Correlates of cognitive deficits in the mouse model of Multiple Sclerosis

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Abstract:

Multiple sclerosis (MS) is a demyelinating and neurodegenerative disease of the central nervous system leading to different types of motor, neuropsychiatric and cognitive problems. In MS patients, cognitive deficits correlate poorly with inflammatory activity, being better explained by pathological processes in the grey matter, like demyelination and consequent neuronal damage. The most widely used animal model of MS is the experimental autoimmune encephalomyelitis in the mouse, but data on their cognitive performance and its underpinnings are lacking. In the present work, we characterized cognitive deficits in the EAE mouse model of multiple sclerosis and explores its underpinnings. In a pattern similar to that of patients with MS, animals with EAE display both memory and executive function deficits. Interestingly, cognitive performance in the mouse model seems to be ascribed more to demyelination and damage to the white matter tracts than to changes in neuronal structure or integrity, which appear relatively unaffected. More importantly, we have also shown that such cognitive deficits can be prevented/ameliorated by reducing brain inflammatory infiltrates.