Olfactory deficits in mice with experimental autoimmune encepharomyelititis, a model of human multiple sclerosis

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Olfactory deficits are commonly recognized in a variety of human neuro-inflammatory diseases including multiple sclerosis (MS). The relationship between autoimmune CNS disease multiple sclerosis and olfactory deficits is under debate. To address this question in human MS, we induced experimental autoimmune encephalomyelitis (EAE) in mice, as an animal model of human MS, and compared the behavioral performance of food searching, and neuropathologically examined olfactory bulbs which are one of central tissues of odor processing. Neuro-inflammatory lesions including the infiltration of inflammatory cells and activation of glial cells were found in the olfactory bulbs of EAE-affected mice, as do in the spinal cords. Some inflammatory cells were found along the olfactory nerves and in the submucosa of olfactory mucosa. Analysis of differentially expressed genes revealed that olfaction related genes including olfactory marker protein were significantly down-regulated in the olfactory bulbs of EAE. Behaviorally, the searching time for a bait pellet was significantly delayed in EAE mice. These findings all together suggest that neuro-inflammation in the olfactory bulbs in EAE-mice would be related with olfactory deficits as far as mice EAE is concerned. This research was supported by the National Research Foundation of Korea (Grant number: NRF-2017R1A2B4012487).