

Coronavirus variants can evade antibodies by spreading via supercells

June 10 2021, by Zania Stamataki



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The antibodies we create after we're infected with a virus or vaccinated against it can be very powerful. A virus typically spreads within our bodies by entering a cell and using it as a factory to create copies of

itself, which then burst out and find new cells to infect. Our antibodies work by binding to the virus and this can block it from attaching to and entering our cells in the first place.

But what happens if a virus does not need to exit the cell in order to spread to neighboring [cells](#)? Can our antibodies be effective against it?

Scientists [recently](#) asked this question for SARS-CoV-2, which causes COVID-19. This highly infectious coronavirus can change [human cells](#), making them fuse with two or more [nearby cells](#). These super-cells, with large merged cell bodies, are excellent viral factories.

The super-cells, known as [syncytia](#), share multiple nuclei (the part of the cell that contains the genetic material) and abundant cytoplasm (the jelly-like substance that surrounds the nucleus). Having more of these components in one giant cell helps the virus replicate more efficiently. And by fusing cells, SARS-CoV-2 increases its resources without being exposed to the neutralizing antibodies that slosh around outside our cells.

The study by Alex Sigal and colleagues tested two coronavirus variants (alpha and beta) for their ability to transmit from cell to cell and investigated whether this mode of transmission was sensitive to antibody neutralization. The alpha variant (first identified in the UK) is sensitive to antibodies, and the beta variant (first identified in South Africa) is less sensitive to these antibodies.

[The Sigal study](#), which is yet to be published in a scientific journal, revealed that cell-to-cell transmission with both variants successfully evaded antibody neutralization. This shows that when the virus takes hold, it will be more difficult to eliminate in cells that can fuse with each other.

We have just posted a new paper in bioRxiv showing that once

cells are infected with SARS-CoV-2, they can infect other cells in ways which are very difficult to inhibit with antibodies <https://t.co/JtTbbwtidR>

— Alex Sigal (@sigallab) [June 2, 2021](#)

Viruses have coexisted with humans and animals for millennia, so they have evolved tricks to avoid being recognized by our [immune system](#). Such an immune evasion strategy is the [direct transmission from cell to cell](#), which doesn't always require cell fusion.

It is also possible for viruses to travel to their next host cells by exploiting tight associations between neighboring cells that shield them from antibodies. It is reasonable to assume that antibodies are most effective at preventing entry into the host cell and less effective in parts of the body where the [infection](#) is already established.

Does this mean that our vaccines will be ineffective against viruses that move directly from cell to cell? Luckily, our immune system has also evolved alongside viruses, and we have learned to [build defenses](#) that work in many ways.

Not the only line of defense

T cells are white blood cells that, following vaccination or infection, are trained to recognize and kill infected cells. They don't rely on recognizing free-floating virus, so cell-to-cell transmission does not reduce their ability to seek and destroy viral factories. Like cells capable of producing antibodies, T cells can remember a previous infection and act rapidly when the same virus comes along again.

It's not wise to put all your eggs in one basket, which is why vaccines induce both antibodies and virus-specific T cells. Antibodies bind to

viruses either before they enter our cells or after the release of new viruses following infection. T cells act to reduce fertile cell hosts for virus replication, until the infection is eliminated. Many other cells (with no immunological memory) also work together to eradicate the virus from the body completely.

What happens in those of us that may have older or dysfunctional parts of our immune system? Coronavirus infection is usually controlled within two weeks in most young, healthy adults and children. In people with dysfunctional T cell responses, cell-to-cell transmission could hinder neutralizing antibodies and hence prolong the infection. Persistent infection increases opportunities for viruses to mutate and better adapt their lifecycle to our bodies, leading to the potential emergence of [variants of concern](#).

We don't need to worry about cell-to-cell transmission disabling our vaccines, but it is important to understand how a virus spreads so that we can target it more effectively. A few years ago [my colleagues and I showed](#) that the hepatitis C virus transmits from cell to cell in the presence of neutralizing [antibodies](#). This hasn't stopped scientists from developing highly successful antivirals that can [cure people](#) who had been infected with hepatitis C for decades.

With effective vaccines and antivirals, we can aim to eradicate viruses that don't integrate their genomes with our own (such as SARS-CoV-2) from human populations as we have done [before](#). Broad resistance to infection in humans achieved by vaccination if we all [work together](#) means that if the same [virus](#) jumps again from animal hosts, its transmission journey in people would be very short. The latest technologies that enable quick vaccine updates can ensure effective control against emerging variants.

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