A Brief Study on Rate of Morphological and Genomic Similarities between SARS and COVID-19

Chandra Mohan1,*, Surya Chauhan2, Shashank Shekhar3, Payal Kaushik4, Madhu5, Mahima Sharma6, Vinod Kumar7

1,7 Assistant Professor, SBAS, K. R. Mangalam University, Gurugram 122103, Haryana, India
2,3,5,6 Students, K. R. Mangalam University, Sohna road, Gurugram 122103, Haryana, India.
7 Assistant Professor, SBAS, K. R. Mangalam University, Gurugram 122103, Haryana, India.

ARTICLE INFO

Received: 12 May 2020
Accepted: 01 Jun 2020

This is the third time in the 20th century on which humanity under foe due to a viral infection of coronavirus, after Sever Acute Respiratory Syndrome (SARS) in 2002 and Middle East respiratory syndrome coronavirus (MERS-CoV) in the year 2012. Recently, in the year 2019 in Wuhan, China, the novel viral SARS-CoV-2, caused the disease COVID-19 possessing identical morphological and genetical features showed by SARS in 2002. Researchers also believe that these similarities can explore the pathogenesis and help to find out the treatment of COVID-19. The current review focused on the morphological and genomic study of novel COVID-19 with SARS and also explores the sequences of events occurring in the pathogenesis of the SARS-CoV-2 virus.

KEY WORDS: SARS, COVID-19, life cycle, worldwide scenario, morphology of SARS-CoV-2, genomic structure.

1. INTRODUCTION

Throughout the history, humanity has recognized various different pandemics, in which some of them were highly destructive than others. At present, we all are facing the new pandemic condition known as novel COVID-19 infection. On 30th January 2020, the COVID-19 epidemic was officially declared as a public health emergency of
International Concern. At the initial stage it started from 12th December 2020 possibly related to the seafood market, Wuhan, province of China [1,2] and now rapidly spreading across the world. Coronavirus is basically a family of viruses in the Nidovirales order [3]. It is a very significant viral pathogen in both humans as well as humans. SARS is considered to be the first serious emergent disease of the 21st century. It was found to be caused by a previously unknown lineage of coronavirus (SARS-CoV), most likely of zoonotic origin [4]. The SARS-CoV genome is about 30 kb long. Differential transcription of its genes generates a gradient of subgenomic RNAs (sgRNA) with a common 3′ end. This implies that genes at the 3′ end should be transcribed at high levels and that sgRNA species produced by transcription of these genes might be the most abundant during cell infection. The Clinical symptoms of COVID-19 patients range from mild to severe. Generally it takes 2-14 days after exposure for symptoms to develop in the human body [5]. The symptoms include fever, cough, fatigue, shortness of breath or difficulty [6] in breathing and a small population of patients also appeared gastrointestinal infection symptoms as shown in fig. 1. Other symptoms include, tiredness, aches, runny nose, sore throat, headache, diarrhea, vomiting and also many people experienced the loss of smell or taste [7]. Some people may have no symptoms at all but can be infected by the disease [8].

**2. MORPHOLOGY OF SARS AND COVID-19**

Coronavirus and Torovirus, differs in nucleocapsid morphology, the former being helical and the latter being tubular. COVID-19, primarily targeted the lung and caused alveolar contraction by altering the surface tension and exchange of gases. In humans, another viral species known as Severe acute respiratory syndrome (SARS) causes similar respiratory disease characterized by fever, cough, and muscle ache, often with progressive difficulty in breathing [9]. Coronavirus are enveloped, pleomorphic or spherical particles, 150 to 160 nm in size, associated with positive single stranded RNA, with viral proteins (N), membrane glycoprotein (M), and spike glycoprotein (S), however, spike surface glycoprotein plays an essential role in binding to receptors on the host cell and determines host tropism shown in fig. 2. COVID-19 differs from other coronaviruses by encoding an additional glycoprotein named acetyl esterase and hemagglutination (HE) [10-12].

![Fig 2: Representation of SARS-CoV and SARS-CoV-2 morphology.](image)

**3. GENETIC STRUCTURE AND RNA SEQUENCE IN COVID-19**

The researchers formulated the whole genomic of the new virus now known as SARS-CoV-2 causing COVID-19 disease have 79 percent similar genomic structure to as SARS-CoV identified in the year 2002. However, it also showed about 51.8 percent similar to the MERS-CoV identified in the year 2012, and about 87.6 - 87.7 percent similar to other SARS-like cows, based upon above finding we postulate that the novel virus may be originated from bats [13,14]. Till now six different viruses have been identified, named HCoV-NL63, HCoV-229E, HCoV-OC43, HCoV-HKU1, SARS-CoV and MERS. Novel coronaviruses have positive strand RNA and its 2/3 portion encodes viral polymerase [15]. Necessary for the RNA synthesis and two large nonstructural polyproteins that are not involved in host response modulation. The other one-third of the genome encodes four structural proteins (spike (S), envelope (E), membrane (M) nucleocapsid (N), and the other helper proteins. The released viruses can infect kidney cells, liver cells, intestines, and T lymphocytes, as well as the lower respiratory tract, where they form the main symptoms and signs. It also makes the antiviral T-cell response irregular due to the stimulation of T-cell apoptosis, thus causing a collapse of the immune system [16, 17].
4. LIFE CYCLE OF COVID-19

The first step is virus infection is the interaction of sensitive human cells with Spike Protein. Genome encoding occurs that after entering in the cell and facilitates the expression of the genes, proteins, which advance the adaptation of CoVs to their human host. Genome changes resulting from recombination [18], gene exchange, gene insertion, or deletion is frequent among CoVs.

