Immune System Damage From COVID-19 Is Different From HIV/AIDS — But the Advocacy Has Parallels

Some advocates have called COVID-19 “airborne AIDS,” but this is a misnomer.

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When the HIV/AIDS epidemic first hit, health professionals were baffled when apparently young and healthy individuals had Kaposi sarcoma, a type of tumor typically only seen in severely immunocompromised patients. Scientists eventually discovered the reason: lymphopenia from HIV/AIDS.

Lymphopenia is a health condition in which people have low white blood cell [lymphocyte] count in their blood. Without these cells, the body is unable to defend itself effectively against pathogens. Lymphopenia is one type of way your body can be immunocompromised; it can be caused by many things, including infections, inherited conditions, autoimmune disorders, and nutritional deficiencies.

Earlier in the pandemic, scientists learned that some patients can develop lymphopenia from SARS-CoV-2 infection — but there’s been little research into it since. Although this condition is more rarely seen in COVID-19 as compared to HIV/AIDS, some advocates have suggested the two diseases are similar in their damage to the immune system.

[Editor’s note: AIDS, or advanced HIV disease, is specifically characterized by deficiency of CD4 helper T cells, one type of lymphocyte, which can lead to opportunistic illnesses. SARS-CoV-2 has been linked to changes in various types of lymphocytes, with inconsistent results across studies.]

Lymphopenia can be a serious health condition among the many other serious health complications that patients can get from COVID-19, such as an increased risk of stroke, heart attack, autoimmune disease, and cancer. While we don’t currently know how many people get persistent lymphopenia from COVID-19 or exactly how it happens, the research so far demonstrates that this is an important risk worth reducing through public health measures such as cleaning indoor air, wearing high-quality masks, and staying up-to-date on vaccines.

Unfortunately, there is still limited research about lymphopenia from COVID-19. One 2021 study found about 7% incidence, while another found 12%. Some of these patients recover from it.

But the number of patients with persistent lymphopenia isn't known.

For example, the second paper found 14% of COVID-19 patients had persistent lymphopenia. But upon closer examination, this isn't a reliable finding, as most of the study's original participants dropped out. The available public health data on lymphopenia from SARS-CoV-2 infection seems to have stalled since 2021. This is a concerning gap in the literature as lymphopenia can be a serious health condition.

With HIV, most people who are infected go on to develop lymphopenia if left untreated. For SARS-CoV-2, however, the situation is different, Dr. Michael Peluso, a long COVID and HIV researcher at the University of California, San Francisco told The Sick Times.

Citing a study he co-authored with other researchers in the National Institutes of Health's RECOVER program, Peluso said that there's no evidence of people with Long COVID developing lymphopenia on a wide scale. With COVID-19, lymphopenia is typically only seen among some people who have severe acute cases, he said — and some of those patients recover from the condition in tandem with recovering from COVID-19.

As for how comparable HIV/AIDS and COVID-19 are, Peluso said, "It's like comparing apples and oranges. Sure, they are both fruit, but no one would say the two are the same thing. Ultimately, these viruses work very differently."

HIV specifically targets and replicates inside of T-cells, a type of immune system cell, which can destroy them and limit their functioning. Meanwhile, SARS-CoV-2 can target every cell with an ACE-2 receptor — which is to say, most cells in the human body. Although theoretically ACE-2 is the receptor that SARS-CoV-2 might use to enter T-cells, T-cells barely express ACE-2, suggesting that the coronavirus might be entering T-cells another way.

Currently, Peluso and a team of researchers are working on a study through PolyBio Research Foundation to determine if SARS-CoV-2 can infect blood precursor cells in the bone marrow.

SARS-CoV-2 also replicates differently than HIV. HIV inserts itself in the T-cell's nucleic genome and uses the T-cell's nucleus to initiate viral replication. The SARS-CoV-2 virus targets and alters the functioning of cellular components such as the mitochondria, which provides the cell with energy. So, the coronavirus could potentially target the functioning of the mitochondria of T-cells.

There are other factors that might lead to lymphopenia from COVID-19, Peluso said. One is that people naturally have different levels of T-cells before they get sick. COVID-19 also activates a persistent inflammatory state, which can suppress immune cells. Additionally, SARS-CoV-2 disrupts the delicate interplay of other viruses already present in the human body, such as Epstein-Barr virus (EBV); this can, in turn, disrupt the immune system.

