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Sars-Cov-2 Kills T-Cells, Just Like HIV Says Wuhan Lab Scientists - GreatGameIndia

6-8 minutes

Is Sars-Cov-2 airborne HIV? Two days ago, an interesting article came out:

This article was not written by a bunch of random scientists, but instead was written by people from the Wuhan Institute of Virology, including the infamous batwoman Shi Zheng-Li. Just keep this in mind. It was originally submitted in Sep 2021 and revised in January 2022, so it does not involve Omicron.

The article is saying the following:

- Many patients who had severe Sars-Cov-2 had “lymphopenia”, that is, depletion of the all important immune T lymphocyte cells

- This depletion was caused by cellular suicide (apoptosis) of T cells after infection
- In experimental setups involving infecting laboratory cell lines of human T cells, Sars-Cov-2 virus was able to penetrate and infect T cells
- This tropism (attraction to) T cells and ability to infect them was UNRELATED to the usual way Sars-Cov-2 infects other cells, such as lung cells, that express ACE2 and TMPRSS2 receptors, because T cells do not have those receptors.
- Infection of T cells occurs via "LFA-1, the protein [that] exclusively expresses in multiple leukocytes"
- It turns out that HIV's gp120 protein is the one that "Activates LFA-1 on CD4 T-Lymphocytes and Increases Cell Susceptibility to LFA-1-Targeting Leukotoxin"
- I would like to remind you that HIV's gp120 protein also was mysteriously transplanted into Sars-Cov-2
- Additionally, gp120 protein is located in the spike protein of Sars-Cov-2, and spike protein is used in all "Covid vaccines".

  So, now we have a full new mystery: Sars-Cov-2

destroys immune T cells just like HIV does, Sars-Cov-2 has a transplanted gp120 HIV insert, and it is that specific gp120 insert that allows HIV to enter lymphocytes via the same LFA-1 receptor!

Let's look at this more closely:

