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Virulence of COVID-19

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About the Study

The SARS-CoV-2 pandemic impacting the respiratory system poses serious difficulties to public health and necessitates immediately that we enhance understanding of the pathophysiology of COVID-19, particularly host factors that facilitate infection and replication. The SARS-CoV-2 virus utilizes angiotensin converting enzyme (ACE-2) for cell entry and infection. The binding site for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is ACE-2, which mediates virus entrance into cells. Furthermore, co-expression of ACE-2 promotes SARS-CoV-2 entrance and proliferation in epithelial cells *via* cellular mechanisms of receptor activity. It was shown that the affinity of spike proteins in respiratory cells to ACE2 was far greater than that of any human coronavirus previously discovered. This enzyme also plays a crucial role in the renin-angiotensin-aldosterone system, which helps to maintain cardiovascular function. The impact of such ACE-2 overexpression should not be underestimated. Increased vulnerability and pathogenicity of SARS-CoV-2 *via* 7-nAChR, as well as elevation of small airway ACE-2 expression, may also be significant. The transitory secretory population of ACE2 bronchial branches was also identified in the recent studies. Coronaviruses have been found to employ several cellular input pathways for membrane fusion after receptor binding including endosomal and non-endosomalentry in the presence of proteases.

The rate of human-to-human transmission with an overall number of deaths being substantially more than that in SARS and MERS patients within 3 months of the COVID-19 outbreak has been significantly higher for SARS-CoV-2 infections. The emerging global transmission of SARS-CoV-2 and its substantial impact on public health require cooperative efforts to improve our understanding of pathology, its entrance into host cells and the rapid replication of host components, which explain the high rates for human-to-human transmission. Investigation into the tissues of the upper and lower airways of COVID-19 patients as well as healthy individuals is immediately required to improve our understanding of host factors facilitating the entry and replication of the virus, ultimately leading to SARS-CoV-2 infection treatment strategies, even though our understanding of host genetic factors involved in COVID-19 disease outcome is still poor. The studies showed that dataset resources has the ability to derive novel hypotheses and evaluate existing models which should be followed by independent investigations.

Previous research has revealed that they can increase the virulence and inflammatory profile of infections like *Streptococcus pneumoniae*, among other negative biological effects. Nevertheless, a number of demographic variables make such estimations unlike the predominance of their COVID-19 patients recorded by very few of the epidemiological research. This research will be crucial in the advancement of SARS-CoV-2 research and in giving credible, peer-reviewed, and real-world evidence to the youth of the 2020s. It is impossible to focus on anything other than the immediate threat during a pandemic. The “primacy of rescue” has eclipsed precautionary measures. Easy development of those vulnerable to serious diseases or able to be transmitted asymptomatically is a significant objective for the community management of the disease. COVID-19 is a dress rehearsal for the next pandemic, and the next, and the one after that: the new norm.