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EXISTING VACCINES VERSUS NEW COVID VARIANTS

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A health worker collects swab sample for a COVID-19 test in Jammu on April 8, 2023. | Photo Credit: PTI

As COVID-19 cases began rising yet again in India in March, many wondered whether the existing vaccines, based on the SARS-CoV-2 virus that was first reported in China, would still be effective against newer versions of the same virus. To answer this question, we need to look at a few basic principles of immunology.

Vaccines generate a two-pronged immune response. The first is the production of antibodies by B cells, a type of white blood cells. Antibodies directly attack and destroy viruses. The second is the T-cell response. T cells are another type of white blood cells. They have many roles, of which one is to patrol the body and destroy virus-infected cells. Both these arms also give rise to specialised memory cells, which are stored away for future needs.

These two commodities are ‘freshly made’ by our body following an encounter with the antigen, introduced during vaccination. Soon after vaccination, our antibody levels go up. This provides an early window of protection from infection. However, the levels of ‘freshly made’ antibodies start dropping within three months or so, and eventually plateau to a low baseline. This low level is not enough to prevent infection later.

Why do the levels drop? It is natural for the body to scale down the production of antibodies after the immediate threat has passed. If this immune contraction did not occur, our blood would be as thick as grease from all the antibodies produced against every pathogen we have encountered in our lives.

The gradual drop in the level of antibodies is one reason why people sometimes pick up infections despite vaccination — this can occur even after receiving multiple booster doses. For instance, in an early analysis of the XBB.1.16 variant in Pune, India, 26% of the people had already received a third dose.

The other reason is that the virus has altered itself, and some of the older antibodies are not able to lock on to the new targets. It is not feasible to repeatedly give vaccine doses to everyone in the hope that these antibody levels can be kept high. It is hard to imagine taking a vaccine shot every four-six months. Besides, there is evidence of a plateauing of the T-cell immune response after repeated vaccine doses. Recent research on the latest variants such as XBB.1.5 shows almost no neutralising activity in the blood six months after a booster dose.

It is logical to think of updating the first-generation vaccines using parts derived from recent versions of the virus such as Omicron. Unfortunately, the real-world performance of updated bivalent mRNA boosters has not matched the expectations generated by early laboratory studies.

To understand how antibodies work, it helps to look at the barcode on a shampoo bottle at a supermarket. The shampoo is the antigen, while the scanner at the checkout is the antibody. The scanner (antibody) recognises the shampoo (antigen) because of the barcode (epitope). Even a tiny alteration in the barcode would mean that the bottle of shampoo will fail to be scanned or recognised. The virus is constantly altering the epitopes targeted by antibodies. This immune escape is the reason why new versions of Omicron like XBB.1.16 can easily infect someone who was infected not too long ago by earlier versions such as BA.2 and BA.5. Likewise, a T-cell epitope is a specific part (barcode) of the virus protein that is recognised by our T cells. Unlike antibodies that identify large targets, T cells target much smaller epitopes located all over the virus, about 15-20 of them, each one being only eight to 15 amino acids in length. To alter a T-cell epitope is not an easy task for the virus, because there are about 3,800 base pairs on the spike alone, and about 30,000 for the whole virus.

As each T-cell epitope is of short length, mutations created by the virus are far too few to change all the epitopes on it. If the virus was a book, antibodies look at a whole chapter; T cells look at a single sentence. If we change a few words in the book, a few chapters may look different, but single sentences are unlikely to be altered. Besides, individual HLA differences ensure that each person chooses a slightly different set of T-cell epitopes from a large menu provided by the virus. This essentially means that the virus cannot fool everyone all at once.

In other words, the second arm of our immune response remains effective even against an altered form of this virus. It is worth noting that T cells do not stop infection; they can only work on viruses that have already entered the cells. Their job is to search, find and destroy virus-infected cells, each one of which will otherwise release hundreds or thousands of virus copies. By doing this, the spread of the virus within the body can be halted an organ damage can be limited. As a result, vaccinated people are less likely to die even if they got infected afterwards. Recent research has confirmed that T-memory cells remain active beyond two years; the upper limit is not known yet. They will rise to the occasion if and when future infections occur. An easy way to remember this basic immunology is that antibodies are effective before the virus enters our cells, while T cells come to our rescue after the virus infects our cells. To simplify this further, so far the virus has been able to trick the first arm of our immune response (antibodies), but not the second.

A healthy person who has received a full dose of the vaccine will have long-term memory generated in both the T-cell and B-cell compartments. Thus, it does not seem necessary or feasible to make a vaccine for every new sub-lineage that comes along.

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