Lymphopenia is also associated with exhausted T-cells from viral persistence, though it's possible to have an exhausted immune system without meeting the criteria for lymphopenia. An exhausted immune system does not cause lymphopenia. Other health conditions, like chronic infections and

cancer, can lead to an exhausted immune system. Scientists think an exhausted immune system may be a factor behind Long COVID as well as myalgic encephalomyelitis (ME).

T-cells become exhausted because T-cells are only meant to be active for a short amount of time. When active, T-cells use the metabolic pathway glycolysis. This gives T-cells an extra boost of energy in the short term, but the process is inefficient in the long term. When T-cells are forced to be active for a long time, such as when fighting against persistent viral infections or against cancer, it is like asking sprinters to sprint through a marathon. The T-cells become dysfunctional and worse at fighting infection.

The history of HIV/AIDS offers lessons for current organizing to curb the spread of COVID-19 and long COVID. During the emergence of AIDS in the 1980's, public health authorities minimized the disease. News outlets reassured the public that having a "strong immune system" would prevent and stop HIV infection. We now know that is not the case. The vast majority of individuals who were infected would eventually go on to succumb to complications from lymphopenia, since they did not have antiretroviral treatments at that time. Since 1981, more than 42 million people have died of HIV, according to the World Health Organization.

Not only did many authorities misrepresent the threat of infection to these patients in the 1980s, but they minimized their deaths as well. AIDS became known as a disease of marginalized communities, including gay men, drug users, and Haitians. News outlets sensationalized deaths among these vulnerable groups. Similarly, in the past few years. the mainstream media has often minimized deaths from COVID-19 as the "elderly" and "disabled." The reality of long COVID is also often erased or minimized.

We have learned from both HIV/AIDS and COVID-19 that placing the burden of responsibility for a disease on communities that already lack resources is a bad public health policy. All communities, including privileged communities, need to be vigilant in preventing the spread of disease. For example, Ryan White, a white suburban teenager who became infected with HIV through blood transfusions, became a tireless advocate by pushing back against the stigma of HIV.

Understanding the similarities and differences between HIV/AIDS and SARS-CoV-2, COVID-19, and long COVID is vital for informed advocacy. When patients understand what is most critical in terms of risks and prevention of diseases, they can better allocate necessary resources, such as PrEP for HIV prevention or respirators and masks for COVID-19 prevention.

Some COVID-19 and long COVID advocates have called COVID-19 "airborne AIDS" in a well-intentioned attempt to get the public to care about mitigation efforts. While some individuals may get persistent lymphopenia from SARS-CoV-2 infection, it is not a significant risk for most individuals as is the case with HIV.

For that reason, "airborne AIDS" is a misnomer. We wouldn't want to use the stigmatization of one disease in order to move people to care about another disease — and if history has anything to say about it, this isn't an effective tactic. Instead, we should look to destigmatize both diseases, care about people more vulnerable to these diseases, and think of our communities as

interconnected.

After all, people with HIV are at increased risk of severe illness due to COVID-19, especially if they are not being treated for HIV. People with HIV may also have a higher risk of developing long COVID.

If there is just one important lesson to take from the HIV/AIDS epidemic, it is to be brave in the face of indifferent leadership and to become active as an advocate for the health of your community. While the public was up against an indifferent Reagan administration during the AIDS epidemic, there were countless people who rose to the occasion and became advocates for people living with HIV/AIDS, such as Hydeia Broadbent, Simon Nkoli, Ryan White, and the Blood Sisters.

Many COVID-19 advocates face a similar negligence under the Biden and incoming Trump administrations. While harm reduction measures to prevent HIV/AIDS infection such as clean needle exchange, free condoms, and accessible PrEP are specific to the way in which that virus spreads, masking in public and making masks accessible to everyone is a similar concept for an airborne disease like COVID-19.

I'd like to think that if Ryan White were still alive today, he would remind us that you'd be surprised sometimes by who a virus will most impact. Thinking of yourself as an exception might come back to haunt you.

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