

SARS-CoV-2 Molecular Biology and Pathogenesis and Therapeutic Options

Kanwal Abbasi*

Pakistan Council of Scientific and Industrial Research (PCSIR), Karachi, Pakistan

*E-mail: kanwalabbasi@yahoo.com

ABSTRACT

The novel coronavirus disease 2019 (COVID-19) emerged in December 2019 in China caused by severe acute respiratory syndrome coronavirus 2. It (SARS-CoV-2) has been declared a pandemic by the World Health Organization (WHO) in March of 2020 (SARS-CoV-2). SARS-CoV-2 is the seventh member of the coronavirus family to affect humans. The present talk highlights the genomic, proteomic, pathogenesis, and therapeutic strategies in SARS-CoV-2 infection. The coronavirus genome is a single-stranded positive-sense RNA (+ssRNA), coronavirus genome is organized by 5'-leader-UTR-replication-ORF1a/b-Spike-envelope-protein-membrane-Nucleocapsid-3' UTR-poly(A) tail with additional genes interspersed in structural genes at the 3' end of the genome. SARS-CoV-2 primarily infects ciliated bronchial epithelial cells and type II pneumocytes, binds to the surface receptor, angiotensin-converting enzyme 2 (ACE2), through spike glycoprotein. Viral entry and cell infection trigger the host's immune response, and the inflammatory cascade is initiated the uncontrolled inflammatory responses characterized by marked pro-inflammatory cytokine release in patients with severe COVID-19, leading to lymphopenia, lymphocyte dysfunction, and granulocyte and monocyte abnormalities. Therapeutic approaches against COVID-19 targeting the specific COVID-19 immune profiles, such as by enhancing lymphocytes or inhibiting inflammation, are promising treatment strategies for severe cases. Enhancing lymphocytes includes NK cell-based therapy, immunomodulators, or convalescent plasma therapy. Though a considerable amount of work has enhanced our knowledge of SARS-CoV pathogenesis and therapeutic treatment design, many questions remain unanswered.

Keywords: Coronavirus, immune response, lymphopenia, RNA, therapeutic approaches.

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