

History:

Received: December 11, 2019
Acceptance: December 23, 2019
First Published: Dec. 27, 2019
Revised: February 17, 2020
Revised again: June 19, 2020
Collection year: 2019
Confirmation of publication: 2019

Identifiers and Pagination:

Year: 2019
Volume: 4
First Page: 3
Last Page: 7
Publisher Id: AdvJBiomed Sci.4.3
DOI:http://dx.doi.org/10.21065/AdBiomedSci.4.3

Corresponding author:

Saeed Ur Rasheed Nazir
B.Pharm., M.Phil., Ph.D Assistant
Professor, Faculty of Pharmacy,
College of Pharmacy, University of
Sargodha, Sargodha 40100,
Pakistan. T.: +1 +92 301 462 9275 |
E.: srnazir@yahoo.com |
srnazir@uos.edu.pk

Citation:

Taha Nazir, Ruqaiya Rasheed
kayani, Saeed Ur Rasheed Nazir,
Saba Manzoor, Nida Taha.
Epidemiology, genomic evolution
and molecular features of severe
acute respiratory syndrome-related
Coronavirus. Adv J Biomed Sci.
2019; Vol. 4. p. 3-7

Review Article

EPIDEMIOLOGY, GENOMIC EVOLUTION AND MOLECULAR FEATURES OF SEVERE ACUTE RESPIRATORY SYNDROME-RELATED *CORONAVIRUS*

Taha Nazir¹, Ruqaiya Rasheed kayani², Saeed Ur Rasheed Nazir³, Syed Muzzammil Masaud⁴, Saba Manzoor⁵, Nida Taha¹

1. Microbiology and Molecular Biology Research Group, Advanced Multiple Incorporation, Mississauga ON Canada.
2. Clinical Pharmacist and Consultant Pharmacologist, 74-C, Gulshan-e-Lahore, Lahore, Pakistan.
3. Faculty of Pharmacy, College of Pharmacy, University of Sargodha, Sargodha 40100, Pakistan.
4. Manager Oncotreatments Pvt Ltd, 192/ 8, Paris City Block F, Sector H-13, Islamabad, Pakistan.
5. Clinical/ Community pharmacist, Fazal din Pharma Plus, Valencia Town, Lahore, Pakistan.

Authors' contributions: This work was carried out in collaboration among all authors. **Taha Nazir, Nida Taha** and **Syed Muzzammil Masaud** designed the study, collected the primary information and wrote the initial draft of this article. **Ruqyya Kayani** further elaborate the description. Whereas, **Saeed ur Rasheed Nazir** and **Saba Manzoor** incorporated the citation and managed the literature search. All authors read and approved the final manuscript.

Corresponding Author: Saeed Ur Rasheed Nazir B.Pharm., M.Phil., Ph.D., Assistant Professor, Faculty of Pharmacy, College of Pharmacy, University of Sargodha, Sargodha 40100, Pakistan. T.: +1 +92 301 462 9275 | E.: srnazir@yahoo.com | srnazir@uos.edu.pk

Introduction

In the end of year 2019, in the city of Wuhan (China), individuals were presented with viral pneumonia caused by microbial agent that was unidentified. The agent causing the diseases was identified as novel *Coronavirus*, which was named provisionally 2019 novel *Coronavirus* (2019-nCoV).

2019 novel *Coronavirus* is adequately contradictory from SARS-CoV to be deliberated as a different human-contaminating beta *Coronavirus*. Though the phylogenetic investigation proposes that bats might be the novel virus host, a seafood animal sold in China might characterize a transitional host expediting the appearance of the virus in human beings. Significantly, structural investigation proposes that 2019-nCoV might be capable to tangle to the Angiotensin-Converting Enzyme (ACE)-2 receptor in human beings.

Funding:

The authors received no direct funding for this research.

Competing Interests:

The authors declare no competing interests

Additional information is available at the end of the article.