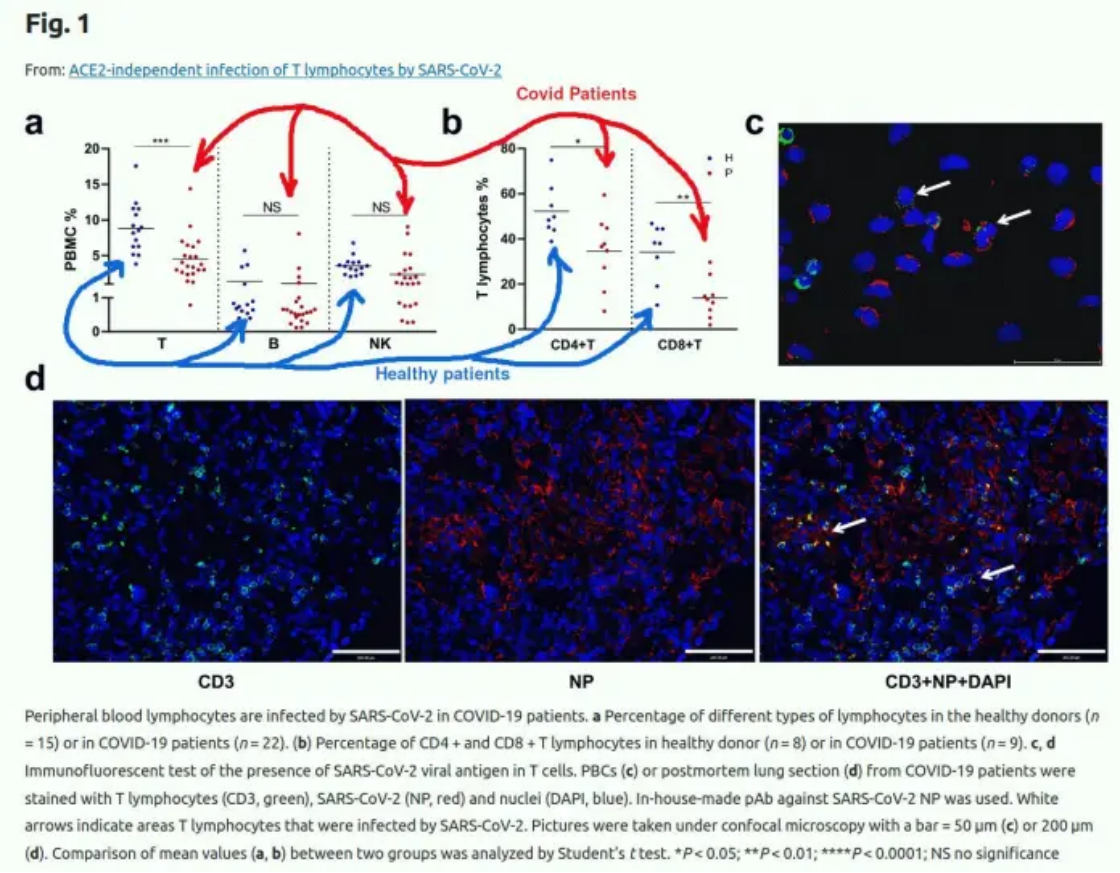
Lymphopenia

T Lymphocytes are cells that are responsible for killing infected or cancerous cells.

> T cells are a type of white blood cell known as a lymphocyte. Lymphocytes protect the body against cancerous cells and cells that have become infected by pathogens, such as bacteria and viruses. T cell lymphocytes develop from stem cells in bone marrow. These immature T cells migrate to the thymus via the blood. The thymus is a lymphatic system gland that functions mainly to promote the development of mature T cells. In fact, the "T " in T cell lymphocyte stands for thymus derived.
>
> T cell lymphocytes are necessary for cell mediated immunity, which is an immune response that involves the activation of immune cells to fight

infection. T cells function to actively destroy infected cells, as well as to signal other immune cells to participate in the immune response.

On this graph from the article, you can see dramatic declines in T cells, and also specific big declines of CD4 and CD8 cells:



For example, the picture above shows that both CD4 and CD8 cells decline.

Then the authors explain how they performed genetic tests to make sure that the T cells actually get infected:

We then analyzed the presence of SARS-CoV-2 viral antigens in PBCs using flow cytometry

or by immunofluorescence assay (IFA). The results suggested that T lymphocytes were infected and in certain patient CD4 + T cells showed a high infection rate (Supplementary Fig. S1a). We also confirmed the presence of viral antigen in T lymphocytes from patient blood by immunofluorescence analysis (IFA) (Fig. 1c). Furthermore, we prepared postmortem lung sections from patients with a fatal infection and analyzed T lymphocytes infiltration and virus infection. We found T lymphocytes infiltration in the lung section, and many T lymphocytes were also positive for SARS-CoV-2 NP staining, indicating virus infection (Fig. 1d). A similar finding has also been reported.[13] Taken together, we showed the presence of SARS-CoV-2 viral antigen in T lymphocytes either in the blood or in the lung section from the COVID-19 patients.

HIV and Sars-Cov-2 Use gp120 to Enter T cells

The primary mechanism of AIDS is depletion of CD4 cells. For Sars-Cov-2, we see depletion of CD4 and CD8 cells as well. Science has long answered how HIV infects T cells (1991):

Mechanism of HIV-1 entry into CD4+ T cells

B S Stein [1], E G Engleman

Affiliations + expand

PMID: 1685857 DOI: 10.1007/978-1-4684-5976-0_6

Abstract

Although the mechanism responsible for HIV-1 entry into susceptible CD4+ T cells is incompletely understood, a number of key components are now known. For example, the tropism of HIV-1 for cells expressing the CD4 membrane glycoprotein reflects the use of this protein as a specific viral receptor to which the HIV-1 gp120 envelope protein binds with high affinity. This binding apparently results in the exposure of hydrophobic domains of the gp41 transmembrane protein to apposing plasma membrane components, resulting in the fusion of viral and plasma membranes to one another which, in turn, releases HIV-1 RNA into the cytosol. This fusion event, which is requisite for viral entry as well as HIV-1 associated syncytia formation, occurs in a pH-independent fashion, but requires antecedent T cell activation. In the absence of T cell stimuli, resting CD4+ cells are resistant to HIV-1 entry, which may explain the observation that at any given time the vast majority of CD4+ T cells in HIV-1 seropositive patients are not infected despite the presence of relatively large quantities of free virus in the blood of such patients. The mechanism of HIV-1 entry into other

CD4+ cell types, such as macrophages and dendritic cells, remains to be determined.

