

Haemagglutinin, neuraminidase and haemagglutinin esterase on the surface of SARS-CoV-2?

Dear Editor

We read an article by Alketbi et al.,¹ in which the authors stated that 'Binding of surfactant proteins (SP), which importantly contribute to the surfactant behaviour as a defence system, to the virus occurs by recognition of haemagglutinin and neuraminidase glycans on the surface of the virus, thereby hindering the ability of the virus to enter the cell. However, the hemagglutinins found on severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) exhibit antigenic variations that resulted in reduced binding, leading to greater virulence and subsequent high mortality and morbidity in patients. SARS-CoV-2 haemagglutinin-esterase (HE) provides classical glycan-binding lectin activity, while exhibiting haemagglutination and destruction of the surfactant proteins'. However, according to the scientific evidence and phylogenetic analysis of the genome of SARS-CoV-2 lacks the haemagglutinin, neuraminidase, and HE gene and it has no HE glycoprotein, thus HE can't be used for the SARS-CoV-2 entry.²



Severe acute respiratory syndrome coronavirus 2, the viral agent of COVID-19, belongs to betacoronavirus genera (lineage B) and is the third agent of coronavirus related epidemics in the last two decades after SARS-CoV and MERS-CoV.^{3,4} According to virology text, betacoronaviruses have four structural proteins, including spike glycoprotein (S), envelope protein (E), membrane protein (M), nucleocapsid protein (N).⁴⁻⁶ Although the lineage A betacoronaviruses such as HKU1-CoV, and HCoV-OC43 can encode HE, the betacoronaviruses lineage B including SARS-CoV-2 cannot encode HE glycoprotein.^{7,8}

AUTHOR CONTRIBUTIONS

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CONFLICT OF INTEREST

No conflict of interest declared.

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DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

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