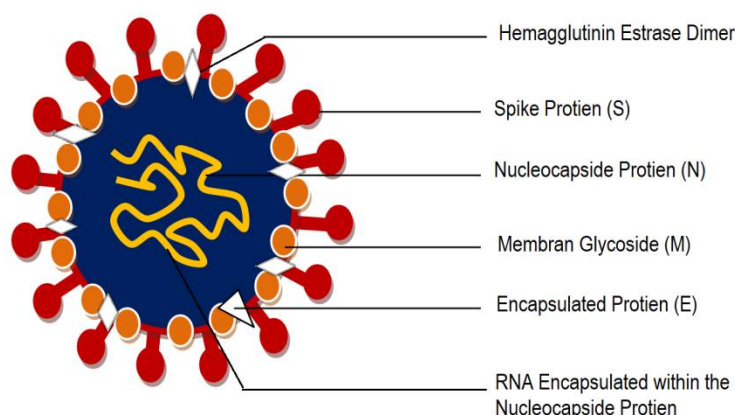


Figure 1. Molecular features of SARS-CoV-2

Additionally, the imminent adaptation, evolution, and extent of this virus permit imperative research (1). The evolutionary association among SARS-COV-2 and the illnesses related to respiration system triggering *Coronavirus* disease in human beings is not related diligently. Relationship was pursued to distinguish the proteins that were translated in SARS-COV2 with further *Orthocoronavirinae* species. Using genome sequence a phylo-genetic hierarchy was fabricated. A constellation hierarchy was established from the retrieved summaries from the absence and presence of homologs of 10 proteins of 2019-nCoV. The collective information was recycled to illustrate the possible association of the proteins of novel *Coronavirus* to further *Orthocoronavirinae* species. The investigation done consistently proposes that novel *Coronavirus* is diligently associated to Bat CoV RaTG13 and belongs to Beta *Coronavirus* (*Sarbecovirus* subgenus), collectively with Bat-SARS- and SARS resembling *Coronaviruses* (2). The phylo-genetic describing collection of homolog proteins of single marked novel *Coronavirus* protein contrary to additional genome classifications exposed two clades of 10 proteins of novel *Coronavirus*. Clade 1 comprised of a cluster of preserved proteins in *Ortho-coronavirinae* containing Nucleocapsid protein, Orf1ab polyprotein, and Membrane protein Spike glycoprotein. Clade 2 contained 6 proteins limited to *Hibecovirus* and *Sarbecovirus*. Two of 6 Clade 2 proteins that were nonstructural, NS7b and NS8, were absolutely preserved amongst SARS-COV2, BatSARS-like Cov and BetaCoV_RaTG. NS7b and NS8 have formerly been revealed to distress immune reaction indicating in the SARS-CoV investigational prototypical. Therefore, we ventured that information of the well-designed variations in the NS7b and NS8 proteins through advancement may deliver significant data to discover the social infectious assets of novel *Coronavirus* (3).

The worldwide blowout of the novel *Coronavirus* is enduring and affecting rapidly. The hazard is hovering worldwide extraordinary, as specified by the World Health Organization. A primary phylogeographic and phylodynamic investigation of this novel *Coronavirus* is provided. A determined clade integrity tree has been constructed by means of the twenty nine accessible whole genome sequences of novel *Coronavirus* and two whole genome categorizations that are extremely comparable sequences from Bat SARS-like *Coronavirus* existing in GeneBank (4). Mechanism of transmission can

be clarified amongst the countries that have delivered the novel *Coronavirus* sequence isolates from their patients. The reconstruction of Bayesian phylogeographic displays that the novel *Coronavirus* most undoubtedly instigated from the Bat SARS-like *Coronavirus* mingling in the bat family of *Rhinolophus* (5). In covenant with epidemiological interpretations, the greatest possible geographic beginning of the novel epidemic was China (Wuhan city), where novel *Coronavirus* time of the most current communal antecedent appeared, conferring to molecular clock scrutiny, about in 25th of November 2019. These outcomes, organized with formerly verified epidemics, propose a recurrent arrangement of periodical epizootic nature because of Beta *Coronavirus*. Furthermore, research defines the identical populace genetic dynamic origin, the SARS 2003 rampant, and recommends the imperative necessity for the improvement of operative molecular investigation approaches of Beta *Coronavirus* amongst animals and bat family *Rhinolophus* (6).

Pathogenesis & Immunity

2019-nCoV was characteristically restricted to the mucosal cells of the respiratory tract. Around 50% of infections are not symptomatic; it is uncertain what character they play in the extent of contamination. Immunity succeeding infection seems to be transitory, and reinfection may happen. Pneumonia triggered by SARS *Coronavirus* is categorized by rambling edema causing in hypoxia. The virus binding to AEC enzyme-2 on the epithelium surface of respiratory tract may promote to the dysregulation of balance in fluids that stocks the alveolar space edema (7).

Epidemiology and Features

The common cold triggered by 2109-nCoV was described as *rhinorrhea*, runny nose scratchy sore throat, and, low-grade fever. This infection characteristically continues quite a few days and has no enduring sequelae. *Coronaviruses* also cause the bronchitis (8). The infection can be identified as “common cold” because that is considered major symptom of *Coronavirus*. If MERS OR SARS is suspected, PCR-based tests or antibody antigen reaction based diagnosis can be used for confirmation. Moreover, there is no rational antiviral therapy or vaccine available yet. A blend of steroids and ribavirin has been tried in the treating life-threatening cases of SARS, but their effectiveness is still unclear.

Genome Evolutionary though Emerging Recombination

We also have tried to illustrate the genetic associations of the novel *Coronavirus* to explore the knowledge of *sarbecovirus* subgenus. The intensities of genetic resemblance amongst the RaTG13 and 2019-nCoV suggest that there may not direct connection and/ or factor that particularly trigger the epidemic in human beings, but the assumption that novel *Coronavirus* has initiated from bats is very prospective. Evidence shows that SARS-COV2 is non-mosaic comprising in virtually partial of its genome of a discrete ancestry inside the beta-*Coronavirus*. These genomic topographies and their prospective relationship with features of virus and virulence in human beings require additional consideration.

