

## Genomics of SARS-CoV-2

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### ABSTRACT

the etiological representative of the COVID-19 pandemic and changes to evade together host immune systems and intervention strategies. To diminish the short-term and long-term impacts of coronavirus (CoV), CoV differences at the nucleotide and protein level and CoV genomic variation associated with epidemiological variation and geography.

**Keywords:** COVID-19; Genomics; Nucleotide

## INTRODUCTION

SARS-CoV-2 is a member of the Coronavirus family which recently created from the Wuhan province of China and feasted very rapidly through the world infecting more than 4 million people. In the past, other Coronaviruses have also been found to cause human infection, but not as widespread as COVID-19. Since Coronavirus sequences constantly change due to mutation and recombination, it is important to understand the pattern of changes and likely path the virus can take in the future.

Coronaviruses have a single stranded, positive sense, RNA (ssRNA+) genome with a conserved arrangement of genes. Starting from the 5'-end, most of the genome (~21 kb) codes for the non-structural proteins (nsps) 1a and 1b, and these large polyproteins are cleaved by proteases into several mature proteins. The next gene (~3.8 kb) encodes the spike (S) protein. The other structural proteins envelope (E), membrane (M) and nucleocapsid (N) proteins are encoded towards the 3' end of the genome. Additional accessory proteins are also encoded in this region and generally show more divergence between different coronaviruses.

SARS-CoV-2 is a member of the genus Betacoronavirus, subgenus Sarbecovirus of the family Coronaviridae. The genomes of multiple SARS-CoV-2 isolates derived from the first COVID-19 patients in Wuhan, China shared 99.98-99.99% nucleotide uniqueness, coronaviruses that are closely related to SARS-CoV-2. In particular, the virus from the Guangdong pangolins shares 91% nucleotide identity with SARS-CoV-2 and 90.5% identity with RaTG13-CoV. Although, over the whole genome, this Pangolin-CoV is less carefully related to SARS-

CoV-2 than RaTG13-CoV or RmYN02-CoV are, the amino acid sequence of its RBD is much closer to the SARS-CoV-2 RBD than that of any other virus. In particular, within the Guangdong Pangolin-CoV RBD, 5 amino acid residues that are critical for binding to ACE-2 are conserved with SARS-CoV-2, though these are divergent in the RaTG13-CoV and RmYN02 S proteins [2]. When comparing mutation rates, a difference is made between rates calculated as the number of substitutions per nucleotide per cell infection (s/n/c) or substitutions per nucleotide per round of copying (s/n/r). The dissimilarity reflects whether virus genomes replicate via a "stamping machine" model, in which a single template is copied repeatedly, or if replication is semiconservative, in which replicated strands act as templates for additional synthesis. Using the former method, estimated error rates for DNA viruses RNA viruses usually have advanced mutation rates than DNA viruses because they lack a proof-reading activity associated with their RNA-directed RNA polymerases (RdRp) and so have lower fidelity. The few RNA viruses with genomes greater than 20 kb have, however, all acquired a proof-reading activity that correlates with the expression of a viral exonuclease.

## CONCLUSION

If vaccination with one of the ~140 SARS-CoV-2 candidate vaccines in development is effective, the question then is whether SARS-CoV-2 will evolve to escape immunity induced by prior infection or vaccination? The answer to this is unknown. However, as the evolutionary rate of SARS-CoV-2 is lower than that of, for example, influenza A virus, any such antigenic evolution is also expected to continue more slowly.

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## REFERENCES

1. Mateus J. Selective and cross-reactive SARS-CoV-2 T cell epitopes in unexposed humans. *J Sci.* 2020;370(6512):89-94.
2. Siegel R, Naishadham D, Jemal A. Cancer statistics. *J Clin.* 2013;63(2):11-30.
3. Walls AC, Kubrusly MS, Faria MF, Dazzani B, Fonseca RS, Maracaja-Coutinho V et al. Structure, Function, and Antigenicity of the SARS-CoV-2 Spike Glycoprotein. *Cell.* 2020;181(3): 281-292.
4. Gallaher WR. A palindromic RNA sequence as a common breakpoint contributor to copy-choice recombination in SARS-COV-2. *Arch Virol.* 2020;165(2):2341-2492.