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Covid-19 Parallel Infection Cycle

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Abstract
My nights have been rather sleepless since January while I watch the coronavirus decimate people around the world, and currently here in America. As a research scientist, curiosity and a desire to understand what is happening to people led me to develop this biomechanical model of infection. From a purely scientific perspective, this is what my research shows is going on right now before our very eyes.

While reviewing the volumes of research data on Covid-19, I have been conducting my own, independent research of the biomolecular pathways of infection. SARS-CoV-2 is the name of the virus, and Covid-19 is the name of the disease caused by the virus. How could this virus be so stealth, so virulent and so deadly all at the same time? Why is there such a highly skewed mortality curve in contrast to previous pandemics? Why do some people experience complications while others have very mild symptoms? And why do people placed on ventilators die?

My exploration has led me to only one common denominator, solving every variable in this complex Covid-19 equation. That common denominator is called Antibody Dependent Enhancement, or ADE. ADE is a strategy found in certain viruses, such as dengue fever, which explains concisely why the mortality rate increases with age and why children and younger people are largely asymptomatic, or merely exhibit mild symptoms; the elderly typically have more antibodies, at least in this first spring wave.

If my model is accurate, degree of virulence is proportional to quantity of infectious dose a person receives and the quantity of specific antibodies a person has from previous infections. It's not a lowered immune system that results in complications; rather, a competent immune system reacts violently to viral load and secondary bacterial infections. And if my hypothesis is accurate, which I really hope it's not, this fall will be hell, a replay of the fall of 1918, unless a solution is developed and utilized before then. Please prove me wrong.

More particularly, SARS-CoV-2 can exploit specific antibodies so the virus can travel throughout the body essentially unhindered. The more specific antibodies, the faster the unchecked spread and the differentiation between a more classic lung infection vs red blood cell destruction, a process called hemolysis, in addition to lung infection. It is the degree of hemolysis that contributes to the extreme complications and high death.

The ancestor of SARS-CoV-2 is a bat coronavirus, a zoonotic pathogen that cross-vectored into humans. SARS-CoV-2 causes human infection as it were two separate pathogens. In a person without certain specific antibodies, the infection is basically a lung infection with symptoms more

like seasonal flu, with little or no hemolysis. But in stark contrast, if a person possesses the specific antibodies, hemolysis can result and the blood cannot transport sufficient oxygen, in addition to the lung infection. Tissue hypoxia is therefore relative to the degree of hemolysis.

I originally assumed in January that this disparity was due to the fact that the greater the age of a human being, the more antibodies typically exist. However, my update shows that apparently prior to 1960 there was a widespread viral pandemic that stretched at least across North America and Europe. Those exposed to that virus, ostensibly prior to 1960 (with occurrence rapidly diminishing afterwards) possess specific immunological protein tags exploited by SARS-CoV-2. These protein tags serve to differentiate the two separate infection cycles for Covid-19: 1) viral lung infection; and, 2) hemolysis plus viral lung infection.

Red blood cells (RBC) are what transports oxygen throughout the body and gets rid of CO2 in exhaled breath. When the red blood cells are destroyed, iron is released and causes ferritin levels to go up. Malaria parasites also destroy red blood cells. A primary reason why hydroxychloroquine, a selective zinc ionosphore, works well in treating malaria is that by inhibiting zinc excretion and elevating zinc levels, it prevents the parasite from replicating and destroying the red blood cells. Preventing SARS-CoV-2 from replicating and destroying red blood cells is also the main reason why hydroxychloroquine works. A person can't get enough oxygen to breathe without the proper number of functioning red blood cells. But this is only one part of the parallel infection cycle.

The most noticeable part is the other infection cycle that infects the lungs and causes an illness similar to pneumonia. SARS-CoV-2 prevents lung surfactant from being made and so the lungs dry out. Dirt and germs are inhaled with ordinary breathing, and when the lungs dry out these germs can cause lung infections. The immune system then mounts a defense to protect the lungs. The complications seen are typically from what is called a "cytokine storm." A cytokine storm occurs when the body’s immune system goes into overdrive.

In a person with these specific antibodies, SARS-CoV-2 enzymatically splits the heme off of the hemoglobin and then attaches to the remaining porphyrin ring, and uses the porphyrin to gain entry into the cell, a process called porphyrin permeability, to commandeer control of the cell and force it to replicate the virus. A primary cause of death from Covid-19 is due to a significant loss of oxygen in the blood (tissue hypoxia), which can trigger a heart attack. Depending on the number of red blood cells destroyed, a doctor may need to give enough whole blood to restore transport oxygen sufficiently to restore a patient’s oxygen levels.

This blood loss can be likened to a soldier being shot on the battlefield. The bullet causes bleeding, and so blood needs to be transfused after the bleeding is stopped in order to save the life of the soldier.

A whole blood transfusion may be required before being given oxygen to provide enough red blood cells (RBC) for oxygen transport, and it is important that one takes an antiviral, such as hydroxychloroquine, to prevent the newly transfused red blood cells from being destroyed by the

virus.

Studies show that a person is most contagious about two days prior to displaying symptoms until a day after onset of symptoms. Symptoms usually show up about 5 days after exposure, but SARS-CoV-2 has the ability to hide from the immune system in multiple ways. Sometimes it can slowly reproduce without showing any symptoms, sometimes for weeks; much like embers smoldering for days before a fire suddenly erupts. Since the infection can smolder and then suddenly flare, testing only for a SARS-CoV-2 positive doesn't show how bad the infection really is. Instead, a blood test for ferritin levels and RBC count can help the doctor identify how advanced the hemolysis is. Antibody testing could be valuable once tests are properly developed.

What concerns me at the moment is a potential second wave like was seen during the flu pandemic of 1918. There is a high degree of probability that after a summertime lull, SARS-CoV-2 will experience a Gain-of-Function and come roaring back in the fall with increased virulence, attacking those possessing the antibodies from an infection in spring 2020 with a vengeance.

If societies and economies are going to survive this viral onslaught, early treatment options must necessarily focus on: 1) Blocking or inhibiting viral replication; 2) Preventing secondary bacterial infections in the lungs, and; 3) Moderating inflammation and resulting cytokine release.

Hydroxychloroquine is an ionophore, inhibiting zinc excretion and transporting zinc ions across the lipid membrane and into the red blood cells, where it blocks viral replication. It can be an excellent treatment option for those who can tolerate it.

Treatment options for late stage viral infections with hemolysis may need to include whole blood transfusion to provide adequate RBC count for oxygen transport, as well as using a nebulized lung surfactant and High Dose IV Vitamin C to control the dreaded cytokine storm.

Hyperbaric Oxygen Therapy (HBOT) has been shown to provide significant results in both early stage inhibition of disease progression and in late stage treatment to eliminate tissue hypoxia by elevating O2 levels in the body, and thereby prevent death, especially amongst the 50 years and older age groups.

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