Journal of Down Syndrome & Chromosome Abnormalities

OPEN ACCESS Freely available online

Perspective

Innate Immune System and its Role in Treating Down Syndrome

Jyothi Nayak*

Department of Medical Genetics, Sanjay Gandhi Post Graduate Institute of Medical Sciences, Lucknow, Uttarpradesh, India

DESCRIPTION

Activation and inflammation of the innate immune system are associated with and may influence clinical outcomes in individuals with Down syndrome (DS), neurodegenerative illnesses including Alzheimer's Disease (AD), and normal ageing. Innate immune system activation and inflammation may contribute to or cause these illnesses in addition to acting as potential diagnostic indicators, which raises the possibility that effective treatments should aim to reduce their effects. Activating the innate immune system and inflammation, however, may actually be therapeutic, according to recent intervention studies using the innate immune system activator Granulocyte-Macrophage Colony-Stimulating Factor (GM-CSF) of DS, AD, and normal ageing as well as in an AD clinical trial. We analyze whether and when would be therapeutically advantageous to limit or encourage such activation during the disease process after taking into account data suggesting innate immune system activation and inflammation are associated with DS, AD, and normal ageing.

One in 700–1,000 live births worldwide are affected with Down syndrome (DS), the most common genetic cause of both Intellectual Disability (ID) and age-associated cognitive decline. DS is most frequently caused by triplication of human chromosome 21 (Hsa21). Since the APP gene is located on chromosome 21, its extra copy is primarily to blame for the fact that all people with DS develop AD brain pathology by the age of 40, including amyloid-(A) plaques and cerebral amyloid angiopathy. Amyloid Precursor Protein (APP) plays a significant role in the pathophysiology of Alzheimer's Disease (AD). Adults with DS also experience persistent neuroinflammation, oxidative stress, vascular abnormalities, neurofibrillary tangles of hyperphosphorylated tau, and other pathologies that are often seen in people with AD and other neurodegenerative disorders.

Despite strong epidemiological, pharmacological, and genetic evidence against the amyloid cascade hypothesis, other elements of brain physiology, particularly the innate immune system and neuroinflammation, have the ability to influence the AD pathogenic pathway. Based on the observation of aberrant glial cells around amyloid plaques, Alois Alzheimer was the first to propose a potential role for inflammation in AD. These preliminary indications were confirmed by the finding that particular inflammatory proteins, including the cytokine Interleukin-1 (IL-1) and the inflammation/acute-phase protein 1-Antichymotrypsin (ACT), were elevated in the AD brain and associated with amyloid plaques.

The activity of inflammation, a complicated multifactorial process that affects both the peripheral and the Central Nervous Systems (CNS), depends on the stage of the disease. Growing evidence indicates that other cells, such as astrocytes, neurons, oligo-dendrocytes, and pericytes, also play important roles in the innate immune system and neuroinflammation in the brain. Additionally, brain inflammation in age-associated AD varies from that in DS-associated AD. Accordingly, neuroinflammation may be crucial in the emergence of AD, but it is yet unclear what causes this clinical manifestation and how it relates to DS.

CONCLUSION

Both the CNS and the periphery are characterised by inflammation and innate immune system activation. Instead of claiming that inflammation only has negative effects, they suggest that, depending on the stage of the condition, both inhibition and activation of the innate immune system as well as neuroinflammation may be advantageous. The immune-modulating cytokine Granulocyte-Macrophage Colony-Stimulating Factor (GM-CSF), apolipoprotein E (apoE) inhibitors, and microglial depletion via drugs that target the Colony-Stimulating Factor-1 Receptor (CSF1R) will all be discussed as final therapeutic approaches for controlling neuroinflammation.

Correspondence to: Dr. Jyothi Nayak, Department of Medical Genetics, Sanjay Gandhi Post Graduate Institute of Medical Sciences, Lucknow, Uttarpradesh, India, E-mail: jyothinayak@gmail.com

Received: 02-Nov-2022, Manuscript No. JDSCA-22-20798; **Editor assigned:** 04-Nov-2022, Pre QC No. JDSCA-22-20798 (PQ); **Reviewed:** 18-Nov-2022, QC No. JDSCA-22-20798; **Revised:** 25-Nov-2022, Manuscript No. JDSCA-22-20798 (R); **Published:** 02-Dec-2022, DOI: 10.35248/2472-1115.22.08.211

Citation: Nayak J (2022) Innate Immune System and its Role in Treating Down Syndrome. J Down Syndr Chr Abnorm. 8:211

Copyright: © 2022 Nayak J. 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.