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

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Introduction

Animals of many different species can contract coronaviruses. Over the past 20 years, two extremely contagious and pathogenic coronavirus members have spread throughout several nations. SARS (severe acute respiratory syndrome) and Middle East respiratory syndrome (MERS) coronaviruses initially surfaced in in 2002 and the Middle East in 2012, respectively. A novel coronavirus, acute respiratory syndrome coronavirus 2 (SARS-CoV-2), then produced an illness in 2019 that was later known as coronavirus disease (COVID-19). In, researchers found the COVID-19 virus, which sparked a pandemic that sickened billions of people worldwide. A new coronavirus called SARS-CoV-2 shares 79% and 50% of its genome sequence with the MERS-CoV24 and SARS-CoV, respectively. The replicase (ORF1a/ORF1b), spike protein (S), envelope protein, membrane protein (M), and nucleocapsid are the six functional open reading frames (ORFs) that make up SARS-CoV-2. They are organised in a 5′-3′ order (N). Among the structural genes, there are also a few genes, such as ORFs, that encode auxiliary proteins. The 29,903 nucleotides of the full-length SARS CoV-2 virus code for 27 viral proteins.

Description

RNA viruses have a propensity to mutate more readily due to the instability of the single-stranded RNA structure and the challenge of rectifying mistakes during viral reproduction. As COVID-19 spreads among humans, SARS-CoV-2 has undergone several changes. The random process of virus mutation can happen at any point and place during the replication phase. However, other studies have discovered that the S protein's pathogenicity and infectiousness are increased by mutations. The S protein mutation is a variant of concern due to its increased impact on societal predominance. As a structural protein of SARS-CoV-2 [1-3], the S proteinlargely facilitates the virus's attachment to the ACE2 receptor angiotensin-converting enzyme. There have been several SARS-CoV-2 variants found so far, with the five major variants of Alpha, Beta, Gamma, Delta, and Omicron attracting the most attention. Therapeutic approaches for the mutants have undergone substantial research due to the enhancement of the mutants' transmissibility and pathogenicity to varied degrees.

We will briefly summaries each of the five above-mentioned variants in this review, as well as current prospective therapeutic approaches [4] and molecular targets for these variants is referred to as a deletion of histidine and valine at the NTD site, which can significantly impair immune responses in immunocompromised persons following infection and raise the virus's contagiousness. Viral mutation sites that interact, as in, have the potential to make the virus more contagious and virulent. H69 and mutant strains exhibit faster cell-cell fusion kinetics than wild-type virus strains, according to several investigations that identified a superimposed effect between H69 and or mutations.

To confirm the immunological escape brought on by the H69/V70 deletion, further research is needed because the in vitro experiments lacked a systemic immune barrier. The N501Y mutation, which largely affects the S gene's receptor binding motif region, is defined by the replacement of tyrosine for aspartic acid at position. As was already mentioned, S region alterations can, to varied degrees, improve the affinity of the viral receptor. The N501Y strain greatly increases the virus's affinity for ACE2, and there is evidence that the strain significantly increases its affinity for ACE2 while decreasing its affinity for neutralising antibodies [5] is only marginally elevated. As was already mentioned, the S gene mutation makes the variation more prone to immune evasion and increases its affinity for the receptor. E484K, N501Y, and K417N thereby contribute significantly to the toxicity of the Beta versions. Deep mutational scans have shown that E484K improves the binding affinity of the ACE2 receptor.

Conclusion

The E484K mutation has been demonstrated to decrease the efficacy of antibody treatment and promote immunological escape. RBD is also the main target of plasma antibody neutralisation activity. Additionally, it has been demonstrated that K417N causes the S protein to shift shape, making the virus more infectious and challenging for antibodies to recognise.

References

1. Blustein DL. "Unemployment in the time of COVID-19: A research agenda. " *J Vocat Behav*. (2020); 119:103436.
2. Cucinotta D, Vanelli M. "WHO declares COVID-19 a pandemic. " *Acta Biomed: Atenei Parmensis*. (2020); 91:157.
3. Akkermans J, Richardson J, Kraimer ML. "The Covid-19 crisis as a career shock: Implications for careers and vocational behavior. " *J Vocat Behav*. (2020); 119:103434.
4. Vijayananthan A, Nawawi O. "The importance of Good Clinical Practice guidelines and its role in clinical trials. " *Biomed Imaging Interv J*. (2008); 4.
5. Rennert L. "Modelling the impact of presemester testing on COVID-19 outbreaks in university campuses. " *BMJ open*. (2020); 10:e042578.

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