

NF-KB: A Key Transcription Factor in Cytokine Storm of Patients with COVID

Shrihari T G*

*Department of Oral Medicine and Oral Oncology, Krishna Devaraya College of Dental Sciences and Hospital, Bangalore -562157, Karnataka, India.

Received September 05, 2022; Accepted October 09, 2022; Published October 12, 2022

ABSTRACT

SARS- COVID2 is a current global pandemic infectious disease caused by corona virus affecting mankind worldwide. Surface antigenic glycoprotein present on the virus recognized by PRR(Pattern recognition receptors) belongs to TLR activate NF-KB a key transcription factor, deregulated NF-KB, a key transcription factor involved in activation of inflammatory mediators responsible for cytokine storm involved in lung damage, pulmonary thrombosis, pulmonary fibrosis, and severe acute respiratory distress syndrome, later leads to death. This article brief about the role of NF-KB, a key transcription factor in cytokine storm of patients with SARS-COVID 2.

Keywords: IL-1, IL-6, TNF- α , ikB, SARS, UPA, Mmp's

INTRODUCTION

SARS-COVID2 is a global pandemic infectious disease caused by corona virus has an affinity to bind to ACE receptors present on the lungs. NF-KB is a ubiquitous transcription factor present in cytosol of every cell. NF-KB a key transcription factor controls more than 500 genes. NF-KB a key transcription factor normally in an inactive state by I κ B (inhibitory kappa beta) factor, when activation degradation of I κ B occurs [1-6].

NF-KB: A KEY TRANSCRIPTION FACTOR IN CYTOKINE STORM

Activation of NF-KB by surface antigenic glycoprotein of corona virus results in NF-KB binding to DNA by shifting the NF-KB a key transcription factor from cytosol to nucleus leads to transcription of inflammatory mediators [7-9]. Deregulated NF-KB activation results in release of inflammatory mediators responsible for cytokine storm from chronic inflammatory cells such as neutrophils, macrophages, and mast cells release cytokines (IL-1, TNF- α , IL-6, TGF- β), free radicals (ROS, RNS), proteolytic enzymes (UPA, Mmp's 2, 9), chronic inflammation (IL-1, TNF- α , IL-6), angiogenesis (IL-8, COX-2, HIF-1 α), immune modulation by (IL10, IL-4, IL-5, IL-13) involved in lung damage, pulmonary fibrosis, pneumonia, pulmonary thrombosis, and Severe Acute respiratory distress syndrome (SARS) then later death. Cytokine storm in SARS-COVID patients is mainly by NF-KB key transcription factor activation is graded in to mild, moderate, and severe responsible for disease severity causes lung alveoli damage,

pneumonia, severe acute respiratory disease, acts as a therapeutic target and prognostic marker [10-16].

CONCLUSION

NF-KB, a key transcription factors a ubiquitous transcription factor activated by surface glycoprotein present on the corona virus. NF-KB, a key transcription factor activates inflammatory mediators such as cytokines, growth factors, and proteolytic enzymes involved in lung damage. Thorough understanding of NF-KB transcription factor activation and it's mechanisms of actions in patients with SARS-COVID2 helpful for therapeutic target and prognostic marker.

REFERENCES

1. Cascella M, Rajnik M, Aleem A, Dulebohn SC, Di Napoli R (2020) Features, Evaluation and Treatment Corona virus (COVID-19) [Updated 2020 May 18]. In: Stat Pearls [Internet]. Treasure Island (FL): Stat Pearls Publishing.

Corresponding author: Shrihari T G, Assistant Professor, Department of Oral Medicine and Oral Oncology, Krishna Devaraya College of Dental Sciences and Hospital, Bangalore -562157, Karnataka, India, Tel: +91 7892121331; E-mail: drshrihariomr@gmail.com

Citation: Shrihari TG. (2023) NF-KB: A Key Transcription Factor in Cytokine Storm of Patients with COVID. J Microbiol Microb Infect, 5(1): 157-158.

