As per UGC guidelines an electronic bar code is provided to seure your paper

International Journal for Modern Trends in Science and Technology, 8(01): 324-327, 2022

ISSN: 2455-3778 online
DOI: https://doi.org/10.46501/IJMTST0801055
Available online at: http://www.ijmtst.com/vol8issue01.html

Coronavirus outbreaks: immunity and human health

Ashish A. Patil[1] | Pradnya Bhujangrao Dhutmal[2] | Kaustubh Singh[3] | Khan Farhana Mahreen N.A.[4*]

[1]Assistant Professor, College of Food Technology Ashti, Maharashtra, India
[2]Assistant Professor, Gramin Science Vocational College, Vishnupuri Maharashtra, india.
[3]M.Sc in Food Technology, Banaras Hindu University, Varanasi, Uttar Pradesh, India.
[4*]Assistant Professor, Indian Institute of Food Science and Technology, Aurangabad, Maharashtra, India.
*Corresponding Author Mail Id: khanfarry444@gmail.com

To Cite this Article
Ashish A. Patil, Pradnya Bhujangrao Dhutmal, Kaustubh Singh and Khan Farhana Mahreen N.A.. Coronavirus outbreaks: immunity and human health. *International Journal for Modern Trends in Science and Technology* 2022, *8* pp. 324-327. https://doi.org/10.46501/IJMTST0801055

Article Info
Received: 06 December 2021; Accepted: 19 January 2021; Published: 21 January 2022

ABSTRACT

Coronaviruses were discovered in the 1960s and were given their name because of their crown-like form. A coronavirus can infect both animals and humans on rare occasions. The a etiology of coronavirus disease 2019 (COVID-19) was identified as an acute respiratory disease caused by a novel coronavirus (severe acute respiratory syndrome coronavirus-2 or SARS-CoV-2 previously known as 2019-nCoV) as it swept throughout China and then around the world. The World Health Organization had recorded a total of 13.1 million confirmed cases and 572,426 deaths as of July 14, 2020. (WHO). SARS-CoV-2 is a member of the -coronavirus family with a large genetic overlap with bat coronavirus, implying that bats are the natural host. SARS-CoV-2 exploits the same receptor as SARS-CoV, the coronavirus linked to the 2003 SARS outbreak, angiotensin-converting enzyme 2 (ACE2). It spreads mostly through the respiratory system, with lymphopenia and cytokine storms in the blood of patients with severe illness. This points to the possibility of immune dysregulation as a side effect of the virus's severe sickness. Early detection of this immunological characteristic could aid in the early identification of patients who will develop severe illness. The data on the immune response during COVID-19 infection is reviewed here. The current study outlines our current knowledge of how immune dysregulation and altered cytokine networks contribute to COVID-19 patients' pathogenesis.

KEYWORDS: coronavirus, immune system, respiratory system, human health, Lymphopenia, gut microbial dysbiosis.

1. INTRODUCTION

The Coronavirus illness 2019 (COVID-19), caused by the novel coronavirus SARS-CoV-2, first appeared in Wuhan, Hubei province, China in December 2019 **(Li et al., 2020)** and quickly spread over the world **(Li et al., 2020)**. COVID-19 virus has infected over 9.2 million people worldwide, resulting in over 470,000 fatalities. There are few therapeutic alternatives available, and there is no clinically proven vaccination. Safe and effective therapies are especially needed to prevent, minimize susceptibility, and lessen the severity of COVID-19 **(Amanat &, Krammer 2020)**. During the pandemic, the heterogeneous presentation and clinical consequences of infected individuals across different geographic locales was a startling observation. Elderly people, as well as those with pre-existing disorders like diabetes, hypertension, cardiovascular disease, and cancer, have been shown to have a higher chance of getting more

