between the genome sequence of the new

coronavirus (SARS-CoV-2) and SARS-like CoVs,

the comparative analysis recognized a furin-like

cleavage site in the SARS-CoV-2 S protein that is

missing from other SARS-like CoVs (99). The furin-

like cleavage site is expected to play a role in the life

cycle of the virus and disease pathogenicity and

might even act as a therapeutic target for furin

inhibitors. The highly contagious nature of SARS-

CoV-2 compared to that of its predecessors might be

the result of a stabilizing mutation that occurred in

the endosome-associated-protein-like domain of

nsp2 protein.

Similarly, the destabilizing mutation near the

phosphatase domain of nsp3 proteins in SARS-CoV-

2 could indicate a potential mechanism that

differentiates it from other CoVs (100). Even though

the CFR reported for COVID-19 is meager

compared to those of the previous SARS and MERS

outbreaks, it has caused more deaths than SARS and

MERS combined (101). Possibly related to the viral

pathogenesis is the recent finding of an 832-

nucleotide (nt) deletion in ORF8, which appears to

reduce the replicative fitness of the virus and leads to

attenuated phenotypes of SARS-CoV-2 (256).

Coronavirus is the most prominent example of a