Microglia Cell, Major Player in the Central Nervous System Inflammation

Masoud Azarakhsh1, Farshid Hamidi*, Hadi Mohebalian2

1Department of Basic Sciences, Faculty of Veterinary Medicine, Ferdowsi University of Mashhad, Mashhad, Iran
2Department of Pathobiology, Faculty of Veterinary Medicine, Ferdowsi University of Mashhad, Mashhad, Iran

Abstract

Inflammation, a self-defensive reaction against various pathogenic stimuli, may become harmful self-damaging process. Increasing evidence has linked chronic inflammation to a number of neurodegenerative disorders including alzheimer’s disease (AD), parkinson’s disease (PD), and multiple sclerosis (MS). In the central nervous system, microglia, the resident innate immune cells play major role in the inflammatory process. Although they form the first line of defense for the neural parenchyma, uncontrolled activation of microglia may directly toxic to neurons by releasing various substances such as inflammatory cytokines (HMGB1, IL-1β, TNF-α, IL-6), NO, PGE and superoxide. Our recent study demonstrated that activated microglia, example BV2 cell, phagocytose not only damaged cell debris but also neighboring intact cells. These cells originated from yolk sac and fetal live in embryonic stage and after birth from bone marrow. Microglia bears some kinds of pattern recognition receptors (PRR) including TLR4 that can recognize pathogen associated molecular pattern (PAMP) and damage associated molecular pattern (DAMP). One of the most important items for PAMP is LPS which included in cell wall of bacteria especially gram negative bacteria. Moreover, LPS can be used by researchers in order to induction of inflammatory situation. It further supports their active participation in self-perpetuating neuronal damaging cycles. Besides, these interesting cells bear mannose and scavenger receptor for phagocytosis.

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*Corresponding Author: Farshid Hamidi
E-mail: masoud.a.1369@gmail.com