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Muthanna Medical Journal
2022; **9**(2):105-110

http://dx.doi.org/10.52113/1/1/2022-105-110

A glance on severe acute respiratory syndrome coronavirus 2 (SARS COV-2): A brief review

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Abstract

"Coronavirus disease 2019" is a highly contagious and infectious illness and cause globally acute upper respiratory systemic disease at latest 2019, major causing agent is "coronavirus-2" descent; also called "SARS COV-2". A "coronavirus" has a lower mortality in parallel with others, (SA.RS-CoV and ME.RS-CoV). In majority of people illness is in medium state; nevertheless, pneumonia may be developed, acute distress syndrome (AR.DS), and dysfunction in multiple organs (typically the aged and who having several other illness). many people are asymptomatic, while around 20% have moderate-to-severe illness. In current topic, can summarize the virologic basic of "coronavirus", which includes clinical features, ligand usage, mutations, epidemiology, pathogenesis, and highlighting its significant differences from previously known coronaviruses.

Keywords: COVID-19, SARS-CoV-2, ARDS; MERS-CoV, Pneumonia

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Received 27 July 2022, Accepted 05 September 2022, Available online 27 September 2022.

General description:

Coronaviruses are large group of infectious agents have ability to causing disease with different degree of severity in animal and human. The 2 extremely deadly zoonotic "coronavirus" appeared in 2002 and 2012, respectively were (SARS-CoV and MERS-CoV). They are caused fatally illness, upon the twenty-first century, emerging coronaviruses have become a newly national health problem [1]. A newly "coronavirus-2" appeared in the town of Wuhan during 2019 and causing flare up sever lung infection. As well as it has ability of rapidly spreading [2]. These continuing outbreaks are presented a considerable danger to worldwide health [3, 4].

Genomic and Structural biology:

The "coronavirus-2" it has (30 kb) roughly genome of (+ RNA and single-stranded family nature). It is containing several genetic fragments about (14) called open reading frames which may be fused seldomely. The 5'UTR has 265 nucleotides while the 3'UTR has 342 nucleotides. The virion has a diameter of around 50–200 nanometers. Several of proteins found in virion composition these includes (spike, envelope, and membrane proteins) these participate in envelope formation, while nucleocapsid proteins hold the RNA genome and its stability [5].

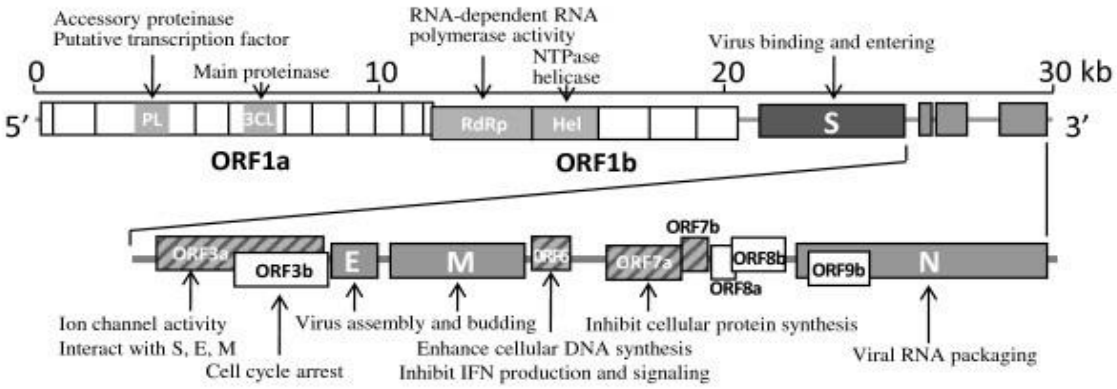

Figure 1.

Genome organization of "coronavirus-2" hosted by https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2676248/**.**

Mutations

According into genomic sequencing, the mutated form of closely related "coronavirus" is the receptor binding domain. Recently thought that "coronavirus-2" started in vampire then evolved to infect other mammals. A slight variation (at a specific location in the genome) might greatly improve (the Wuhan coronavirus's) tendency of interrelate with human angiotensin receptor called (ACE2). As a result, Wuhan coronavirus evolution in patients should be monitoring in stationary manner because of appearance of newly mutations at the 501 and at 494 positions but in low degree in its genome, in order to forecast the likelihood of a more significant outbreak than has been witnessed thus far [6].

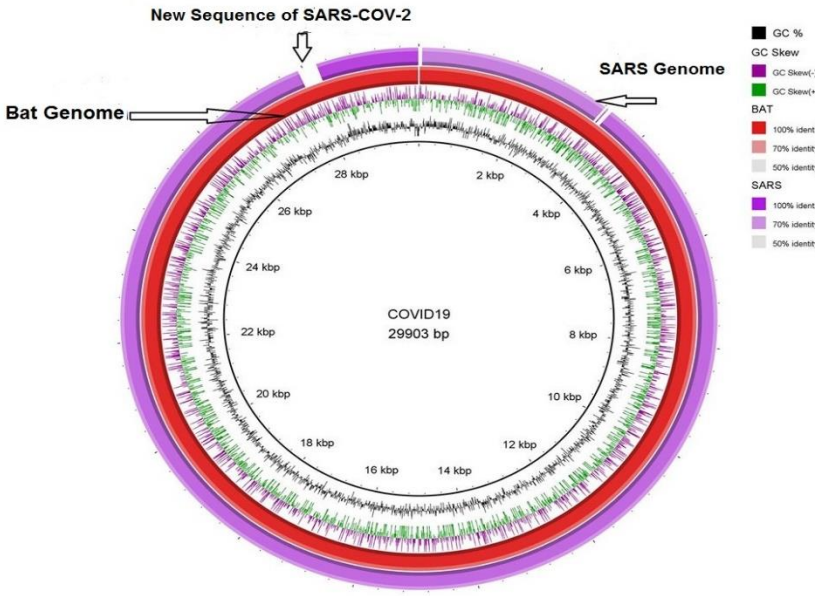

Figure 2.

