Risk Factors For Severe or Fatal SARS-CoV-2

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Why is it that some people never developed symptoms from SARS-C0V-2 (COVID-19) while others were sick for days or weeks, or were hospitalized or even died? SARS-CoV-2 is a coronavirus – different than other coronaviruses because it was specifically engineered to be different. The insertion of the spike protein was designed to facilitate easier infection and spread of illness. Another difference was that patients with COVID-19 had increased risk of cardiovascular complications such as myocardial infarction and stroke as compared with other coronaviruses.

But in many ways, SARS-CoV-2 was similar to other coronaviruses in that severity of disease was, in part, based on health status and co-morbidities at the time of infection.

A new study reveals the relationship between specific health issues and outcomes for patients infected with SARS-CoV-2.

Researchers analyzed coronary artery autopsy specimens from eight patients diagnosed with SARS-CoV-2 between May 2020 and May 2021. Average age was 69.6 years, most were male, and all had coronary artery disease, in addition to three or more cardiovascular risk factors such as hypertension, overweight/obesity, and hyperlipidemia.

The researchers found evidence of SARS-CoV-2 replication in all analyzed arteries. But replication was highest in coronary lesions labeled as "pathological intimal thickening" or PIT. PIT lesions are early-stage, and as they progress, foam cells, or fat and cholesterol-filled macrophages, develop and form plaque on artery walls. SARS-CoV-2 showed a stronger affinity for foam cells which was related to the degree of infiltration, or accumulation at the site of injury. The researchers reported that SARS-CoV-2 replicated best in foam cells as compared to other macrophages, at sites of accumulation, and that these cells likely acted as reservoir of SARS-CoV-2 viral debris in atherosclerotic plaque. This then led to the persistence of SARS-CoV-2 in the autopsy specimens.

The researchers further wrote that SARS-CoV-2 infection in macrophages and foam cells promoted an inflammatory response which resulted in the release of cytokines which contributed to further atherosclerosis and an increased risk of events such as myocardial infarction and stroke.[1]

It's been known for a long time that higher dietary fat and cholesterol intake is a risk factor for coronary artery disease and cardiac events.[2] [3] [4] And more and more evidence – this study is not the only one that has looked at this issue – have concluded that

SARS-CoV-2 more negatively impacted people who had coronary artery disease or risk factors for it.[5]

There is a particular dietary pattern that is best for humans, and it's made up of mostly fruits, vegetables, whole grains, and legumes. Adopting a well-structured plant-based diet, and paying attention to issues like hydration, exercise, and weight status, are the keys to longer and better life – AND to much lower risk of severe disease or death from coronaviruses, including SARS-CoV-2.

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[4] Bonomini F, Tengattini S, Fabiano A, Bianchi R, Rezzani R. "Atherosclerosis and oxidative stress." *Histol Histopathol* 2008 Mar;23(3):381-90.

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