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Application of Disease Progression in Immune Cell Function

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DESCRIPTION

The current COVID-19 pandemic began in Wuhan (China) in December 2019 and quickly spread to become a global sanitary and economic emergency. It is caused by the coronavirus SARS-CoV-2. COVID-19 has a wide range of clinical manifestations, ranging from asymptomatic infection to severe pneumonia with multisystemic failure, which can result in death. The immune response to SARS-CoV-2 is known to involve all immune system components that appear to be responsible for viral elimination and recovery from infection. Nonetheless, such immune responses are thought to be involved in the disease's progression to a more severe and lethal state. This review describes the general aspects of COVID-19 and its etiological agent SARS-CoV-2, emphasising similarities with other severe coronavirus infections such as SARS and MERS, but more importantly, pointing to evidence supporting the hypothesis that the clinical spectrum of COVID-19 is a result of the corresponding variable spectrum of immune responses to the virus. The critical point at which disease progression occurs appears to be the loss of immune regulation between protective and altered responses as a result of inflammatory exacerbation. Finally, it appears possible to identify certain major challenges that merit further investigation in order to better understand COVID-19 immunopathogenesis, thereby assisting in the development of more effective diagnostic, therapeutic, and prophylactic strategies. The purpose of this review is to examine the main aspects of the immune response to SARS-CoV-2, as well as the relationship between the protective and inflammatory responses and the clinical spectrum of COVID-19, which ranges from asymptomatic to severe clinical presentations. The review also emphasises the major immunological research challenges posed by COVID-19 pandemics. The immune response in humans and experimental animals to SARS-CoV and MERS-CoV infection has been extensively studied, and there are numerous excellent reviews. However, due to the similarities between COVID-19 and SARS and MERS, it will be necessary to cite the research done on those infections at certain points. Wu et al. and Zhou et al., who named the virus WH-Human 1 and 2019-nCoV, respectively, identified the virus responsible for the Wuhan outbreak. These scientists also deciphered the virus genome, its origin from bat coronaviruses, and ACE2 as its receptor on host cell membranes. The WHO officially named the infection COVID-19 and the virus SARS-CoV-2 on February 11, 2020. SARS-CoV-2 is a member of the Coronaviridae family, which contains a large number of species capable of infecting a variety of wild animals, some of which also affect humans. Most coronavirus infections in humans result in mild respiratory infections and may be responsible for 20–30% of common colds. SARS-CoV and MERS-CoV, both of which emerged in the last two decades. The three more serious coronaviruses are beta-CoVs, and despite their genomic and structural similarities, they differ significantly epidemiologically. SARS-CoV and MERS-CoV have low transmissibility but high lethality, whereas SARS-CoV-2 has extremely high transmissibility but no global lethality. Coronaviruses have a spheroidal shape, a single-stranded positive RNA of nearly 30 Kbp, and a diameter of 80–120 nm. Inside the virion, their envelope contains the spike –S-, membrane –M-, and envelope –E-, proteins, and the nucleocapsid –N-. The genes for the replicases ORF 1a,b, which occupy two-thirds of the genome and code for the polyproteins pp1a and pp1b, are located on the genome from 5′ to 3′.

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CONFLICT OF INTEREST

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