



Neuroimmunoactive biomaterial design

Maedeh Khodkam

Neuroscience Research Group (NRG), Universal Scientific Education and Research Network (USERN), Tehran, Iran

Correspondence to Maedeh Khodkam, Neuroscience Research Group (NRG), Universal Scientific Education and Research Network (USERN), Tehran, Iran

Email: Maedehkhodkam1379@gmail.com

Published online 20 June 2022

Cite this article as: Khodkam, M. Neuroimmunoactive biomaterial design. Neurology Letters, 2022; 1(1): 17-18. https://doi.org/10.52547/nl.1.1.17.

Dear Editor,

According to the mounting evidence, neuroinflammation and neuroimmune activation play an important role in the etiology of neurological disorders such as Alzheimer's disease, stroke, Parkinson's disease, and Multiple Sclerosis. To explore the neuroimmune activation properly, the first step is clarifying the immunity. The immune system is a distributed information-processing system that protects the body from infectious disease, injury, and toxic and allergenic substances and maintains the body's homeostasis (1). It consists of innate and adaptive immunity. Innate immunity is non-specific, has a rapid response, and includes physical agents, chemical components such as cytokines and chemokines, and inflammatory cells but adaptive immunity is specific and consists of antibodies and cells (antigen-specific T and B lymphocytes). Innate and adaptive responses usually work together to invade pathogens (2).

For a long time, the CNS was known as an immunologically privileged site, which under normal conditions, the immune system cannot be present as other parts of the body. But after years, this belief became invalid and now neuroimmune signaling is considered a critical component of neuronal processes underlying memory, emotion, and cognition. It was found that the CNS is actively involved in immunological phenomena under both physiological and pathological conditions and the brain is a highly immunologically active organ due to the CNS resident cells of the innate immune system, and also invading peripheral immune cells (3). Neuroinflammation is considered the infiltration of immune

cells into the damage site in response to the peripheral or central nervous system. The neuroimmune response in the brain is predominantly an innate immune response, although the adaptive immune system including T-cells is critical for normal neural function, cognition, and production of cytokines in the brain during illness (3, 4). However, we mention illness or injury as a triggering factor that activates the immune and neuroimmune systems, in the naïve homeostatic baseline, neuroimmune signaling or activity is not absent and it interacts with neurons and is responsible for the regulation of neural function and synaptic plasticity (3). Under pathological circumstances, during neuroimmune activation, the activation of endothelial cells, microglia, and astrocytes cause the production of cytokines, chemokines, and the expression of surface antigens that leads to the immune cascade without infiltration of immune cells to the site of injury and robust pathological sequelae. It was seen that glial activation, cell migration and trafficking, adhesion molecule expression, antigen presentation, and cytokine production, can be detected during the diseases of CNS such as neurodegenerative disorders (5-7). In sum, neuroimmune activation is commonly known as the microglia activation, or by rising in the level of expression of immune molecules, especially cytokines and chemokines (8, 9).

According to the role of the immune system in the pathogenesis of neurodegenerative diseases, targeting the immune reactions inside the brain may have enormous potential for treating these disorders. So biomaterials that can interact with the immune components, in the particular innate immune system, such as cytokines, chemokines, complement,



glia cells, and astrocytes that take part in the neurodegenerative disorders, can be a potential treatment (10-13).

Keywords: Neuroimmunoactive, biomaterials, neuroinflammation

Declaration

Funding

We do not have any financial support for this study.

Conflict of interest

The authors declare no conflict of interest regarding the publication of this paper.

Ethical approval

No need

Consent for publication

This manuscript has been approved for publication by all authors.

References

- 1. Hofmeyr SA, Forrest S, editors. Immunity by design: An artificial immune system. Proceedings of the 1st Annual Conference on Genetic and Evolutionary Computation-Volume 2; 1999: Citeseer.
- 2. Delves PJ, Roitt IM. The Immune System. New England Journal of Medicine. 2000;343(1):37-49.
- 3. Tchessalova D, Posillico CK, Tronson NC. Neuroimmune Activation Drives Multiple Brain States. Frontiers in Systems Neuroscience. 2018;12(39).

- 4. Kipnis J, Gadani S, Derecki NC. Pro-cognitive properties of T cells. Nature Reviews Immunology. 2012;12(9):663-9.
- 5. Colburn RW, DeLeo JA, Rickman AJ, Yeager MP, Kwon P, Hickey WF. Dissociation of microglial activation and neuropathic pain behaviors following peripheral nerve injury in the rat. J Neuroimmunol. 1997;79(2):163-75
- 6. El Khoury J. Neurodegeneration and the neuroimmune system. Nature medicine. 2010;16(12):1369-70.
- 7. Scheiblich H, Trombly M, Ramirez A, Heneka MT. Neuroimmune connections in aging and neurodegenerative diseases. Trends in immunology. 2020;41(4):300-12.
- 8. McCusker RH, Kelley KW. Immune–neural connections: how the immune system's response to infectious agents influences behavior. Journal of Experimental Biology. 2013;216(1):84-98.
- 9. Becher B, Spath S, Goverman J. Cytokine networks in neuroinflammation. Nature Reviews Immunology. 2017;17(1):49-59.
- 10. Tsui C, Koss K, Churchward MA, Todd KG. Biomaterials and glia: Progress on designs to modulate neuroinflammation. Acta Biomaterialia. 2019;83:13-28.
- 11. Ricklin D, Lambris JD. New milestones ahead in complement-targeted therapy. Seminars in Immunology. 2016;28(3):208-22.
- 12. Kaminska B, Gozdz A, Zawadzka M, Ellert-Miklaszewska A, Lipko M. MAPK Signal Transduction Underlying Brain Inflammation and Gliosis as Therapeutic Target. The Anatomical Record. 2009;292(12):1902-13.
- 13. Dai H, Navath RS, Balakrishnan B, Guru BR, Mishra MK, Romero R, et al. Intrinsic targeting of inflammatory cells in the brain by polyamidoamine dendrimers upon subarachnoid administration. Nanomedicine. 2010;5(9):1317-29.