How an Arthritis Drug Could Treat Coronavirus Infections

By Alex Berezow, PhD — March 5, 2020

At first glance, rheumatoid arthritis and coronavirus have little in common. But an underlying pathological mechanism that involves an over-reactive immune response may allow a drug developed to treat arthritis to save the lives of coronavirus victims.

Why do microbes kill some people but not others? This is the hardest question in all of medical microbiology. The reason it's so hard is because the answer depends not just on the microbe in question but also on the person.

Sometimes, it's because the microbe is particularly nasty. No matter how healthy a person is, smallpox, HIV, Ebola, cholera, and plague all wreak havoc on the human body.

But that's not the case for all microbes. Billions of people get scratched every year, but only a handful will develop an infection with a flesh-eating bacterium. Millions of people go swimming in lakes every year, but only a handful will become infected with a brain-eating parasite. Why? Is it simply "wrong place, wrong time," or is there a more sophisticated answer?

Increasingly, the latter appears to be true. As a case-in-point, scientists discovered that a single mutation (i.e., a single letter change in our 3-billion-letter genome) may have doubled a person's risk of becoming infected with the 2009 pandemic strain of influenza. In other words, an
individual's genetics -- particularly when it comes to genes involved with the immune system (as was the case in the flu research) -- may be just as much to blame as the microbe's genetics.

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An effective immune response must be of the proper strength and directed at an appropriate target. If these two criteria aren't met, bad things can happen, such as autoimmune disease or a lethal over-reaction to a peanut. The same is true when the immune system targets a dangerous microbe. The response must not be too weak or too strong. If it's too weak, the microbe will overcome the body; if it's too strong, the body can suffer from collateral damage.

One way collateral damage can occur is when the immune system sends out too many pro-inflammatory signals. The immune system is constantly "talking" to other parts of the immune system using chemical messengers known as cytokines. Some encourage inflammation, others discourage it. If too many pro-inflammatory cytokines, such as one called interleukin-6 (IL-6) are released, a "cytokine storm" [3] is said to have developed.

Typically, IL-6 [4] is deployed in order to elicit a quick response to infection and tissue injury. But if too much is produced, the entire body can undergo an inflammatory response, which can be lethal if it leads to multiple organ failure. Ultimately, it's this sort of system-wide immunological overreaction that causes people to die from septic shock. It may also be responsible for some of the deaths due to influenza and SARS.

Something similar may be happening with people who become infected with the Wuhan coronavirus and develop the disease now known as COVID-19. Perhaps they have an immunogenetic profile that, for some reason, overreacts to the virus. Thus, a drug that targets IL-6 could, in theory, help block an overreactive immune response.

And that's precisely what China has just discovered. An antibody drug that blocks IL-6 receptors (called Actemra or tocilizumab) has shown promise in treating patients in Chinese clinics. FiercePharma [5] explains that this drug was approved by the FDA in 2017 to treat cytokine storms and in 2010 to treat rheumatoid arthritis, an autoimmune condition. What causes that? You guessed it: **Too much IL-6** [6].

Let's hope this drug helps save some lives.
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