SARS-CoV-2-Proposed Mechanism

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Figure 1 Possible mechanism of action of SARS-COV-2. For the binding of SARS-COV-2 to its ACE-2 (Angiotensin-converting enzyme) receptor present in the respiratory epithelium and alveoli of the lungs, the subunits of S protein cleaved into S1 and S2 domains by the proteases followed by cleaving and shedding of ACE-2 by ADAM 17 into the extra membrane space, which leads to a reduction in the amount of Angiotensin II causes vascular permeability and alveoli injury by conversion of angiotensin I to angiotensin II by ACE2 leads to respiratory distress. After binding of the virus to the host cell, the virus fuses with the membrane with the help of S2 protein and enters into the cell, which is followed by a translation of viral polymerase protein, and replication of the RNA proteins, ORF8b, ORF3a, TRAF3, E proteins recruits inflammasome followed by the transcription of NF-KB pathway which leads to the production of cytokine and further results in cytokine storm which leads to respiratory distress.