severe disease as well as death, suggesting that the immune system may play a key role **(Wang, et al., 2020)**. Lympopenia and a cytokine storm were common in patients with severe illness, leading to acute respiratory distress syndrome and multi-organ failure. Lympopenia can impair antiviral immunity and damage the innate and adaptive immune systems, resulting in a poor prognosis **(Azkur et al., 2020)**. Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has been identified as a zoonotic beta-coronavirus, comparable to previous SARS and MERS (Middle East Respiratory Syndrome) coronaviruses **(Liu et al., 2020)**. COVID-19 causes SARS-CoV-2, a severe acute respiratory illness (SARS) with a fatality rate ranging from 2% in China **(Xu et al., 2020)** to 12% in certain regions of Italy **(Conticini et al., 2020).** These scientists conducted post-mortem investigation on lung, liver, and heart tissue from a patient who died from SARS-CoV-2, which revealed severe oedema and desquamation in the lungs, as well as obvious indications of breath difficulties and exhaustion. Some COVID-19 patients had gut microbial dysbiosis, with lower levels of probiotics such Lactobacillus and Bifidobacterium, implying that all patients' nutritional and gastrointestinal function should be assessed **(Xu et al., 2020)**. Previously, dysbiosis of the human gut microbiota has been related to a variety of health problems, including RTIs, via the gut-lung axis **(Chan et al., 2020)**. Many research has looked at the link between the ingestion of probiotics or symbiotics in various forms and the beginning of disorders, with a particular focus on RTIs **(Auinger *et al.*, 2013; Cohen *et al.*, 2013; Gerasimov *et al.*, 2016; Panigrahi *et al.*, 2017)**. In COVID-19 infected patients, nutritional support and the use of prebiotics or probiotics were recommended to balance the intestinal microbiota and lower the risk of secondary infection due to bacterial translocation **(Xu *et al.*, 2020).** Other researchers have theorized that COVID-19 is linked to the gut microbiota, citing evidence of a secondary gut infection or dysfunction in patients with RTIs, maybe as a result of antibiotics that aren't selective for dangerous bacteria. This suggests that there is a gut–lung interaction, and that probiotics can modify symptoms to some extent, improving gastrointestinal symptoms while simultaneously preserving the respiratory system **(Gao *et al.*, 2020)**.

SARS-CoV-2 (Severe Acute Respiratory Syndrome Coronavirus 2) is a novel coronavirus that causes corona virus disease (COVID-19) in humans, a respiratory infection that was first detected in December 2019 in Wuhan, China. Coronavirus primarily affects the human immune system and weakens it **(Tewari *et al.*, 2021).** The zoonotic beta-corona virus family includes this SARS-related coronavirus **(Rodriguez-Morales *et al.*, 2020; Xie and Chen, 2019)**. SARS-CoV-2 is a single-stranded positive sense RNA virus with an enclosed genome **(Zhu *et al.*, 2020)**. Coronaviruses get their name from their crown-like forms and lengthy surface spikes **(Zu *et al.*, 2019)**. Humans, as well as other vertebrate reservoirs such as camels, bats, masked palm civets, mice, dogs, and cats, host coronaviruses **(Jiang *et al.*, 2020; Lu *et al.*, 2019)**. COVID-19 was thought to have been first housed by bats and then transmitted to people via wild animals; nevertheless, the virus spread later by human-to-human transmission **(Zu *et al.*, 2019)**.

Coronaviruses are known to cause respiratory, gastrointestinal (GI), and neurological problems. Except for SARS-CoV-1 and Middle East respiratory syndrome coronavirus (MERS-CoV), which are highly pathogenic viruses linked with severe infections and fatalities (**Jiang *et al.*, 2020**), the majority of coronaviruses found cause moderate human disease. SARS-CoV-1 first arose in China in 2002, while MERSCoV was discovered in Saudi Arabia in 2012 **(Jiang *et al.*, 2020)**. Despite being more transmissible than MERS-CoV and SARS-CoV-1, SARS-CoV-2 had a lower fatality rate **(Jiang *et al.*, 2020)**. COVID-19 is extremely pathogenic, and the number of people infected has risen dramatically over the world. As a result, WHO classified COVID-19 a pandemic, with at least 32.5 million cases and over 986.000 deaths confirmed through September 26, 2020. COVID-19 has a

