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SARS-CoV-2 spillover transmission due to recombination event

Nariman Shahhosseini [a,*], Gary Wong [a,b], Gary P. Kobinger [a,c,d,e], Sadegh Chinikar [f,g]

[a] Département de Microbiologie-Infectiologie et d'Immunologie, Université Laval, Québec City, Québec, Canada
[b] Pasteur Institute of Shanghai, China
[c] Department of Medical Microbiology, University of Manitoba, Winnipeg, Manitoba, Canada
[d] Department of Immunology, University of Manitoba, Winnipeg, Manitoba, Canada
[e] Department of Pathology and Laboratory Medicine, University of Pennsylvania School of Medicine, Philadelphia, PA, USA
[f] Institute of Virology, University of Veterinary Medicine, Vienna, Austria
[g] Pasteur Institute of Tehran, Iran

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ABSTRACT

In late 2019, a novel Coronavirus emerged in China. Perceiving the modulating factors of cross-species virus transmission is critical to elucidate the nature of virus emergence. Using bioinformatics tools, we analyzed the mapping of the SARS-CoV-2 genome, modeling of protein structure, and analyze the evolutionary origin of SARS-CoV-2, as well as potential recombination events. Phylogenetic tree analysis shows that SARS-CoV-2 has the closest evolutionary relationship with Bat-SL-CoV-2 (RaTG13) at the scale of the complete virus genome, and less similarity to Pangolin-CoV. However, the Receptor Binding Domain (RBD) of SARS-CoV-2 is almost identical to Pangolin-CoV at the aa level, suggesting that spillover transmission probably occurred directly from pangolins, but not bats. Further recombination analysis revealed the pathway for spillover transmission from Bat-SL-CoV-2 and Pangolin-CoV. Here, we provide evidence for recombination event between Bat-SL-CoV-2 and Pangolin-CoV that resulted in the emergence of SARS-CoV-2. Nevertheless, the role of mutations should be noted as another influencing factor in the continuing evolution and resurgence of novel SARS-CoV-2 variants.

1. Introduction

Emerging infectious diseases, including Coronaviruses (CoVs), are often the result of a cross-species transmission of viruses from animals to humans. This can happen through several genetic mechanisms including recombination and mutations that give a virus new features and enable the virus to bind and enter into a new host cell with greater efficiency, avoid the immune system, and modifying its virulence (Longdon et al., 2014). An example of this cross species transmission is Severe Acute Respiratory Syndrome (SARS) (China-2002) and Middle East Respiratory Syndrome (MERS) (Saudi Arabia-2012). In the both scenarios, the CoV originated from bats, and amplified in a mammalian host, e.g. Himalayan palm civet (SARS) and dromedary camels (Chan et al., 2020a).

In December 2019, a novel coronavirus was reported in Wuhan, China. The virus was named SARS-CoV-2 and causes Coronavirus disease 2019 (COVID-19) with severe pneumonia symptoms (Guan et al., 2020). In the beginning of 2020, COVID-19 became a national disaster in China, and soon after swept the world. On 11 March 2020, the World Health Organization characterized COVID-19 as a pandemic (Millán-Oñate et al., 2020). In the late 2020, a novel variant of SARS-CoV-2 known as Variant Of Concern (VOC-202012-01) was emerged in England, and very soon became the dominant circulating viral variant in numerous countries around the world (Simmonds, 2020).

The pathogenicity of CoVs differs from each other (Cunha and Opal, 2014). Human CoVs mainly cause mild symptoms (e.g. 229E and OC43), but two Betacoronaviruses cause severe illness and fatality; (i) SARS-CoV infected 8000 humans (774 deaths) (Donnelly et al., 2003), (ii) MERS infected 2494 humans (858 deaths) (Lee et al., 2016). Like all other unknown viruses, very little is known about SARS-CoV-2.

The CoVs have several non-structural proteins (nsps), and structural proteins (sps). The most studied structural protein is the Spike protein (S-protein), which plays a key role in the pathogenicity of SARS-CoV. It is known that SARS-CoV uses human angiotensin-converting enzyme 2 (hACE2) as one of the main receptors (Lai et al., 2020). Mutations in the

Abbreviations: CoV, coronavirus; SARS, severe acute respiratory syndrome; Bat-SL-CoV-2, Bat SARS like Coronavirus 2; RBD, receptor binding domain; MERS, Middle East Respiratory Syndrome; COVID-19, coronavirus disease 2019; hACE2, human angiotensin-converting enzyme 2.

* Corresponding author.
E-mail address: nariman.shahhosseini.1@ulaval.ca (N. Shahhosseini).

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