

Review Article

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HOW & WHY MISCHIEVOUS CORONA VIRUS TRANSFORM IN TO TERRORIST COVID 19

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ABSTRACT

Earlier in past the whole family members of family Coronviridae happened to be only mischievous family, among which some genus may cause common cold and flue like very non fetal clinical syndrome. Few years back some of them transform into murders after some mutatoin- causing SARS and MERS, but still both of these viruses do not have capacity to spread fast, so could not cause very big pandemic like Covid 19. The biggest factor making Covid 19, is the development of spike protein which can bind to ACE2 receptor on the some type of human body cells, and may be source of disease China, which like Pakistan hide this micro terrorist and deliberately allow it to spread this virus to whole world, as even American president Mr. Trump raise doubt that why Covid 19 did not spread in China country but spread through out the world.

INTRODUCTION

Coronaviridae is a family of enveloped, positive-sense, single-stranded RNA viruses. The viral genome is 26-32 kilobases in length. The particles are typically decorated with large (~20 nm), club- or petal-shaped surface projections (the "peplomers" or "spikes"), which in electron micrographs of spherical particles create an image reminiscent of the solar corona. Coronaviruses are a group of related RNA viruses that cause diseases in mammals and birds. In humans, these viruses cause respiratory tract infections that can range from mild to lethal. Mild illnesses include some cases of the common cold (which is also caused by other viruses, predominantly rhinoviruses), while more lethal varieties can cause SARS, MERS, and COVID-19. Symptoms in other species vary: in chickens, they cause an upper respiratory tract disease, while in cows and pigs they cause diarrhea. There are as yet no vaccines or antiviral drugs to prevent or treat human coronavirus infections. Coronaviruses constitute the subfamily *Orthocoronavirinae*, in the family *Coronaviridae*, order *Nidovirales*, and realm *Riboviria*.^[1,2] They are enveloped viruses with a positive-sense single-stranded RNA genome and a nucleocapsid of helical symmetry.^[3] The genome size of coronaviruses ranges from approximately 26 to 32 kilobases, one of the largest among RNA viruses.^[4] They have characteristic club-shaped spikes that project from their surface, which in electron

micrographs create an image reminiscent of the solar corona, from which their name derives.^[5]

As the corona virus keeps spreading around the world, it will probably keep changing. Experts may find new strains. It's impossible to predict how those virus changes might affect what happens. But change is just what viruses do.

With the recent detection of SARS-CoV-2, there are now seven human corona viruses. Those that cause mild diseases are the 229E, OC43, NL63 and HKU1, and the pathogenic species are SARS-CoV, MERS-CoV and SARS-CoV-2 Corona viruses (order Nidovirales, family Coronaviridae, and subfamily Orthocoronavirinae) are spherical (125nm diameter), and enveloped with clubshaped spikes on the surface giving the appearance of a solar corona. Within the helically symmetrical nucleocapsid is the large positive sense, single stranded RNA. Of the four corona virus genera $(\alpha, \beta, \gamma, \delta)$, human corona viruses (HCoVs) are classified under α -CoV (HCoV-229E and NL63) and β-CoV (MERS-CoV, SARS-CoV, HCoVOC43 and HCoV-HKU1). SARS-CoV-2 is a β -CoV and shows fairly close relatedness with two bat-derived CoV-like coronaviruses, bat-SL-CoVZC45 and bat-SL-CoVZXC21. Even so, its genome is similar to that of the typical CoVs. SARS-CoV and MERS-CoV originated in bats, and it appears to be so for SARS-CoV-2 as well. The possibility of an intermediate host facilitating the emergence of the virus in humans

has already been shown with civet cats acting as intermediate hosts for SARS-CoVs, and dromedary camels for MERS-CoV. Human-to-human transmission is primarily achieved through close contact of respiratory droplets, direct contact with the infected individuals, or by contact with contaminated objects and surfaces.

The lungs are the organs most affected by COVID-19 because the virus accesses host cells via the enzyme angiotensin-converting enzyme 2 (ACE2), which is most abundant in type II alveolar cells of the lungs.^[6] The virus uses a special surface glycoprotein called a "spike" (peplomer) to connect to ACE2 and enter the host cell.^[7] The density of ACE2 in each tissue correlates with the severity of the disease in that tissue and some have suggested decreasing ACE2 activity might be protective,^[9,10] though another view is that receptor increasing ACE2 using angiotensin II blocker medications could be protective.^[10,8] As the alveolar disease progresses, respiratory failure might develop and death may follow.^[9]

SARS-CoV-2 may also cause respiratory failure through affecting the brainstem as other coronaviruses have been found to invade the central nervous system (CNS). While virus has been detected in cerebrospinal fluid of autopsies, the exact mechanism by which it invades the CNS remains unclear and may first involve invasion of peripheral nerves given the low levels of ACE2 in the brain.^[11,12]

Severe co lateral damage to the lungs may be one trigger that activates and over stimulates the immune system through a barrage of signaling chemicals, known as cytokines.