1–14 day incubation period. COVID-19 has a wide range of clinical symptoms, from asymptomatic to severe disease. Patients who are asymptomatic can act as disease spreaders **(Jiang *et al.*, 2020)**. Fever, dry cough, shortness of breath, myalgia, and exhaustion are all common COVID-19 symptoms. COVID-19 **(Jiang *et al.*, 2020).** has also been linked to headache, rhinorrhea, sneezing, sore throat, loss of odor, and pneumonia. Gastrointestinal symptoms such as diarrhoea, nausea, vomiting, and abdominal discomfort are also unusual signs of the condition **(Zu *et al.*, 2019; Jiang *et al.*, 2020)**.

2. IMMUNOPATHOLOGY OF COVID-19 DISEASE

The impact of the coronavirus on the immune systems of humans. Because this virus primarily disrupts the immune system's equilibrium, people must maintain their immunity and strengthen their immune systems to combat coronavirus. Immunity is a state of resistance to invading pathogens (biotic and abiotic) and their detrimental effects on organisms **(Tewari *et al.*, 2020).**

The pathogenesis of COVID-19 is unknown, however reports from several nations suggest that the virus enters or invades host cells in the same way that SARS-COV does. The origin of SARS-CoV-2 is unknown; nevertheless, it is known that bats are the source of similar viruses and that human-to-human transmission is important in its pathogenesis. Following Spike protein interaction with its receptor, Spike protein enters target cells, Encapsulated and polyadenylated viral RNA encodes a variety of structural and non-structural polypeptide genes. Proteases that have chymotrypsin-like activity cleave these polyproteins. Despite the fact that transmembrane serine protease 2 (TMPRSS2) is the major protease associated with CoV activation and has been linked to SARS-CoV-2 activation, recent evidence from single cell RNA-sequencing (scRNA-seq) analysis shows that ACE2 and TMPRSS2 are not expressed in the same cell, implying that other proteases such as cathepsin B and L are involved in this process.

In general, pattern recognition receptors (PRRs) recognize invading pathogens including viruses **(Ben Addi *et al.*, 2008).** Viruses elicit several key host immune responses such as increasing the release of inflammatory factors, induction and maturation of dendritic cells (DCs) and increasing the synthesis of type I interferons (IFNs), which are important in limiting viral spread **(Ben Addi *et al.*, 2008)**. SARS-CoV-2 triggers both the innate and acquired immune responses. CD4 + T cells induce B cells to create virus-specific antibodies such as immunoglobulin (Ig) G and IgM, while CD8 + T cells destroy virus-infected cells directly. To aid other immune cells, T helper cells create pro-inflammatory cytokines and mediators. SARS-CoV-2 can thwart the immune system of the host by inhibiting T cell activities and triggering programmed cell death, such as apoptosis. In addition, complement factors such as C3a and C5a, as well as antibodies produced by the host, are crucial in countering viral infection **(Niu *et al.*, 2018).**

3. CONCLUSION:

Lympopenia and cytokine storms in the blood of people with severe sickness spread it mostly through the respiratory system. This suggests that immunological dysregulation could be a secondary effect of the virus's severe illness. This immunological characteristic's early detection could aid in the early diagnosis of people who would develop serious illness. Here's a look at the data on the immunological response to COVID-19 infection. The current study summarises what we now know about how immune dysregulation and altered cytokine networks contribute to the pathophysiology of COVID-19 patients.

REFERENCES

1. Amanat, F., & Krammer, F. (2020). SARS-CoV-2 vaccines: Status report. Immunity, 52(4), 583–589.
2. Auinger A, Riede L, Bothe G, Busch R, Gruenwald J. 2013. Yeast (1,3)–(1,6)-beta-glucan helps to maintain the body's defence against pathogens: a double-blind, randomized, placebo-controlled, multicentric study in healthy subjects. Eur J Nutr. 52(8):1913–1918.
3. Ben Addi, A., Lefort, A., Hua, X., Libert, F., Communi, D., Ledent, C., ... & Robaye, B. (2008, May). Modulation of murine dendritic cells function by adenine nucleotides and adenosine: involvement of the A2B receptor. In PURINERGIC SIGNALLING (Vol. 4, pp. S57-S58). VAN GODEWIJCKSTRAAT 30, 3311 GZ DORDRECHT, NETHERLANDS: SPRINGER.
4. Chan CKY, Tao J, Chan OS, Li H-B, Pang H. 2020. Preventing respiratory tract infections by symbiotic interventions: a systematic review and meta-analysis of randomized controlled trials. Adv Nutr. 11(4):979–988.

