

The molecular biology of SARS-CoV-2 pathogenesis, host genetics and associated interactions in infection

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Abstract

This new human pathogenic coronavirus belongs to lineage B betacoronaviruses, taking into account its genome. The SARS-CoV-2 genes code for the structural and non-structural proteins used throughout their life cycle and pathogenesis. These proteins govern the functional characteristics of SARS-CoV-2 infection and its association with host proteins in pathogenesis. These open new horizons in the battle virus/host and provide a better understanding of COVID-19 infection. These understandings are revealed by transcriptome-wide and genome-wide analyses showing viral and host crosstalk from viral entry to disease development. It also discloses the acquaintance about antiviral immunity against SARS-CoV-2 and disease severity. The most crucial event is the combat between the virus and the host's antiviral immunity. Based on insight into immunopathogenesis and pathology, potential susceptibility genes are involved in immune dysregulation, auto-inflammation, or autoimmunity mechanisms. This paper addresses host susceptibility, considerations of immune responses, and the use of these prospects for antiviral therapeutic leads. Significant interactions between SARS-CoV infection and host antiviral pathways address innate immune signaling, which can be a crucial genetic determinant in analyzing COVID-19 susceptibility and disease outcomes. The susceptibility of individuals is highly associated with genetics and genetic variants, and their frequencies cause the deviation in the patient vulnerability, which further decides the clinical condition of viral infection in patients. These associations provided relationships among host and viral genes that uncover hidden aspects of giving insights into the vaccine design and antiviral treatment strategies.

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