The news here is that **Sars-Cov-2 also infects T cells**, and **Sars-Cov-2 also has the gp120 insert**:

Igor's Newsletter

Covid, Vaccine, HIV and VAIDS -- an Explanation

We know that when fact checkers say something, they sometimes are covering something up. But what? This article attempts to organize what I know so that we avoid misspeaking when talking about complicated matters. I usually do not like editing my articles more than a day after publishing, and my edits would only correct typos, calculation errors or add m...

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22 days ago · 210 likes · 255 comments · Igor Chudov

LFA-1 Receptor

Remember that for the last two years we have heard how Sars-Cov-2 infects cells expressing ACE-2 receptor ad TMPRSS2 protein. Guess what, our T-cells have neither of those!

SARS-CoV-2 infection of T cells is ACE2 and TMPRSS2-independent

It is generally believed that ACE2 is the entry receptor for SARS-CoV-2. However, major cell populations in PBCs express extremely low levels of ACE2, raising the question whether ACE2 also mediates SARS-CoV-2 virus entry of T cells. We first tested whether an ACE2 knockdown could dampen SARS-CoV-2 infection of T cells. The data showed ACE2 was successfully knocked down by ACE2-shRNAs in Caco2 cells. Jurkat T cells do not express detectable ACE2 under either mock or knocked down conditions (Fig. 3a). Correspondingly, ACE2 knockdown resulted in dramatically decreased SARS-CoV-2 infection in Caco2 cells but

not in Jurkat T cells (Fig. 3b). To further confirm this finding, we did ACE2 knocked out in Caco2 and Jurkat cells (Fig. 3c). Similar to ACE2-knockdown cells, viral load decreased in Caco2-ACE2-KO cells but not in Jurkat-ACE2-KO cells (Fig. 3d). These results suggested that SARS-CoV-2-infected T cells in an ACE2-independent manner.

So, how do they get infected? The WIV article that I am discussing, conveniently, found the mechanism: **it is a so called LFA-1 receptor**.

could also reduce the viral load in Jurkat cells (Fig. 5i). Collectively, our results suggested that LFA-1 should be an attachment factor or potential entry molecular for SARS-CoV-2 during its infection in Jurkat cells.

Amazingly enough, if you still believe in coincidences, HIV also uses the same LFA-1 receptor to enter lymphocytes, and uses the same gp120 protein to facilitate the entry.

PLoS One

HIV Envelope gp120 Activates LFA-1 on CD4 T-Lymphocytes and Increases Cell Susceptibility to LFA-1-Targeting Leukotoxin (LtxA)

Catarina E. Hioe,[1,*] Michael Tuen,[1] Gaia Vasiliver-Shamis,[2] Yelina Alvarez,[1] Kathleen C. Prins,[1,2] Sagarika Banerjee,[1] Arthur Nádas,[3] Michael W. Cho,[4] Michael L. Dustin,[2] and Scott C. Kachlany[5,6]

Andrew D. Badley, Editor

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Abstract Go to:▸

The cellular adhesion molecule LFA-1 and its ICAM-1 ligand play an important role in promoting HIV-1 infectivity and transmission. These molecules are present on the envelope of HIV-1 virions and are integral components of the HIV virological synapse. However, cellular activation is required to convert LFA-1 to the active conformation that has high affinity binding for ICAM-1. This study evaluates whether such activation can be induced by HIV itself. The data show that HIV-1 gp120 was sufficient to trigger LFA-1 activation in fully quiescent naïve CD4 T cells in a CD4-dependent manner

Summary

From the articles cited, we can see that

- Covid-19 causes lymphocytopenia (depletion of lymphocytes) in real life patients
- HIV causes depletion of lymphocytes also
- Both Sars-Cov-2 and HIV use the same receptor LFA-1 to enter T cells
- HIV uses gp120 protein to bind to LFA-1 receptor
- Sars-Cov-2 also has gp120 insert as well, mysteriously

And, therefore, the effect of Sars-Cov-2 and HIV on lymphocytes is in many ways similar.

