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Poster Presentation

Obesity-Induced Neuroinflammation: Focus on Hypothalamic Inflammation

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Abstract

Obesity is a Health issue around the world. Obesity is not limited to body weight, generally associated with low grade inflammation and with a cluster of disorders generally referred to as “metabolic syndrome”. Regarding obesity and relapse, long-term concentration was set on the hypothalamus. Most recently, obesity- Originated neuroinflammation has been shown to affect other brain structures such as the hippocampus, cortex, brainstem, or amygdala. Additionally, obesity is accompanied by an increase in central disturbances such as depression and cognitive impairment. The hypothalamus is a key brain region in the regulation of energy balance. The hypothalamus is a key brain region in regulating energy balance. Especially controls food intake and both energy storage and expenditure through integration of humoral, neural and nutrient-related signals. The hypothalamus cells and glial cells act jointly to orchestrate. We discuss the effects and mechanisms of obesity- originated neuroinflammation, with a specific emphasis on extra-hypothalamic structures, as well as the consequences of neuroinflammation for some cerebral functions. Therefore, the existence of a causal link between hypothalamic inflammation and deregulations of feeding behavior, such as involuntary weight-loss or obesity, has been recommended. Among the inflammatory mediators that could induce deregulations of hypothalamic control of the energy balance, chemokines represent interesting candidates. Chemokines, primarily known for their chemical attraction role of immune cells to the inflamed site, have also been recommended capable of neuromodulation.

Keywords: Obesity, Neuroinflammation, Hypothalamus

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