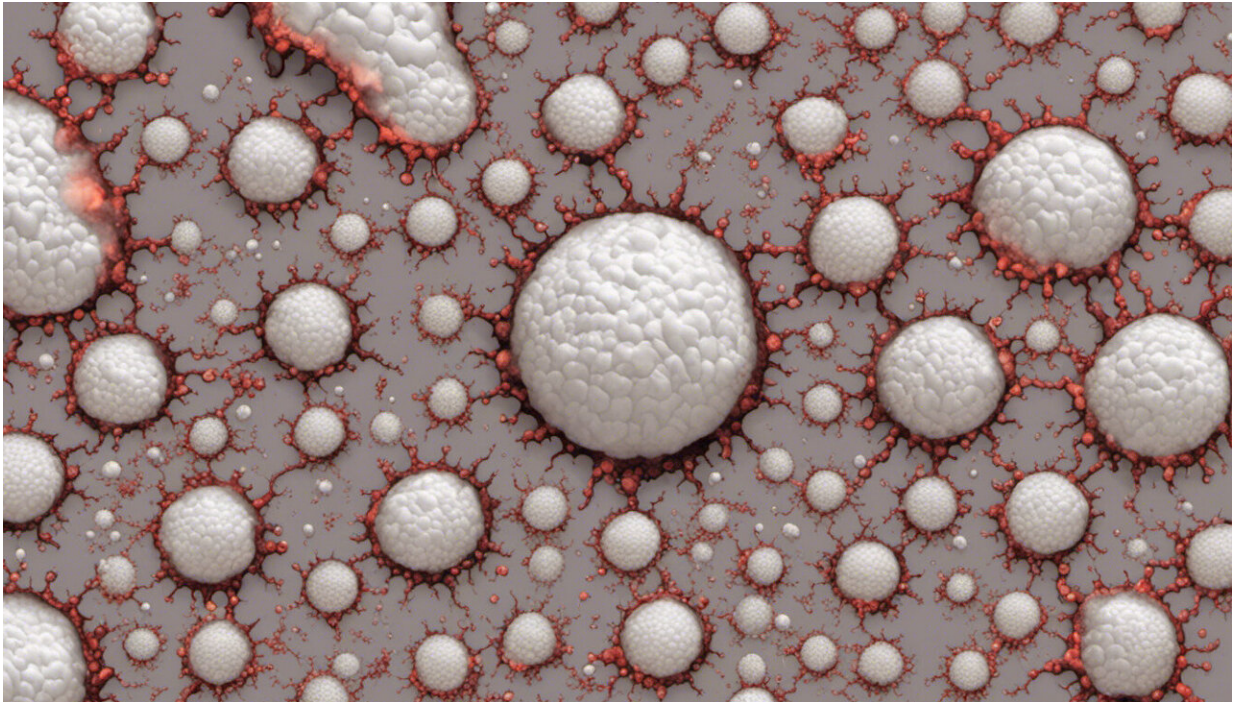


# Coronavirus: B cells and T cells explained

July 20 2020, by Raj Thaker

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Credit: AI-generated image ([disclaimer](#))

To get the upper hand on the coronavirus, we first need to understand how our immune system reacts to it. Understanding this will lead to better treatments, effective vaccines and knowing how near we are to herd immunity—and if it's even achievable.

Every day, new research adds to this knowledge and is widely reported in the media. To follow the discussion, you need to know about two very

important [cells](#): B cells and T cells. Here is a quick primer to get you up to speed.

The immune system is a network of intricately connected cells to protect the body from internal and external threats. It is broadly classified into two sub-types: innate (or natural) and adaptive (or acquired). The key differences between the two are the specificity and agility of the responses generated towards a perceived threat.

The innate system is the first line of defense, capable of detecting many common infectious agents, such as viruses and bacteria, as soon as they find their way into the body. Although it may respond quickly, the innate system cannot always eliminate infectious organisms and it doesn't recognize all the pathogens.

Because of the intricate nature of the immune system, the innate system also provides cues in the forms of chemical signals (cytokines) or degraded products of infectious organisms (antigens) to activate the adaptive immune system, using a process known as "antigen presentation." Without these cues, the adaptive immune system cannot be activated.

The adaptive immune system has evolved to provide a more versatile and highly target-specific defense with an ability to distinguish very subtle differences in the make-up of infectious agents. But the adaptive immune system is slow and can take several days before two key cell types—B cells and T cells—are brought into play.

T cells are further grouped into two sub-types, CD4+ and CD8+ cells. CD4+ are helper T cells that help the activity of other immune cells by releasing cytokines. The cytokines prime the maturation of B cells, which become plasma cells and produce [antibodies](#) to neutralize the pathogen. CD8+ cytotoxic T cells, on the other hand, directly kill

infected cells.

Once the adaptive immune system has vanquished the invader, a pool of long-lived memory T and B cells are made. These memory lymphocytes remain dormant until the next time they encounter the same pathogen. This time, though, they produce a much faster and stronger immune reaction. Memory is the key feature of the adaptive immune system, enabling long-term protection.

## **T cells and B cells in COVID-19**

Since most people have not been exposed to the novel coronavirus, it can safely be assumed that uninfected people have no memory T and B cells and therefore no protection from a COVID-19 infection. Technically speaking, as with any other infection, COVID-19 should generate an immune response, priming the proliferation of anti-COVID T and B cells.

Around [8.3 million people have recovered](#) from COVID, yet evidence of exactly how the [adaptive immune system](#) responds to the novel coronavirus has, so far, been scarce. But new information is emerging all the time.

A recent [study from the US](#) showed that infected people are able to generate COVID-specific T cells and B cells. This study also showed that even some uninfected people had T cells to COVID-19, suggesting an overlap with the response to previous coronavirus infections—so-called cross-reactivity. (Coronaviruses also cause SARS, Mers and some cases of the common cold.)

Also, recent research from the [Karolinska Institute](#) in Sweden showed that several COVID patients with mild to no symptoms had generated T cells against the virus. This was even the case in patients who had no

detectable levels of antibodies against the virus. More importantly, the researchers also found evidence of memory T cells in convalescent patients. This suggests that COVID elicits a robust memory T cell response, which could prevent recurrent episodes of severe COVID.

## **Disappearing antibodies**

How long antibodies stick around for varies from one pathogen to another. For example, [we know that](#) antibodies to other coronaviruses diminish over time (12 to 52 weeks from the time of infection). Some [studies suggest](#) that COVID-19 antibodies can be detected for seven weeks in recovered patients. But given the huge variability of symptoms and immune responses among patients, the precise timeline is unclear.

Another [recent study](#) comparing groups of symptomatic with asymptomatic people showed that asymptomatic people had much lower antibody levels. And follow-up monitoring showed that about 40% of asymptomatic people had no detectable antibodies after eight weeks.

This suggests that antibodies to COVID may not last very long. But this does not exclude the existence of memory T and B cells, capable of re-emerging from their dormant states to protect against re-infection. In other words, the antibodies that B cells make during initial exposure disappear in a few weeks, but the memory cells generated as a consequence of this persist for much longer.

But there is still a lot we don't know. And without a deep understanding of the [immune system](#) role in COVID, designing effective therapies is going to be difficult.

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