The bats, sitting it Chinese caves a thousand miles from WIV, were clearly very smart when they decided to add gp120 to their natural coronaviruses!

Word of Caution

Before we all get overly excited, please note that this is very much work in progress. **I believe very strongly that most people who have mild Covid do NOT develop permanent lymphopenia.**

How do I know? I am one of them.

My own Test Results

Very conveniently, I had blood tests before, during and after my own Covid. My health care provider lets me see the history of my test results. This became very handy when writing this article.

They show, amazingly, that during Covid I did actually have much reduced lymphocytes, that fortunately recovered. During Covid, which was relatively mild, my lymphocytes dropped almost to the lowest range!

Standard Range	9/24/07	4/2/11	3/30/13	11/17/14	1/19/17	3/9/19	2/29/20	11/18/20	9/18/21
Basophils % %					1.2	0.8		0.3	0.9
Basophils Absolute 0.00 - 0.10 10^3/µL	0.1	0.1	0.1	0.0	0.08	0.05		0.02	0.06
Eosinophils % %	5	6	7	4	5.1	5.0		0.1	5.0
Eosinophils Absolute 0.00 - 0.30 10^3/µL	0.4	0.4	0.4	0.3	0.35	0.33		0.01	0.34
Hematocrit 37.0 - 53.0 %	43.5	50.1	46.6	46.5	45.5	47.9	48.4	52.8	48.8
Hemoglobin 13.0 - 17.0 g/dL	15.2	16.7	15.6	15.6	15.8	16.1	16.3	17.6	16.0
Lymphocytes % %	31	39	35	37	36.4	30.2		17.4	30.5
Lymphocytes Absolute 0.90 - 4.00 10^3/µL	2.8	2.9	2.0	2.5	2.48	1.98		1.20	2.09

Active Covid

Drop during Covid

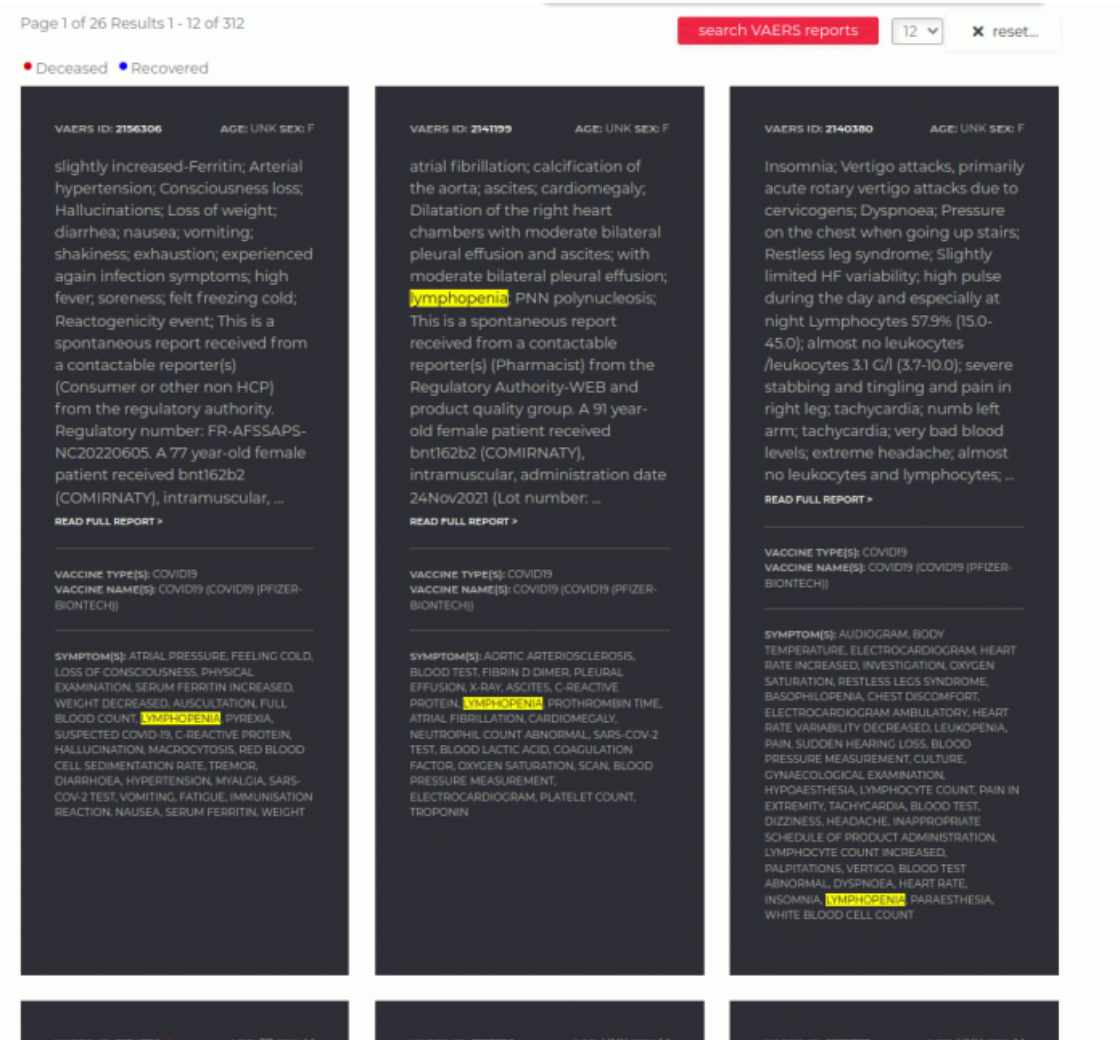
Fortunately, 10 months after infection, they recovered.

