket in Germany using face masks to protect against covid-19

Familiar trajectory

It is also difficult to predict whether SARS-CoV-2 will evolve to be more harmful, says Sironi. But Van Ranst is optimistic. "Its aim is not to kill us or make us sick," he says. "The virus is successful when it is unnoticed and gets transmitted easily."

Most virologists tend to agree, suspecting that SARS-CoV-2 will follow a similar evolutionary trajectory to the four endemic coronaviruses that cause the "common cold", prosaically called 229E, HKU1, NL63 and OC43.

If so, we, as hosts, will be a crucial driver in this change. The key here is that people never seem to first encounter these endemic coronaviruses as adults. In 2013, scientists at the Chinese Center for Disease Control and Prevention (China CDC) in Beijing measured antibodies for these four common viruses. The type of antibodies generated by a first infection (immunoglobulin M, or IgM) were found only in children. Becoming a "common cold" is as much about us as the virus, says Baric. "My guess is that many of these common coronaviruses, if introduced directly into a very, very naive population of adults, would probably be pretty brutal."

Baric believes that as SARS-CoV-2 bumps into more resistance in adults, it may be pushed to evolve in this direction. "It is possible the virus has to change a little just to maintain itself in children," he says. It may evolve to escape immunity by being able to better replicate in the nose, and so turn into an upper respiratory infection, like the other endemic coronaviruses. These occasionally cause serious disease in children, but usually result in little more than a runny nose. "Children typically have less severe disease than adults," says Baric.

If SARS-CoV-2 follows this pattern, then it should become much less deadly. Other coronavirus infections in healthy adults are usually mild, but reoccur. A 1990 experiment revealed that adults infected with 229E were open to reinfection one year later. The China CDC antibody study also found that 70 per cent of adults had antibodies for the four endemic coronaviruses. Every two to three years, it seems people become more susceptible to these viruses, says Baric. They are re-infected, but retain enough immune memory to fight off severe disease and experience only mild symptoms. Reinfection seems to act as an immune booster.

4

Endemic coronaviruses cause the common cold

"Even without relevant genetic changes, SARS-CoV-2 might eventually turn into the fifth endemic coronavirus," says Sironi.

Recent modelling by epidemiologist Jennie Lavine at Emory University in Atlanta, Georgia, and her colleagues supports this, concluding that once the virus is endemic and first exposure is in childhood, SARS-CoV-2 will be relegated to a common cold. "Primary infections tend to be more severe, especially in older people," says Lavine. "As primary infections increasingly are restricted to children, we expect the disease severity to overall become mild."

Unknown timescale

This has all happened before, according to Van Ranst, who in 2005 reported that OC43 probably jumped to people from cattle and triggered a pandemic in the late 19th century dubbed the Russian flu. The bad news is that we don't know how long it took OC43 to dilute to a common cold virus or when SARS-CoV-2 will join the endemic club. "Our model suggests that the quicker people get exposed, the quicker we get to that mild state," says Lavine. Without vaccines, that would push up deaths.

What's more, endemic coronaviruses can still cause pneumonia in older people. In 2003, when a disease ran rampant in an elderly care home in Canada and killed one in 12 of the residents that it infected, a coronavirus was suspected. It turned out to be OC43. So even a much tamer SARS-CoV-2 may still be a threat to older people for a long time to come. ▮

Covid-19

Why eradication is unlikely

VACCINE roll-out in a growing number of countries should eventually allow life to return to normal, but it is unlikely that we will be able to eradicate the coronavirus that causes covid-19 altogether.

"I don't see that these vaccines will be eliminating SARS-CoV-2 any time in the coming years," says Kingston Mills at Trinity College Dublin.

Despite the many variants, the coronavirus mutates less than many other viruses. "It does not seem to be as mutable a virus as influenza," says Mills. That means we shouldn't need to update vaccines every year, although occasional tweaks might be required.

Despite this, wiping out the virus will be really hard even if we manage to vaccinate most people. To stop a disease spreading, infected individuals must pass it on to less than one other person on average.

Early in the pandemic, infected people were infecting around three others on average, leading to estimates that two out of three people, or 67 per cent, need to be immune to halt transmission. This is what we mean by herd immunity.

Some people now think 70 to 90 per cent of the population may have to be immune to achieve this,

"Even vaccinating everyone on the planet might not stop the coronavirus circulating"

especially with more transmissible variants. This could be hard to do. Some covid-19 vaccines don't reach this level of effectiveness when it comes to preventing disease.

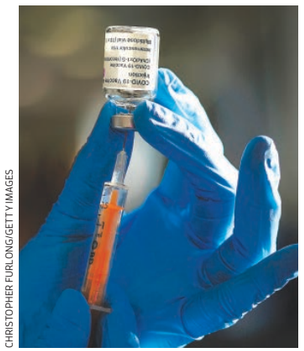
What is more, it isn't yet clear to what extent any of the vaccines prevent transmissible infections, as opposed to merely preventing symptoms, although this is still being investigated.

A few vaccines, such as the one for whooping cough, prevent symptoms, but don't block transmission, says Mills.

This means that viruses – or bacteria in the case of whooping cough – can circulate largely undetected, popping up only when they spread to unvaccinated people and cause disease.

In other words, even vaccinating everyone on the planet might not be enough to stop the coronavirus circulating at low levels, and we are unlikely to get close to this.

In some countries many people say they will refuse the vaccine, such as France, where only 4 in 10 people want it. And no vaccine is yet approved for people aged under 16, who make up a quarter of the world's population.



CHRISTOPHER FURLONG/GETTY IMAGES

While vaccines offer hope, they are no guarantee that the coronavirus will be eradicated

However, we don't have to rely entirely on vaccines to achieve herd immunity. A study by Susan Hopkins at Public Health England and her colleagues suggests that natural infection with the coronavirus provides comparable protection, reducing the risk of reinfection by 83 per cent for at least five months.

Even if we did manage to eradicate the virus in humans, it might lurk in animals and jump back into people later on. SARS-CoV-2 can infect several other species, including cats, dogs, ferrets, bats, hamsters, deers and tree shrews.

"I think this virus is here to stay," says Hopkins, who points out that the smallpox virus is the only one we have managed to eradicate, and that took many years from the start of the campaign to eliminate it. ▮

Michael Le Page