

Review of: "Factors influencing variable symptoms of COVID-19 patients and proposed revision of public policy for COVID-19 vaccination"

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Potential competing interests: No potential competing interests to declare.

Very nice article however your article is mostly generalized summary of how viral infection and immunity works and has very limited detail on SARS-COV2. I loved the way you catergorized infections in 8 groups and hypothezised the difference on infections on those population. Some thoughts:

In Introduction you mentioned "Thus, underlying diseases and ageing might not per se be considered the key factor explaining the severity and mortality." however in your perspective for group 5 you mentioned "these people should be able to survive spontaneously if they do not have any related underlying diseases that can cause additional critical symptoms, especially during the first couple of weeks of infection." can you please explain further what you actually mean?

We know that genetic mutation can favor or be responsible for severe disease. <u>a study</u> led by scientists at The Rockefeller University and Necker Hospital for Sick Children in Paris concluded that 1% to 5% of critical pneumonia cases set off by COVID-19 could be explained by genetic mutations that reduce the production of type 1 interferons — a system of proteins that help the body's immune system fight off viral infections.

Also, it has been postulated that In the COVID-resistant cells, the receptor was inside the cell, rather than outside, making it impossible for SAR-CoV-2 to attach to it.

Casanova co-leads the <u>COVID Human Genetic Effort</u>, a collaboration of more than 400 researchers and 40 sequencing hubs in 50 countries. The group found that, in some cases, the immune system mistakenly flagged and q attacked proteins called type I interferons (IFNs) that help regulate immune response. Normally, antibodies attach to foreign invaders, marking them for destruction by other immune cells. But the researchers discovered that some people made "auto-antibodies," antibodies against their own type I IFNs. Nearly 20% of the people who died from COVID-19 created auto-antibodies.

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