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Editorial

# Immune Defects may Impair Ability to Fight COVID-19

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## EDITORIAL NOTE

Patients who develop life-threatening forms of COVID-19 have genetic or immunological flaws that impair their ability to fight the virus, research has found. the COVID Human Genetic Effort international consortium labels two malfunctions in severely ill COVID-19 patients that prevent them from making a frontline immune molecule called type 1 interferon. The patients would have approved these malfunctions for years before the pandemic, or in the case of the genetic errors, all their lives. The discovery may help to explain a mystery surrounding the coronavirus: why it leaves some sufferers sick or dying in intensive care, while others continue hardly pretentious or asymptomatic. The consortium sequenced all or part of the genomes of 659 severely ill COVID-19 patients from around the world, as well as those of 534 people with symptomless or mild infection, and originate that the severely ill patients were more likely to carry a type of mutation leaving them unable to make interferon. Though each such mutation is rare, collectively they occurred in 3.5% of severe cases. In the second study, involving nearly 1,000 severe COVID-19 patients, they found at least one in 10 patients carried antibodies to their own interferon, which block its action. No such auto-antibodies were found in asymptomatic or mild patients, and they were detected in only a tiny fraction (0.3%) of healthy controls. These findings were described by one scientist as "astonishing".

Casanova suspects human genetics will end up explanation the majority of such cases, however, because the consortium has only observed for mutations in 13 of the 300-odd type 1 interferon-related genes so far already a huge undertaking. Many other genes, including ones not connected to interferon, could affect a person's response to the virus. Type 1 interferon is a molecule produced by the immune system as soon as it notices infection. It works by stopping a virus from replicating. If this first-line defence is effective, a person may not even feel unwell. Even if it is not, it buys the body time to mount an immune response that is more targeted to the virus in question, involving antibodies and immune cells. Without interferon, severely ill COVID-19 patients rely solely on this second defence mechanism, which may take several days to reach full strength giving the Sars-COV-2 virus a head start on damaging the body's tissues. Casanova said the finding of inborn genetic errors in severe COVID-19 patients originated about serendipitously. The auto-antibody finding is particularly exciting, said the consortium member and infectious diseases of Imperial College London, because 95% of severe patients who carry the auto-antibodies are male and over 50.

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