Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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Comment on "COVID-19 and diabetes: Association intensify risk factors for morbidity and mortality."

Sharma et al. presented hemagglutinin esterase as a structural protein on the SARS-CoV-2 surface in a recent review article in Biomedicine & Pharmacotherapy [1]. However, evidence suggests that the SARS-CoV-2 genome lacks the HE gene [2–6]. In addition, the authors did not presented envelope protein in structure of SARS-CoV-2 in [1].

Although most beta coronaviruses recognize 9-O-acetyl-SAs, this has changed as a result of coronavirus evolution. The hemagglutinin esterase (HE) gene was horizontally adapted from an influenza C-specific HEF and transferred to a beta coronavirus lineage A (OC43-CoV, HKU1-CoV, and Bovine-CoV). Cross-species transmission and HE evolution both contribute to HE adaptation. This proves viral compatibility with host glycans [7]. Thus, studying emerging viruses like SARS-CoV-2 may help us better understand the viral evolution process.

COVID-19 is caused by SARS-CoV-2, a beta-coronavirus of lineage B. It encodes four structural proteins: [1] the spike-surface glycoprotein [2], the small envelope protein [3], the membrane glycoprotein, and [4] the nucleocapsid protein, as well as several nonstructural proteins; however, HE is encoded by other betacoronaviruses in lineage A, including HCoV-OC43, HCoV-HKU1, BCoV, and MHV [2,8].

In conclusion, evidence suggests that the SARS-CoV-2 genome lacks the HE gene, and thus HE cannot play a role in SARS-CoV-2 replication.

CRediT authorship contribution statement

Ahmad Hosseinzadeh adli: Conceptualization, Supervision. **Sanaz Baghban Rahimi**: Writing – review & editing.

Declarations

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