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The Role of BATF2 in Experimental Autoimmune Encephalomyelitis and Multiple Sclerosis

Maria Habean

Rachel Tinkey

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Habean, Maria; Tinkey, Rachel; Williams, Jessica

Department of Neuroscience, Lerner Research Institute, Cleveland Clinic, Cleveland, Ohio

Astrocytes play an active role in lesion formation in multiple sclerosis (MS) and the murine model, experimental autoimmune encephalomyelitis (EAE). In EAE and MS, activated astrocytes release cytokines and chemokines that lead to an upregulation in BATF2 expression, a transcription factor previously shown to be upregulated during acute MS and EAE. BATF2 regulates infiltrating immune cells in the central nervous system; therefore, the increased expression of BATF2 by astrocytes may lead to more secure lesions and exacerbated clinical outcomes. Immunohistochemistry was used to characterize lesion size, total BATF2 expression, and the types of infiltrating cells found in the lesion. BATF2 expression was upregulated during acute EAE compared to chronic EAE. The highest proportion of cells expressing BATF2 during acute and chronic EAE was astrocytes, with myeloid lineage cells being the second largest in acute and the fewest cells expressing BATF2 were T cells and B cells in both acute and chronic. These data suggest that BATF2 expression plays a role in astrocyte response to lesion formation in EAE and that BATF2 may represent a liable therapeutic target in MS.