

Special Issue

Endothelial Dysfunction in Long COVID

Message from the Guest Editor

Endothelial dysfunction has emerged as a central mechanism contributing to the persistent symptoms and multisystem manifestations of Long COVID (post-acute sequelae of SARS-CoV-2 infection, PASC). The endothelium is a key regulator of vascular tone, coagulation, inflammation, and microcirculation, and even subtle damage can lead to widespread physiological disturbances. Studies on Long COVID have reported elevated von Willebrand factor (vWF) and factor VIII, increased endothelin-1, persistently high D-dimer in some patients, impaired flow-mediated dilation (FMD), presence of fibrin amyloid micro-clots resistant to fibrinolysis, and reduced nitric oxide bioavailability. Together these findings support ongoing endothelial activation and microvascular pathology that may affect the cardiovascular, neurological, and respiratory systems, with mitochondrial dysfunction. In this Special Issue, we will discuss the different mechanisms that may lead to Long COVID—caused by a virus that affects systemic processes, leading to damage and failure of organs and vital systems—and approaches to managing this disease.

Guest Editor

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Editor-in-Chief

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