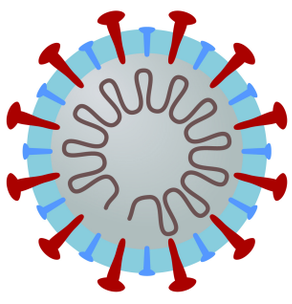
How **SARS-CoV-2** Stacks Up Against Other Coronaviruses

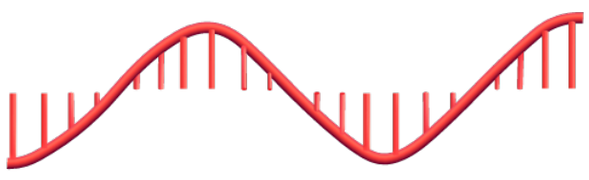
Coronaviruses are a class of virus in the order Nidovirales, single-stranded RNA viruses that replicate using a nested set of mRNAs (hence the name “nido-“, which means nest).[1] Coronaviruses are known for the following unique features:

Their Structure



Coronaviruses have membranes studded with glycoprotein spikes, which surround a genome encased in a nucleocapsid.

Their Genomes



Coronaviruses have the largest viral genomes and are also one of the only RNA viruses with a genomic proofreading mechanism.[2]

Their Zoonotic Origin

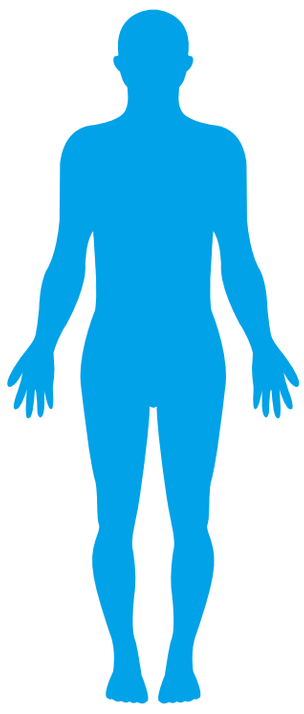
Bats and rodents are the gene source of most alpha and beta-CoVs, while birds serve as the main source of gamma and delta-CoVs.[3]

The Human Coronaviruses

Coronaviruses were first discovered in the 1960s, and until recently they were primarily known for their role in causing 15–30% of common cold cases. It wasn’t until 2001, with the emergence of SARS-CoV, that they became cause for greater concern.[4]

Common cold viruses:

- HCoV-229E
- HCoV-NL63
- HCoV-OC43
- HCoV-HKU1



Coronaviruses causing more severe illness:

- SARS-CoV: severe acute respiratory syndrome (SARS)
- MERS-CoV: Middle East Respiratory Syndrome (MERS)
- SARS-CoV-2: coronavirus disease 2019 (COVID-19)

How They Stack Up

VIRUS	TYPE	EFFECTIVE REPRODUCTIVE NUMBER	INCUBATION PERIOD[5]	HUMAN BINDING SITE[8]	UPPER RESPIRATORY TRACT INFECTION	LOWER RESPIRATORY TRACT INFECTION
229E	Alpha	No Data	2-5 days	APN (CD13)	X	
NL63	Alpha	No Data	2-4 days	ACE2	X	
OC43	Beta	1.56[6]	2-5 days	*O*-Acetylated Sialic Acid	X	
HKU1	Beta	1.85[6]	2-4 days	*O*-Acetylated Sialic Acid	X	
SARS-CoV	Beta	2-4[7]	2-11 days	ACE2		X
MERS-CoV	Beta	<1	2-13 days	DPP4		X
SARS-CoV-2*	Beta	2-3[7]	3-6 days	ACE2	X	X

** All figures for SARS-CoV-2 are estimates based on best available information*

The Perfect Storm

One of the main questions surrounding the current situation is why this coronavirus has led to a global pandemic, while others have had a lifecycle that is fairly benign or self-limiting. The fact that SARS-CoV-2 can take hold in both the upper and lower respiratory tract is thought to be a key factor in this. COVID-19 infections have two places to take hold, thus combining the transmissibility of the common cold with the lethality of SARS-CoV and MERS. In addition, though other coronaviruses bind to ACE2, SARS-CoV-2 does so at a different binding domain that makes it 10–20 times more likely to bind than SARS-CoV.[2] These have been driving factors in creating pandemic conditions with this virus.

References:

1. https://www.uptodate.com/contents/coronaviruses/print
2. https://www.nature.com/articles/d41586-020-01315-7
3. https://www.sciencedirect.com/science/article/pii/S0966842X16000718
4. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3416289/
5. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7098031/#B2
6. https://www.medrxiv.org/content/10.1101/2020.03.04.20031112v1
7. https://www.aljazeera.com/news/2020/06/brazil-coronavirus-death-toll-nears-50000-live-updates-200620230549593.html
8. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5456285/

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