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Commentary

Development of Protein Targeted Drugs against COVID-19

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DESCRIPTION

The COVID-19 pandemic is caused by the SARS-CoV-2 coronavirus, which is responsible for millions of deaths worldwide. Even with effective vaccines, SARS-CoV-2 will likely persist in the human population due to gaps in efficacy, low vaccination rates, and the emergence of new strains. Understanding how SARS-CoV-2 cause's widespread tissue damage as well as developing targeted pharmacological treatments will therefore is critical in combating this virus and preparing for future outbreaks. We divided the SARS-CoV-2 proteins into three categories: host entry, self-acting, and host-interacting. SARS-CoV-2 proteins, as well as drugs that target the virus and virus-interacting host proteins, and summarize current knowledge on how these proteins promote virus replication and disrupt host systems. Many of these drugs are currently being tested in clinical trials for the treatment of COVID-19, which is encouraging. Future coronavirus outbreaks will almost certainly be caused by new virus strains that evade vaccine protection *via* entry protein mutations.

However, three COVs have recently posed serious threats to global human health: the severe acute respiratory syndrome coronavirus, the Middle East respiratory syndrome coronavirus, and the ongoing severe acute respiratory syndrome coronavirus pandemic. These coronaviruses share many characteristics, but there are significant differences in sequence, structure, and function. We need to identify the sites of virus-host interaction and their impact on host systems in order to understand how these viruses cause disease and to identify the most effective targets for therapeutic intervention. Because viruses rely on host mechanisms for replication, these specializations can also be seen in virus-host interactions, such as various tactics for manipulating host systems for immune evasion. Individual virus proteins have revealed a tremendous amount about specific virus-host interactions, effects on host pathways, and resulting detrimental functional consequences in tissue-specific pathology for Human Immunodeficiency Virus (HIV) (Vpr, Tat, and Nef proteins), influenza virus (M2 and NS1 proteins), and Zika virus (ZIKV) (NS4A protein), to name a few human viruses. These studies have revealed the importance of individual virus proteins to pathogenicity by involving host pathways enlisted to facilitate virus replication and promote virus gene expression, hijacking of host proteasome function, evasion of the host immune response (NF-B; JAK/signal transducer and activator of transcription STAT signalling), the role of host endocytic and secretory trafficking pathways, as well as the contribution of host-modified viruses. More importantly, these findings have laid the groundwork for new therapeutics based on pharmacological inhibitors of specific virus-host interactions. The first of these compounds is currently undergoing clinical trials.

COVID-19, the disease caused by the SARS-CoV-2 infection, is notable for having a negative impact on multiple organs and tissues. In-depth research into how individual virus proteins disrupt host pathways will be critical in identifying the most effective targets for preventing tissue damage and developing inhibitors of these specific virus-host interactions for therapeutic intervention. Virus proteins specialized in virus replication (mRNA transcription, translation, and encapsulation) are highly conserved due to their essential nature, whereas proteins tasked with immune evasion, for example, must constantly adapt to counteract new host tactics. Furthermore, because host systems do not contain homologous proteins, inhibiting virus-specific proteins is less likely to cause unintended side effects. These characteristics, taken together, have piqued interest in these replication-dedicated proteins as potential therapeutic targets.

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