Genomic mutation in SARS-CoV-2 hosted by https://lh3.googleusercontent.com\nvqICBzG0W9YVCnd8xEv3EuVXTIhdgGqOy8nTEs_QGvBFQZ4uE-8uIvkQ40gA_qYi_EUg=s106.

Epidemiology

In December 2019 of China, the 2019-nCoV was initially discovered in patients with pneumonia. It has outperformed of "SARS-CoV and MERS-CoV" in terms of transmission, after that, it has ability of spreading over more nations in "Asia, Europe, North America, South America, Africa, and Oceania"; SARS-CoV-2 sated by the WHO as globally during 11 March 2020 and recorded about 1,118,921 total cases as of 4 April 2020. There have been 58,937 fatalities attributable to the virus, with 226,769 persons recovering from illness, suggesting that there are 833,215 ongoing cases [7,8,9,10].

Transmission

Transmission of the virus from "one individual to an another" have documented, through contact with droplet from "coughing, sneezing and conjunctiva; via mouth, nose and eyes, respectively" in restrict about (1m) of patient. The environmental surroundings fomites also which important rout of transmission. As consequence, the spreading of virus could be via "direct as well indirect contact with surrounding region or infected person products" [11,12].

Pathogenesis

The plasma pro-inflammatory cytokines and chemokine's including "interleukin 1 beta IL1-β, IL-1 receptor antagonist IL1RA, IL7, IL8, IL9, IL10, basic fiber growth factor 2 BASIC FGF, granulocyte colony-stimulating factor (G-CSF), granulocyte-macrophage colony-stimulating factor GM-CSF, interferon gamma IFNγ", higher leukocyte numbers as well as abnormal respiratory findings were all found to be higher in patients infected with COVID-19. Several symptoms appear on patients such as "high body temperature, cough, and tiredness, as well as sputum production, headache, hemoptysis, diarrhea, and dyspnea" [13].

Although the patients had clinical pneumonia, which was corroborated by chest CT findings, there were aberrant characteristics such as RNA anemia, adverse respiration, abrupt heart damage, as well as an increased incidence of ambiguity, which ultimately to kill of individual [14].

The spike protein possesses a variable "receptor-binding domain" which connect with the angiotensin enzyme receptors existence on normal tissues "human heart, lungs, kidneys, and gastrointestinal tract cells" with enough affinity to employ them as a cell entrance mechanism. Basigin, also known as a cluster of differentiation 147 (CD147), may be used by SARS-CoV-2 to achieve cell entrance. The "coronavirus-2" binds to the angiotensin enzyme receptors in type 2 lung cells, which sets off lower respiratory tract ignition reaction. However, when S spike protein attaches to angiotensin enzyme receptor, the transmembrane protease 2 proteolytically processes the compound, cleaving "ACE-2" and activating the S protein, enabling cell entering [15].

Immune response is triggered by viral entrance and cell infection, series of inflammatory process is started by "antigen-presenting cells". Which presenting of infectious agent fragments to "CD4+-T-helper Th1" cells and further activation to Th1 via emission of interleukin-12. Which activate "CD8+ T-killer (Tk) cells", which attack foreign antigen that carry by cells. The cytotoxic killing cells furthermore activate "B cells" to generate antibodies specific for antigen [16].

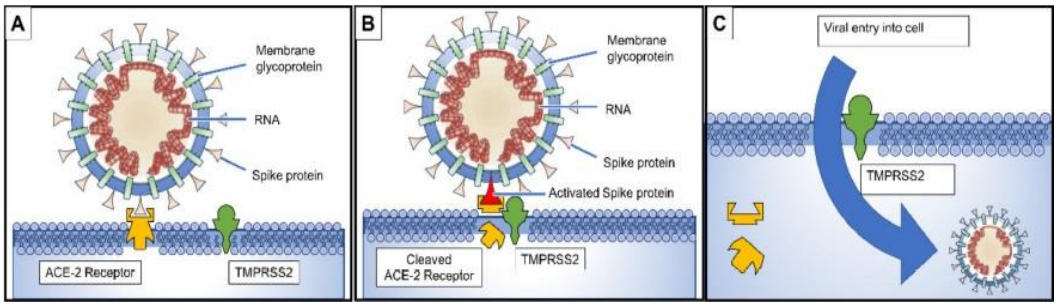

Figure 3.

(**A**) "Coronavirus S proteins bind to angiotensin enzyme receptors called (ACE-2)" on cell exterior (**B**) The type II serine protease enzyme bind and cleave of angiotensin enzyme receptors which leads to S protein activation (**C**) Cleaved angiotensin receptors as well as activated S protein assist in entering of virus; in addition; expression of serine protease enzyme boosts coronavirus entry in cells [15].

Ethical Approval

The study was approved by the Ethical Committee.

Conflicts of Interest

The authors declare that they have no competing interests.

Authors' Contributions

All authors shared in conception, design of the study, acquisition of data, and manuscript writing, the critical revising and final approval of the version to be published.

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