The flood of these chemicals can set off what is referred to as a "cytokine storm." This is a complex interplay of chemicals that can cause blood pressure to drop, attract more killer immune and inflammatory cells, and lead to even more injury within the lungs, heart, kidneys, and brain. Some researchers say cytokine storms may be the cause of sudden decompensation, leading to critical illness in COVID-19 patients.

The common cold and flu causing normal corona virus when develop spike envelop protein S which can bind to ACE 2 (Angiotensin Converting Enzyme 2) receptors on some kind of human cells, it becomes deadly as ACE2 receptors are present numerously in lung causing pneumonia, these ACE 2 receptors are even present on kidney, heart and even brain cells. So now this modified virus due to mutation can attack and invades all most all vital organ of body and its most contagious spreading nature transformed it into great terrorist like Bin Laden who shake USA and whole world. If China could had taken proper strict honest measure to restrict the Covid 19 to its own country, it could had killed some local persons, but never becomes pandemic like this one. Now we anticipate Donald Trump to teach lesson like Barack Obama to take some strict action on China to announce it as a terrorist country who deliberately allow the Covid 19 to spread to pandemic level, saving its own country how question to be investigated.

REFERENCES

- De Groot RJ, Baker SC, Baric R, Enjuanes L, Gorbalenya AE, Holmes KV, Perlman S, Poon L, Rottier PJ, Talbot PJ, Woo PC, Ziebuhr J "Family Coronaviridae". In King AM, Lefkowitz E, Adams MJ, Carstens EB, International Committee on Taxonomy of Viruses, International Union of Microbiological Societies. Virology Division (eds.). Ninth Report of the International Committee on Taxonomy of Viruses. Oxford: Elsevier, 2011; 806–28. doi:10.1016/B978-0-12-384684-6.00068-9. ISBN 978-0-12-384684-6.
- 2. International Committee on Taxonomy of Viruses (2010-08-24). "ICTV Master Species List, 2009; 10: (xls).
- Cherry, James; Demmler-Harrison, Gail J.; Kaplan, Sheldon L.; Steinbach, William J.; Hotez, Peter J. Feigin and Cherry's Textbook of Pediatric Infectious Diseases. Elsevier Health Sciences. p. PT6615. ISBN 978-0-323-39281-5, 2017.
- Woo PC, Huang Y, Lau SK, Yuen KY (August). "Coronavirus genomics and bioinformatics analysis". Viruses, 2010; 2(8): 1804– 20. doi:10.3390/v2081803. PMC 3185738. PMID 21 994708. Coronaviruses possess the largest genomes [26.4 kb (ThCoV HKU12) to 31.7 kb (SW1)] among all known RNA viruses (Figure 1) [2,13,16].
- 5. Almeida JD, Berry DM, Cunningham CH, Hamre D, Hofstad MS, Mallucci L, McIntosh K, Tyrrell DA (November 1968). "Virology: Coronaviruses". Nature. 220(5168): 650. Bibcode:1 968Natur.220.6 50.. doi:10.1038/220650b0. [T]here is also a characteristic "fringe" of projections 200 A long, which are rounded or petal shaped ... This appearance, recalling the solar corona, is shared by mouse hepatitis virus and several viruses recently recovered from man, namely strain B814, 229E and several others.
- Zhang H, Penninger JM, Li Y, Zhong N, Slutsky AS (April). "Angiotensin-converting enzyme 2 (ACE2) as a SARS-CoV-2 receptor: molecular mechanisms and potential therapeutic target". Intensive Care Medicine. 46(4): 586–590. doi:10.1007/s00134-020-05985-9. PMC 7079879. PMID 32125455, 2020.
- Xu H, Zhong L, Deng J, Peng J, Dan H, Zeng X, et al. (February). "High expression of ACE2 receptor of 2019-nCoV on the epithelial cells of oral mucosa" International Journal of Oral Science, 2020; 12(1): 8. doi:10.1038/s41368-020-0074 x. PMC 70 39956. PMID 32094336.
- 8. Gurwitz D (March). "Angiotensin receptor blockers as tentative SARS-CoV-2 therapeutics". Drug Development Research, 2020.
- 9. Li YC, Bai WZ, Hashikawa T (February). "The neuroinvasive potential of SARS-CoV2 may play a

role in the respiratory failure of COVID-19 patients" Journal of Medical Virology, 2020; 92(6): 552–555. doi:10.1002/jmv.25728. PMC 7228394. PMID 3210 4915.

- Baig AM, Khaleeq A, Ali U, Syeda H (April). "Evidence of the COVID-19 Virus Targeting the CNS: Tissue Distribution, Host-Virus Interaction, and Proposed Neurotropic Mechanisms". ACS Chemical Neuroscience, 2020; 11(7): 995–998.
- Gu J, Han B, Wang J (May). "COVID-19: Gastrointestinal Manifestations and Potential Fecal-Oral Transmission". Gastroenterology, 2020; 158(6): 1518–1519.
- Hamming I, Timens W, Bulthuis ML, Lely AT, Navis G, van Goor H (June). "Tissue distribution of ACE2 protein, the functional receptor for SARS coronavirus. A first step in understanding SARS pathogenesis". The Journal of Pathology, 2004; 203(2): 631–7.