Viral Protein Drive the Pathogenesis and Amyloid Aggregation

Protein corona layer is accumulated in biological fluids by synthetic nanoparticle, which expressively affects their bioactivity. As intracellular parasites are viruses segments with numerous biophysical assets in extracellular surroundings. These demonstrate that herpes simplex virus type 1 and respiratory syncytial virus, gather a distinctive and rich protein corona in diverse biological fluids. Furthermore, corona pre-coating differentially distresses viral contagion and activation of immune cells (9). Furthermore, it is demonstrated that amyloidogenic peptides bind viruses in their corona and catalyze amyloid development through surface-supported heterogeneous nucleation. Prominently, HSV-1 catalyzes the aggregation of the amyloid β -peptide (A β 42), a main essential of amyloid plaques in Alzheimer's disease, in animals and in vitro models. Outcomes climax the viral protein corona as a developed layer of that is precarious for the interaction of virus and host, and illustrates a mechanistic conjunction among amyloid and viral pathologies (10).

Replicative Cycle of nCOVID-19

The binding of virus occurs via cells through its spines on surface hemagglutinin; subsequently it goes in the cytoplasm, where uncoating of virus takes place. Two large polypeptides are formed by translating positive-strand genome, which are cleaved itself by the protease. These peptides are then united to produce the RNA polymerase that imitates the genome. Additionally, mRNAs are synthesized and then structural proteins are formed by translation. Assembly of virus takes place and envelope is acquired from the endoplasmic reticulum. (11).

Conclusion

It can be concluded that the most probable carrier of 2109-nCov is bat, human to human transmission takes place by respiratory droplets, so human contact must be minimized to infected patients by using special gears and personal hygiene.

REFERENCES

1. Kleine-Weber H, Elzayat MT, Wang L, Graham BS, Müller MA, Drosten C, et al. Mutations in the spike protein of Middle East respiratory syndrome *Coronavirus* transmitted in Korea increase resistance to antibody-mediated neutralization. *Journal of virology*. 2019;93(2):e01381-18.
2. Lu G, Wang Q, Gao GF. Bat-to-human: spike features determining 'host jump' of *Coronaviruses* SARS-CoV, MERS-CoV, and beyond. *Trends in microbiology*. 2015;23(8):468-78.
3. Braun E, Sauter D. Furin-mediated protein processing in infectious diseases and cancer. *Clinical & Translational Immunology*. 2019;8(8):e1073.
4. Tao Y, Shi M, Chommanard C, Queen K, Zhang J, Markotter W, et al. Surveillance of bat *Coronaviruses* in Kenya identifies relatives of human *Coronaviruses* NL63 and 229E and their recombination history. *Journal of virology*. 2017;91(5):e01953-16.
5. Cheng J, Zhao Y, Xu G, Zhang K, Jia W, Sun Y, et al. The S2 Subunit of QX-

- type Infectious Bronchitis *Coronavirus* Spike Protein Is an Essential Determinant of Neurotropism. *Viruses*. 2019;11(10):972.
6. Reich NG, Lessler J, Varma JK, Vora NM. Quantifying the risk and cost of active monitoring for infectious diseases. *Scientific reports*. 2018;8(1):1-8.
 7. Tao Y, Tong S. Complete Genome Sequence of a Severe Acute Respiratory Syndrome-Related *Coronavirus* from Kenyan Bats. *Microbiology resource announcements*. 2019;8(28):e00548-19.
 8. Tao Y, Tong S. Complete genome sequence of a severe acute respiratory syndrome-related *Coronavirus* from Kenyan bats. *Microbiol Resour Announc* 8: e00548-19. 2019.
 9. Ge X-Y, Li J-L, Yang X-L, Chmura AA, Zhu G, Epstein JH, et al. Isolation and characterization of a bat SARS-like *Coronavirus* that uses the ACE2 receptor. *Nature*. 2013;503(7477):535-8.
 10. Tumbarello M, Treccarichi EM, Corona A, De Rosa FG, Bassetti M, Mussini C, et al. Efficacy of ceftazidime-avibactam salvage therapy in patients with infections caused by *Klebsiella pneumoniae* carbapenemase-producing *K. pneumoniae*. *Clinical Infectious Diseases*. 2019;68(3):355-64.
 11. Izaguirre G. The Proteolytic Regulation of Virus Cell Entry by Furin and Other Proprotein Convertases. *Viruses*. 2019;11(9):837.



© 2017 The Author(s). This open access article is distributed under a Creative Commons Attribution (CC-BY) 4.0 license.

You are free to:

Share — copy and redistribute the material in any medium or format

Adapt — remix, transform, and build upon the material for any purpose, even commercially.

The licensor cannot revoke these freedoms as long as you follow the license terms.

Under the following terms:

Attribution — You must give appropriate credit, provide a link to the license, and indicate if changes were made.

You may do so in any reasonable manner, but not in any way that suggests the licensor endorses you or your use.

No additional restrictions

You may not apply legal terms or technological measures that legally restrict others from doing anything the license permits