Even though most coronavirus diseases are typically mild, two previous outbreaks in the past decades, SARS-CoV and the Middle East Respiratory Syndrome CoV (MERS-CoV), were severe. Collectively, SARS-CoV and MERS-CoV have produced more than ten thousand cumulative cases with a mortality rate of approximately 10% and 37%, respectively [7, 8].

The WHO recently classified SARS-CoV-2 as a β-Coronavirus, its genetic sequence displayed more than 80% similarity to SARS-CoV and 50% to MERS-CoV, both having their origins in bats [8]. Transmission since early on in the SARS-CoV-2 outbreak has been primarily from human-to-human, via direct contact or droplets expelled by sneezing or coughing from an infected individual at close range. Some fecal-oral transmission has been recognized [8, 13]. Prenatal infection of mothers has not shown to result in fetal or newborn infections [10, 11]. The mean reproductive number (R0) based on clinical data of patients for COVID-19 was 2.24–3.58 [11], indicating that each infected individual, on average, would spread the disease to ∼2–3 other people [12, 13].

The virion of SARS-CoV-2 is approximately 50–200 nm in diameter [13]. It has at least four known structural proteins: spike (S) protein, envelope (E) protein, membrane (M) protein, and nucleocapsid (N) protein (see Fig. 1). All are required to assemble a complete viral particle. Recent studies have shown that some CoVs do not need all four proteins to be an infectious virion, suggesting that other proteins with overlapping functionality may also be encoded [15].

The S protein is responsible for promoting host attachment and virus-target cell membrane fusion during virus infection [15, 16]. This protein has three segments that form a crown-like structure on the envelope, from which the family name derives: corona is Latin for crown [30]. The portions are constituted by a single-pass trans-membrane anchor, a short intracellular tail, and a large ectodomain, which consists of an S1 receptor-binding subunit S1 and a membrane-fusion subunit S2. The S1 portion binds to an ACE2 receptor and the serine protease TMPRSS2 on the host cell surface for viral attachment [29], the S2 portion mediates the fusion of the host and viral membranes, allowing viral genomes to enter host cells [17–19].

Earlier this year [20], the ACE2 receptor was recognized as the SARS-CoV-2 receptor, which is present in multiple human tissues, including type I and type II alveolar epithelial cells in the lung, in the gastrointestinal tract, in the basal cell layer of epidermis and hair follicles [21]. Also present at the cellular level in the central nervous system [22, 23], and in the kidneys and testes [24].

The SARS-CoV-2 S protein shares around 76% amino acids with the SARS-CoV and MERS protein [25], which may help explain its relatively high binding affinity to the human ACE2 receptor [23].

The N protein is attached to the CoV RNA genome and builds the nucleocapsid, which is part of the viral structure.

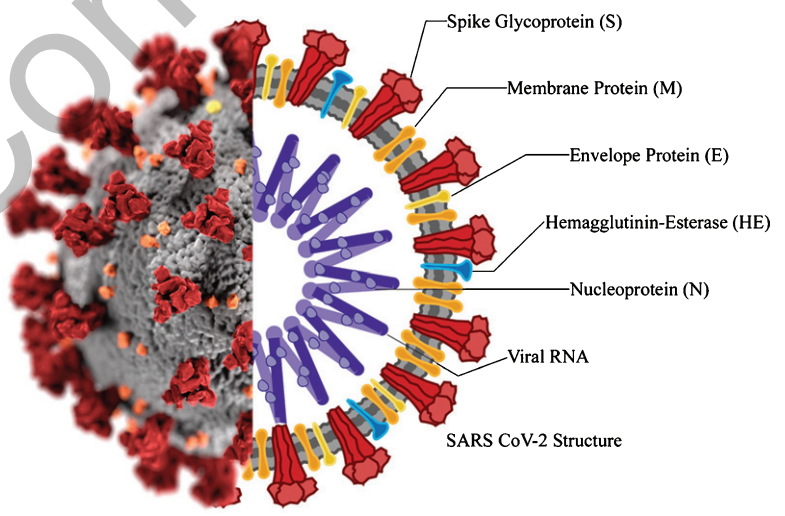

Fig. 1. Illustration of COVID-19 Structure. Adapted from the CDC images library [27] and Encyclopedia Britannica [28]. *Illustrated by Dr. Joe Bolanos*.