Acta Scientific Pharmacology

Volume 1 Issue 11 December 2020

Editorial

The Genomic Material of CoV-19 and their Role in Pathogenesis

Sudip Kumar Mandal[1]*, Sipra Sarkar[2] and Dhrubo Jyoti Sen[3]

[1]*Department of Pharmaceutical Chemistry, Dr. B. C. Roy College of Pharmacy and Allied Health Sciences, Durgapur, West Bengal, India*

[2]*Department of Pharmaceutical Technology, Brainware University, Barasat, Kolkata, West Bengal, India*

[3]*Department of Pharmaceutical Chemistry, School of Pharmacy, Techno India University, Salt Lake City, Kolkata, West Bengal, India*

***Corresponding Author:** Sudip Kumar Mandal, Faculty of Pharmacy, Department of Pharmaceutical Chemistry, Dr. B. C. Roy College of Pharmacy and Allied Health Sciences, Durgapur, West Bengal, India.

Received: October 28, 2020

Published: October 29, 2020

The 2019 novel coronavirus (CoV-19) is a dangerous virus which has caused a severe pandemic throughout the world. The covid-19 is not so much different form severe acute respiratory syndrome coronavirus (SARS-CoV) but their mode of communication is human contacts. The gnomic structure of CoV-19 possesses the largest genomes (26.4 to 31.7 kb) among all RNA virus. Two-third of their RNA has viral polymerase, RNA synthesis material and two large non-structural polyproteins that are not actually involved in host response modulation (ORF1a/ORF1b). Other one-third of the gnome encodes structural proteins such as spike (S), envelope (E), membrane (M), nucleocapsid (N) and other helper proteins. The length of their genome showed high variability for ORF1a/ORF1b and structural proteins. It is generally associated with number and size of proteins. Genomic encoding occurs after entering to the human cell and facilitates the expression of genes and encode according to useful accessory proteins with advance adaptation of the virus to their human host [1].

Genome changes time to time with the changing of environment and resulting form gene recombination, gene expression, gene insertion and gene deletion are very frequent in CoV-19 and this will take place in future pandemics as future epidemics. As a result of this rapid changing the virus is expanding with new generation sequencing. CoV-19s rapidly attack the respiratory tract starting from common cold symptoms to severe pneumonia. The first symptoms then causes fever, cough, shortness of breath although diarrhoea is also seen in some patients. The first death was found from covid-19 pneumonia in China. Intestinal infection is also found in some patients. There are other symptoms like chest pain. Breathlessness, nausea, vomiting and confusion leading to oxygen deficiency and death [2].

More advanced virological studies and genetic studies showed that SARS- CoV and MARS-CoV (Middle East respiratory syndrome coronavirus) are transmitted through bats and then to human being. Gene structures have shown that bats CoVs are mostly alpha-CoV and beta-CoV while most birds CoVs are gamma-CoVs and delta-CoVs. It has been reported that novel virus causing epidemics in human coincides the isolated CoVs from bat. CoVs-19 studies showed that five subgenera of beta corona virus formed with five support branches. The sub genus classified into three well supported classes; two SARS-CoVs are related from *Rhinolophus sp* from Bulgaria and Kenia formed Class 1 the 2019 CoVs from Wuhan, China [3,4].

The SARS like corona virus was derived from Bats and was identified for the long batch separating the human and bat viruses. The SARS corona virus having a complete phylogeny of RNA dependent RNA polymerase gene. This evidence indicates 2019-n CoV is a novel beta corona virus from the sub genus sarbecovirus. As the plot reveals the genetic distances among viruses across the 2019-n CoV genome. The phylogenetic analysis of the major encoding regions of representative members of the subgenus sarbecovirus consistent with the genome phylogeny [5].

The envelope spike protein facilitates receptor binding and membrane fusion crucial for determining the host tropism and transmission capacity. Generally, the spike protein is sub dived into S1 and S2 domain; S1 domain responsible for receptor binding and the S2 domain for cell membrane fusion. Different amino acid variations were inspected in the spike protein of Sarbecovirus which directly engages with the receptor commonly located in the C-terminal domain of S1 in SARS-CoV. From genomic surveillance of the clinical samples from patients with viral pneumonia in Wuhan, China, a novel coronavirus (termed 2019-nCoV) has been identified nine patients samples, showed that the virus belongs to the subgenus Sarbecovirus. 2019 CoV was more similar to two bat-derived coronavirus strains, bat-SL-CoVZC45 and bat-SL-CoVZXC21, than to known human-infecting coronaviruses including the virus that has caused the SARS outbreak in 2003 [6].

The average evolutionary rate for coronaviruses is roughly10^{-4} nucleotide substitution per size per year with changing mutation arising during every recycling process. The primary amino acid sequence for 2019-CoV varies from patients to patients is more similar with 99.1% sequence identity. The finding suggested that 2019-CoV aroused from single source but the detection is done rapidly. So, the mutation occurs when they are infecting from one individual to individual [7].

The genomic structure of a seventh human corona virus (SARS-CoV-2) produces severe pneumonia. The use of genetically recombinant coronavirus has established that the spike protein is a major determinant of tropism in host and cause pathogenicity. In the case of CoV-19 the alteration of spike gene from a severe virulent enteric strain renders the virus enterotropic. So, the spike protein is the major determinant of pathogenesis and influence of infection. However, the major protein is not only the spike protein but also the other genes responsible [8].

Bibliography

1. Sahin Ahmet R., *et al*. "2019 Novel Coronavirus (COVID-19) Outbreak: A Review of the Current Literature". *EJMO Eurasian Journal of Medicine and Oncology* 4.1 (2020): 1-7.

2. Yin Y and Wunderink R. "MERS, SARS and other coronaviruses as causes of pneumonia". *Respirology* 23 (2018): 130-137.

3. Zaki AM., *et al*. "Isolation of a novel coronavirus from a man with pneumonia in Saudi Arabia". *The New England Journal of Medicine* 367 (2012): 1814-1820.

4. Sen DJ., *et al*. "Corona is culprit of ridiculous offensive nonsense air". *World Journal of Pharmaceutical Research* 9 (2020): 503-518.

5. Hui DS., *et al*. "The continuing 2019-nCoV epidemic threat of novel corona viruses to global health -The latest 2019 novel coronavirus outbreak in Wuhan, China". *International Journal of Infectious Diseases* (2020): 264-266.

6. Lu R., *et al*. "Genomic characterisation and epidemiology of 2019 novel coronavirus: implications for virus origins and receptor binding". *The Lancet* 22 (2020): 565-574.

7. Tan W., *et al*. "A novel coronavirus genome identified in a cluster of pneumonia cases-Wuhan, China 2019-2020". *China CDC Weekly* 2 (2020): 61-62.

8. Chiu SS., *et al*. "Human coronavirus NL63 infection and other coronavirus infections in children hospitalized with acute respiratory disease in Hong Kong, China". *Clinical Infectious Diseases* 40 (2005): 1721-1729.