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Variants of SARS-CoV-2, Current Antiviral Drugs, and COVID-19 Therapeutic Implications

Sheryl Lucie*

Department of Medicine & Advanced Technology, University of Helsinki, Finland

Opinion

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***For Correspondence**

Sheryl Lucie, Department of Medicine & Advanced Technology, University of Helsinki, Finland

Email: SherylLucie54@gmail.com

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INTRODUCTION

Coronaviruses can infect a wide range of animals. Two highly contagious and pathogenic coronavirus members have spread in different countries over the last two decades. SARS (severe acute respiratory syndrome coronavirus) first appeared in East Asia in 2002, and Middle East respiratory syndrome coronavirus (MERS) first appeared in the Middle East in 2012. Then, in 2019, a brand-new member of the coronavirus family, acute respiratory syndrome coronavirus 2 (SARS-CoV-2), caused a disease that was later dubbed coronavirus disease 2019 (COVID-19).

The COVID-19 virus was discovered in Wuhan, China, and it fueled a global pandemic that infected billions of people. SARS-CoV-2 is a novel -coronavirus that shares 79% and 50% of its genome sequence with SARS-CoV and MERS-CoV24, respectively. SARS-CoV-2 contains six functional open reading frames (ORFs) arranged in 5′-3′ order: replicase (ORF1a/ORF1b), spike protein (S), envelope protein, membrane protein (M), and nucleocapsid (N). There are also scattered genes encoding accessory proteins among the structural genes, such as ORFs 3, 6, 7a, 7b, 8, and 10. SARS-full-length CoV-2's 29,903 nucleotides encode 27 viral proteins.

DESCRIPTION

Because of the instability of the single-stranded RNA structure and the difficulty in correcting errors during viral replication, RNA viruses tend to mutate more easily. SARS-CoV-2 has undergone multiple mutations as COVID-19 spreads in the human population. Virus mutation is a random process that can occur at any stage and location in the replication process. However, some research has found that mutations in the S protein increase pathogenicity and infectivity. Because of its greater impact on social prevalence, the S protein mutation is considered a variant of concern (VOC). The S protein, as a structural protein of SARS-CoV-2[1-3], primarily mediates the virus's binding to the angiotensin-converting enzyme 2 (ACE2) receptor. A large number of SARS-CoV-2 variants have been discovered to date, with five significant variants receiving extensive attention: Alpha, Beta, Gamma, Delta, and Omicron. Because the mutants' transmissibility and pathogenicity are enhanced to varying degrees, therapeutic strategies for the mutants have been extensively researched.

In this review, we will provide a brief overview of the five variants mentioned above, as well as a summary of current potential therapeutic strategies[4] and molecular targets for these variants. H69/V70 is defined as a deletion of histidine 69 and valine 70 at the NTD site, which can result in significant immune evasion after infection in immunocompromised patients and increase the virus's infectiousness. Interactions between viral mutation sites, as seen in SARS-CoV-2, have the potential to confer transmissibility and virulence to the virus. Some studies have found a superimposed effect between H69/V70 and D614G or N439K mutations, indicating that H69/V70 and D614G mutant strains exhibit faster cell-cell fusion kinetics than wild-type virus strains.

However, because the in vitro experiments did not include a systemic immune barrier, a large number of studies are required to confirm the immune escape caused by H69/V70 deletion. The N501Y mutation is characterized by a tyrosine (Y) substitution of aspartic acid (N) at position 501, with the mutation site primarily located in the S gene's receptor binding motif (RBM) region. As previously stated, S region mutations can increase the affinity of the virus receptor to varying degrees. The virus's binding strength to ACE2 is significantly increased in the N501Y strain, and there is evidence that the affinity of the B.1.1.7 strain for ACE2 is increased by 110%, while the affinity for neutralizing antibodies [5] is only slightly increased. As previously stated, mutations in the S gene predispose the variant to immune evasion and increased affinity for the receptor. As a result, E484K, N501Y, and K417N play an important role in the toxicity of the Beta variants. Deep mutational scans have revealed that E484K increases the ACE2 receptor's binding affinity.

CONCLUSION

Furthermore, RBD is the primary target of plasma antibody neutralisation activity, and studies have shown that the E484K mutation can reduce the efficacy of antibody therapy and lead to immune escape. Furthermore, K417N has been shown to induce a conformational change in the S protein, making the virus difficult to recognise by antibodies and increasing the virus's infectivity.

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