Phase I: Coronavirus entry and replication

Coronavirus gain entry into the host cell by acting on Angiotensin converting enzyme 2 (AEC-2) receptors similarly like SARS and MERS (bind on DPP-4) [19,20]. Angiotensin converting enzyme 2 (ACE2) is an essential regulator of heart function expressed in the heart, lung, kidney and gastrointestinal tract. In addition, ACE2 is a functional receptor that acts as an entry point into human lung cells for coronaviruses such as SARS and novel coronavirus SARS-CoV-2 (2019-nCoV). After getting into host cell it starts replication and make its own copy by using host machinery and finally come out from the cell by exocytosis [21] (Fig. 3).

Phase II: Attack on Upper Respiratory Tract

After entering into the human body, it binds with lung cells and starts killing them, so the lungs begin to fill with fluid making it hard for to breath. At this point, the immune system starts to kick and released several inflammatory mediators like IL1, IL6 and TNF-alpha. These inflammatory mediators stimulate the CNS by which hypothalamus is activated and high body temperature observed. Apart from this patient begins to experience a mild version of symptoms like dry cough, shortness of breath, with headache, muscle pain and tiredness [22, 23].

Phase III: Damage of Lower Respiratory Tract

This virus continues to replicate and journeys further down the windpipe and into the lung, it can cause more respiratory problems like bronchitis and pneumonia by damaging the alveoli cells and enhancing the surface tension with alteration in gas exchange [23, 24]. Restricting oxygen to the bloodstream deprives other major organs of oxygen including the liver, kidney and brain [25].

Phase IV: Need of Ventilator

In a small number of severe cases that can develop into acute respiratory distress syndrome (ARDS), which requires a patient be placed on a ventilator to supply oxygen [26]. However, if too much of the lung is damaged and not enough oxygen is supplied to the rest of the body, respiratory failure could lead to organ failure and death.

GLOBALLY EPIDEMIC CONDITION OF COVID-19

The coronavirus can live around three to four days on nonliving surfaces, like plastic and steel. The virus disintegrates over the course of a day on cardboards and survives for up to 24 hrs. The virus lives longest on plastic and steel, surviving for up to 72 hours. The virus is not airborne but it can travel through the air and stay suspended for about a half-hour [27, 28]. Data obtained from WHO Dashboard, reported that deaths have exceeded 170,000, and more than 2,400,000 people have been infected, while 650,000 have been recovered from COVID-19 infection [11]. According to the World Health Organization (WHO), the USA has now become the epicenter of this COVID-19 pandemic as compared to others countries [29] as shown in Fig.4, with more than 24,000 deaths.

There have been no global SARS outbreaks since 2003.SARS has been successfully contained using public health measures, such as early case detection and isolation, contact tracing and by maintaining social distancing. Will implementing the same measures help in COVID-19 to go away? [30, 31]

Some factors that may contribute to COVID-19 been around for longer include the followings:

- About 80 percent of people with COVID-19 have a mild illness. Some may not even know that they’re sick. This
makes it harder to determine who’s infected and who’s not.

- People with COVID-19 appear to shed the virus earlier in the course of their infection than people with SARS. This makes it more difficult to detect who has the virus and isolate them before they spread it to others [32].
- COVID-19 is now spreading easily within communities. This was not the case with SARS, which was more commonly spread in healthcare settings.
- We’re even more globally connected than we were in 2003, making it easier for COVID-19 to spread between regions and countries [33].

5. CONCLUSION
As per the latest reports received from WHO and Ministries of Health of various countries revealed that more than 33,106 deaths and around 58,411 new cases were observed globally. However, unlike polio and Ebola, the COVID-19 has neither a vaccine nor any clinically approved treatment available till now. Researchers are searching new molecules for the same and we hope very soon we get something in our hand, although, timely taking preventive measures somehow prevent the number of patients and death cases worldwide. We only hope for the best, that very soon we will defeat this deadly situation.

6. ACKNOWLEDGMENT
Authors are thankful to the Management, K. R. Mangalam University, Gurugram, India for providing technical support.

7. REFERENCES
23. Bauch CT, Smith JOL, Coffee MP, Galvani AP. Dynamically modeling SARS and other newly emerging
IIIIIIIII© International Journal of Pharma Research and Health Sciences. All rights reserved


Conflict of Interest: None
Source of Funding: Nil