

Cell replacement therapies to promote remyelination in a viral model of demyelination.

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Public Summary:

Certain viruses are able to infect the brain and cause disease. As such, viruses have been considered as potentially important as a "triggering agent" in initiating the human demyelinating disease multiple sclerosis (MS). Therefore, viral models of demyelination are very important by allowing a further understanding of mechanisms that contribute to neuroinflammation and demyelination. In addition, viral models of demyelination offer an opportunity to investigate novel therapeutic strategies that can be developed to initiate remyelination. This review article highlights recent advances within the field focusing on the use of stem cells to promote remyelination in viral models of MS.

Scientific Abstract:

Persistent infection of the central nervous system (CNS) of mice with the neuroadapted JHM strain of mouse hepatitis (MHV) is characterized by ongoing demyelination mediated by inflammatory T cells and macrophages that is similar both clinically and histologically with the human demyelinating disease multiple sclerosis (MS). Although extensive demyelination occurs in mice persistently infected with MHV there is only limited remyelination. Therefore, the MHV model of demyelination is a relevant model for studying disease and evaluating therapeutic approaches to protect cells of the oligodendrocyte lineage and promote remyelination. This concept is further highlighted as the etiology of MS remains enigmatic, but viruses have long been considered as potential triggering agents in initiating and/or maintaining MS symptoms. As such, understanding mechanisms associated with promoting repair within the CNS in the context of a persistent viral infection is critical given the possible viral etiology of MS. This review focuses on recent studies using either mouse neural stem cells (NSCs) or human oligodendrocyte progenitor cells (OPCs) derived from human embryonic stem cell (hESC) to promote remyelination in mice persistently infected with MHV. In addition, the potential role for chemokines in positional migration of transplanted cells is addressed.

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