Copyright: ©2023 Shrihari TG. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

- options. Clin Immunol 215: 108448.
2. Felsenstein S, Herbert JA, McNamara PS, Hedrich CM (2020) COVID-19: Immunology and treatment
 3. Vabret N, Britton GJ, Gruber C, Hegde S, Kim J, et al. (2020) Immunology of COVID-19: Current State of the Science. Immunity 52(6): 910-941.
 4. Hui DSC, Zumla A (2019) Severe Acute Respiratory Syndrome: Historical, Epidemiologic, and Clinical Features. Infect Dis Clin North Am 33(4): 869-889.
 5. Clark A, Jit M, Warren-Gash C, Guthrie B, Wang HHX, et al. (2020) Centre for the Mathematical Modeling of Infectious Diseases COVID-19 working group. Global, regional, and national estimates of the population at increased risk of severe COVID-19 due to underlying health conditions in 2020: A modeling study. Lancet Glob Health 8(8): e1003-e1017.
 6. Mussbacher M, Salzmann M, Brostjan C, Hoesel B, Schoergenhofer C, et al. (2019) Cell Type-Specific Roles of NF- κ B Linking Inflammation and Thrombosis. Front Immunol 10: 85.
 7. Spinelli SL, Casey AE, Pollock SJ, Gertz JM, McMillan DH, et al. (2010) Platelets and megakaryocytes contain functional nuclear factor-kappaB. Arterioscler Thromb Vasc Biol 30(3): 591-598.
 8. Iba T, Levy JH (2018) Inflammation and thrombosis: roles of neutrophils, platelets and endothelial cells and their interactions in thrombus formation during sepsis. J Thromb Haemost 16(2): 231-241.
 9. Menter T, Haslbauer JD, Nienhold R, Savic S, Hopfer H, et al. (2020) Postmortem examination of COVID-19 patients reveals diffuse alveolar damage with severe capillary congestion and variegated findings in lungs and other organs suggesting vascular dysfunction. Histopathology 77(2): 198-209.
 10. Conti P, Ronconi G, Caraffa A, Gallenga CE, Ross R, et al. (2020) Induction of pro-inflammatory cytokines (IL-1 and IL-6) and lung inflammation by Coronavirus-19 (COVI-19 or SARS-CoV-2): Anti-inflammatory strategies. J Biol Regul Homeost Agents 34(2): 327-331.
 11. Ciceri F, Beretta L, Scandroglio AM, Colombo S, Landoni G, et al. (2020) Microvascular COVID-19 lung vessels obstructive thromboinflammatory syndrome (MicroCLOTS): An atypical acute respiratory distress syndrome working hypothesis. Crit Care Resusc 22(2): 95-97.
 12. Tian S, Hu W, Niu L, Liu H, Xu H, et al. (2020) Pulmonary Pathology of Early-Phase 2019 Novel Coronavirus (COVID-19) Pneumonia in Two Patients With Lung Cancer. J Thorac Oncol 15(5): 700-704.
 13. Song W, Gui M, Wang X, Xiang Y (2018) Cryo-EM structure of the SARS coronavirus spike glycoprotein in complex with its host cell receptor ACE2. PLoS Pathog 14(8): e1007236.
 14. Bauch CT, Lloyd-Smith JO, Coffee MP, Galvani AP (2005) Dynamically modeling SARS and other newly emerging respiratory illnesses: Past, present, and future. Epidemiology 16(6): 791-801.
 15. Chan JF, Kok KH, Zhu Z, Chu H, To KK, et al. (2020) Genomic characterization of the 2019 novel human-pathogenic coronavirus isolated from a patient with atypical pneumonia after visiting Wuhan. Emerg Microbes Infect 9(1): 221-236.
 16. Guo ZD, Wang ZY, Zhang SF, Li X, Li L, et al. (2020) Aerosol and Surface Distribution of Severe Acute Respiratory Syndrome Coronavirus 2 in Hospital Wards, Wuhan, China, 2020. Emerg Infect Dis 26(7): 1583-1591.