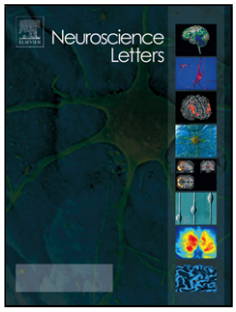

Title: Stroke in CNS White Matter: Models and Mechanisms

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Neuroscience letters – mini review
Title: Stroke in CNS White Matter: Models and Mechanisms
6-8 pages

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Highlights

- White matter stroke is a prominent stroke subtype, the second leading cause of dementia (vascular dementia) and commonly co-occurs with Alzheimer's disease ('mixed dementia')
- Unlike similar sized white matter lesions in multiple sclerosis and animal models of multiple sclerosis, there is no cellular repair process in white matter stroke
- Several animal models of white matter exist, each with strengths and limitations
- Recent studies have defined the effects of age and of specific molecular systems on outcomes in white matter stroke

Introduction/abstract

Subcortical white matter stroke (WMS) is demarcated by the continuous formation of small ischemic lesions within white matter tracts of the central nervous system. WMS is prevalent in older adults and stands as the second leading cause of dementia. In a startling statistic, most human beings will present with WMS by the age of 80 (de Leeuw et al., 2001). Early infarctions in human beings are asymptomatic, hence they are part of a category of "silent stroke". However, WMS is progressive with both an increase in infarction size and amount throughout subcortical white matter of the central nervous system, resulting in cognitive and motor dysfunction. Pathological analyses of post mortem brain tissue samples from WMS patients reveal the disruption of white matter architecture as a chief hallmark of infarction. Furthermore, the recent development of reliable mouse models of WMS has provided corroborating evidence of the centrality of the central nervous system's limited capacity to remyelinate following WMS. Taken together, these data provide an impetus to investigate the pathophysiological mechanisms that underpin demyelination and remyelination in the central nervous system following ischemic injury. This review addresses the current understanding of myelin biology and oligodendrocyte biology, human WMS etiology, the mechanisms of remyelination failure in mouse models of WMS, and the development of potential therapeutic strategies founded upon recent advances in our understanding of myelin plasticity.