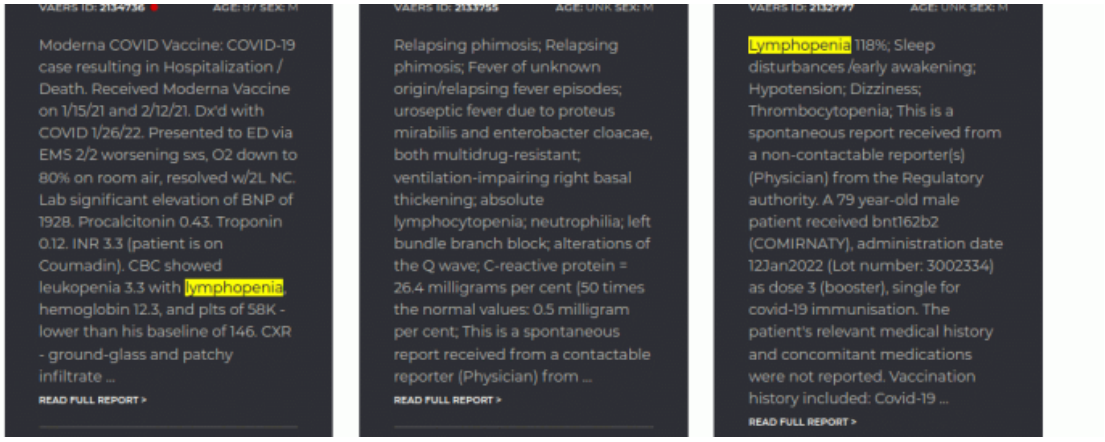
Covid Vaccine Spike Protein Causes Lymphopenia Also

So, spike protein in Sars-Cov-2 has gp120, we know that it affects lymphocytes, and “Covid Vaccines” also make spike protein.

A question arises, **does “Covid Vaxx” also cause lymphopenia**?

A search on OpenVaers reveals 312 reports of lymphopenia, spanning 26 pages:





Obviously, the real number of instances of lymphopenia after vaccination is much higher that openVAERS entries, for many obvious reasons. I will let someone else research that.

Igor Chudov is an author and owner of a popular math website while also running a business. This article was originally published on Substack.

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