5. Conticini E, Frediani B, Caro D. 2020. Can atmospheric pollution be considered a co-factor in extremely high level of SARS-CoV-2 lethality in Northern Italy? Environ Pollut. 261:114465.
6. Gao QY, Chen YX, Fang JY. 2020. 2019 Novel corona virus infection and gastrointestinal tract. J Dig Dis. 21(3): 125–126.
7. Jiang, F. et al. Review of the clinical characteristics of Coronavirus Disease 2019 (COVID-19). J. Gen. Intern. Med. 35, 1545–1549 (2020).
8. Lee, P. I., Hu, Y. L., Chen, P. Y., Huang, Y. C. & Hsueh, P. R. Are children less susceptible to COVID-19? J. Microbiol. Immunol. Infect. 53, 371–372 (2020).
9. Li, G., Fan, Y.-H., Lai, Y.-N., Han, T.-T., Li, Z.-H., Zhou, P.-W., et al. (2020). Coronavirus infections and immune responses. Journal of Medical Virology, 92(4), 424–432.
10. Li, Q., Guan, X.-H., Wu, P., Wang, X.-Y., Zhou, L., Tong, Y.-Q., et al. (2020). Early transmission dynamics in Wuhan, China, of novel coronavirus-infected pneumonia. New England Journal of Medicine, 382(13), 1199–1207.
11. Liu, R., Hong, J., Xu, X., Feng, Q., Zhang, D.-Y., Gu, Y.-Y., et al. (2017). Gut microbiome and serum metabolome alterations in obesity and after weight-loss intervention. Nature Medicine, 23(7), 859–868.
12. Lu, C.-W., Liu, X.-F. & Jia, Z.-F. 2019-nCoV transmission through the ocular surface must not be ignored. Lancet 395, e39, https://doi.org/10.1016/S0140-6736(20) 30313-5 (2020).
13. Niu, P., Zhang, S., Zhou, P., Huang, B., Deng, Y., Qin, K., ... & Tan, W. (2018). Ultrapotent human neutralizing antibody repertoires against Middle East respiratory syndrome coronavirus from a recovered patient. The Journal of infectious diseases, 218(8), 1249-1260.
14. Tewari, S., David, J., & David, B. (2020). A critical review on immune-boosting therapeutic diet against Coronavirus (COVID-19). J Sci Technol, 5, 43-9.
15. Tewari, S.; David, J.; Nakhale, S.; David, B. Mucormycosis: Post COVID-19 Fungal Infection. Int. J. Curr. Microbiol. Appl. Sci. 2021, 10, 64–71.
16. Wang, D., Hu, B., Hu, C., Zhu, F.-F., Liu, X., Zhang, J., et al. (2020). Clinical characteristics of 138 hospitalized patients with 2019 novel coronavirus-infected pneumonia in Wuhan, China. Jama, 323(11), 1061–1069.
17. World Health Organization (WHO). Coronavirus disease (COVID-19) Pandemic. https://www.who.int/emergencies/diseases/novel-coronavirus-2019 (2020).
18. Xu K, Cai H, Shen Y, Ni Q, Chen Y, Hu S, Li J, Wang H,Yu L, Huang H, et al. 2020. Management of Corona Virus Disease-19 (COVID-19): the Zhejiang Experience. Jour Zhejiang Univ. 49(1):0
19. Xu, Z., Shi, L., Wang, Y., Zhang, J.-Y., Huang, L., Zhang, C., et al. (2020). Pathological findings of COVID-19 associated with acute respiratory distress syndrome. The Lancet Respiratory Medicine, 8(4), 420–422.