REG.NO:D7635654-AFINJ JOHP-ISSN: 2348-7704

HEB JOHP

Journal of Hospital Pharmacy
An Official Publication of Bureau for Health & Education Status Upliftment
(Constitutionally Entitled as Health-Education, Bureau)

Primitive to Novel animal models of multiple sclerosis: Finding the gap between Pre-clinical and Clinical research

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Abstract

The general opinion about multiple sclerosis (MS) pathogenesis is abnormal (auto) immune responses, which may arise after infection(s), which are the source of the damaging inflammatory demyelination and neurodegeneration in the central nervous system (CNS), despite the fact that the underlying origin of MS remains unclear. Combining this theory with the limited supply of human brain tissue in the early stages of MS has resulted in the creation of organotypic brain slice cultures, human central nervous system cell cultures, and animal models for autoimmune, viral, and toxin-induced demyelination. An understanding of how environmental factors may cause inflammation, demyelination, and neurodegeneration in the central nervous system has been impacted by viral and autoimmune models, together known as experimental autoimmune encephalomyelitis (EAE). These models also have a significant impact on the development of treatments that address MS's inflammatory aspects. Cuprizone and lysolecithin-induced models of toxicity-induced demyelination provide a superior platform for studying demyelination and remyelination in the absence of overt inflammation. More accurate models that account for axonal and neuronal damage are required because of the paradigm shift in MS from an autoimmune myelin depletion to a neurodegenerative disease. Therefore, these models have served as the primary foundation for the development of neuroprotective therapies. Using the available in vivo and in vitro experimental models, the pathogenic processes and mechanisms responsible for inflammation, demyelination, and neuronal degeneration, as well as remyelination and repair in MS, are vividly reviewed to give the research on MS therapies the right direction.

Keywords: Neurodegeneration, Demyelination, Cuprizone, Encephalomyelitis

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Website: http://www.journalofhospitalpharmacy.in	
Received on 24/03/2025	
Accepted on 09/04/2025 © HEB All rights reserved	