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Mini Review

Sars-Cov-2 Brain Permanent Damage due to Hypoxia a Chance of Symptoms Mitigating

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Abstract

Tool Like receptor a key role in cytokine storm during Sars-Co-2 immune response. Alia-amides are valuable and beneficial for permanent damage due to hypoxia. They can mitigate brain hypoxia symptoms. Tool Like receptor play a key role in cytokine storm, Sars-Co-2 immune response alike. Alia-amides can valuable and beneficial on neuroinflammation causes neurodegeneration and neuronal cell damage. The key role of the immune response appeared to be related to Tool-Like receptors since the early stages of the studies conducted on Sars-Co-2. Nowadays the robust evidence that emerged in literature seems to confirm more than what has been hypothesized. The key role of Tool-Like receptors acts by within the infection, causing a storm by cytokines such as interleukin-1 (IL-1), IL-6, and tumour necrosis factor-α, as well as type 1 interferon. The whole pathway causes internal organ damage resulting not only on long sequela but even on persistent tissue brain damages [1].

Keywords: Sars-Cov-2, Brain permanent damage, Hypoxia, PEA, Symptoms mitigating

Introduction

As study Alia-amides like Palmitoyl-ethanol-amide demonstrated the capability of Tool-Like receptor for aberrant tissue damage by its mediators. Alia-Amides properly and block Tool-Like pathways dependent tissue damage due to Coronavirus infection by the Tool-Like receptor mechanism disease related involving PPAR-α receptors [2,3]. How are Tool-Like receptors involved in the neuroinflammatory process? Toll-like receptors a key role in Sars-Co-2 pathogenesis. Toll-like receptors play a key role in the pathogenesis by the recognition of viral particles and activation of the innate immune system. Activation of TLR pathways leads to the secretion of pro-inflammatory cytokines, such as interleukin-1 (IL-1), IL-6, and tumour necrosis factor-α, as well as type 1 interferon. Different TLRs, like TLR2, TLR3, TLR4, TLR6, TLR7, TLR8, and TLR9 are potentially important in Sars-Co-2 infection [4].

Discussion

Alia-amides can be valuable as a beneficial therapeutic tool on neuronal damage. As it has been widely demonstrated in past research on PEA, OEA and AEA compounds play a key role as endogenous mediators of neurodegeneration due to neuroinflammation [3]. Is it possible that actual brain damage due to proinflammatory mediators can guarantee a barrier against actual brain damage and moderation of neurodegenerative disorders due to Sars-Co-2 pathogenesis. Punctually endosomal TLR, mainly TLR3, TLR7, TLR8, and membrane-bound TLR4 [4]. has a role in the

induction of cytokine storms. TLR7/8 recognizes the ssRNA SARS-COV-2 and when it replicates to dsRNA, it is recognized by TLR3 and drives the TRIF-mediated inflammatory signalling like NF-κB, and MAPK [5]. Such signalling leads to significant transcription and translation of pro-inflammatory genes, releasing inflammatory molecules into the systemic circulation, causing an imbalance in the system. This possible links between SARS-CoV-2 Infection and neurodegenerative disease, [6] by the key role of Toll-Like Receptor 4 in brain stress processes. It is known that the virus can activate mast cells through Toll-like receptors (TLRs) and increase inflammatory mediator expression. Furthermore, The Role of Toll-Like Receptors 4 play a key role linking SARS-CoV-2 Infection and in neurodegenerative disease like Parkinson's Disease [6]. Testimonials emerged in literature shows broad spectrum of neurological manifestations like presented bilateral lesions of the basal ganglia related to a severe acute respiratory distress syndrome in patients.

Conclusion

Passed research involved PEA in a "neuro-regenerative" mechanism called reactive gliosis by glia/toll-like receptor 4-dependent PPAR-α activated [3]. It would be now the chance to be far-sighted on SARS-CoV-2 infection's as well as nefarious long-term neurological effects and use this novel therapeutic approach against this terrible illness permanent damage and clinical consequences to obtain any mitigation.

Acknowledgement

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Conflict of Interest

None.

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