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Editorial

# Microglia the Claims and the Acusations

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Microglia are ubiquitous throughout the brain's parenchyma and perform crucial functions in regulating the brain's internal environment to maintain homeostasis [1]. In adults, parenchymal microglia originate from the yolk sac and are remarkably settled without significant turnover from outside, whereas perivascular microglia, a subtype, are regularly replaced from the bone marrow [2].

Under physiological settings, microglia have a unique morphology consisting of a tiny soma with elongated, branching, and highly active processes, which enables scanning of the surrounding CNS area and surveillance [3]. Microglia have many functions in the nervous system as; neuronal survival promotion and the generation and maintenance of other neural cells. In addition, they enhance synaptic generation, myelination regulation, debris clearance, trophic factor release, and memory formation to enhance learning [1]. Moreover, microglia are typically the first cells to be activated to carry out several well-established tasks, including pathogen identification, inflammatory responses, and phagocytosis when neuronal damage occurs [4,5]. In contrast to rodents, the human white matter has a higher microglia density than grey matter [6,7].

Additionally, compared to microglia in grey matter, those in white matter are more elastic and viscous [8]. Moreover, microglia are more prevalent in the cerebral cortex and hippocampus than in the cerebellum and brain stem. Hence, the regional variations in microglial quantity and morphology show that the local environment controls microglial distribution and morphology [9].

Several pieces of research revealed that the integrity of the white matter is necessary for cognitive, sensory, and motor activities throughout the lifespan, from development to aging. Furthermore, neurodegenerative disorders such as Alzheimer's dementia (AD) and viral encephalitis are detected in myelin abnormalities. These findings suggest a strong connection between white matter integrity and neuronal health and function [10,11]. In the physiological states, microglia are known to be resting or ramified cells whose branches are tiny, and the cell body exhibits few markers. However, upon stimulation with cellular debris or cell mediators like adenosine triphosphate, these tiny cells show morphologic change to the form of ameboid cells capable of phagocytosing such debris, similarly to how macrophages do. The activated microglia exhibit pathogen-associated molecular pattern (PAMP) receptors, also known as Toll-like receptors. Ten TLRs (TLR1 through TLR10) have been discovered in humans [9].

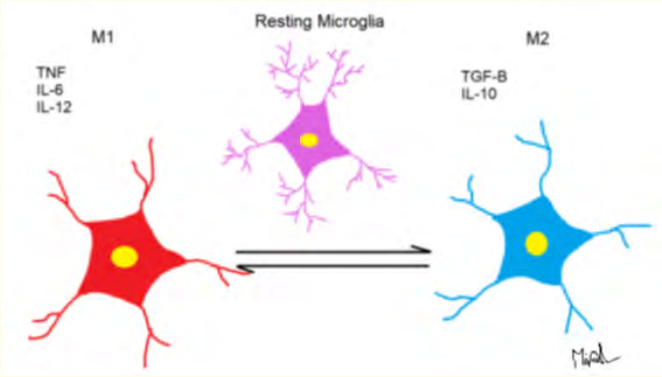


**Figure 1:** Shows the polarization of microglia.

Neurodegenerative illnesses such as Alzheimer's disease (AD), Parkinson's disease (PD), and multiple sclerosis (MS) typically display neuroinflammation driven by microglia. Consequently, microglia can be categorized as either classical (M1) or alternative (M2), with a continuum of intermediate phenotypes between M1 and M2 and the capacity to shift between phenotypes. M1 microglia release neurotoxic proinflammatory mediators such as interleukins 6, interleukins 12, and tumor necrosis factor, whereas M2 microglia release neuroprotective anti-inflammatory mediators such as interleukin 10 and transforming growth factor beta [12] as shown in figure 1. In the pathophysiology of neurodegenerative disorders, the equilibrium between microglia M1/M2 polarization provides a possible treatment opportunity [13].

## Bibliography

1. Prinz M., *et al*. "Microglia Biology: One Century of Evolving Concepts". *Cell* 179 (2019): 292-311.

2. Gomez-Perdiguero., *et al*. "Tissue-resident macrophages originate from yolk-sac-derived erythro-myeloid progenitors". *Nature* 518 (2015): 547-551.

3. Kierdorf K and Prinz M. "Microglia in steady state". *Journal of Clinical Investigation* 127 (2017): 3201-3209.

4. Kettenmann H., *et al*. "Physiology of Microglia". *Physiology Review* 91 (2011): 461-553.

5. Haley MJ., *et al*. "Microglial Priming as Trained Immunity in the Brain". *Neuroscience* 405 (2019): 47-54.

6. De Biase LM and Bonci A. "Region-specific phenotypes of microglia: the role of local regulatory cues". *Neuroscientist* 25 (2019): 314-333.

7. Askew K., *et al*. "Coupled proliferation and apoptosis maintain the rapid turnover of microglia in the adult brain". *Cell Report* 18 (2017): 391-405.

8. Van Wageningen TA., *et al*. "Viscoelastic properties of white and gray matter-derived microglia differentiate upon treatment with lipopolysaccharide but not upon treatment with myelin". *Journal of Neuroinflammation* 18 (2021): 83.

9. Bruce D. "Trapp and Karl Herrup. "Youmans and Winn Neurological Surgery". Elsevier, 7th edition, 2017. Chapter 48 (2017): e326-e346

10. Wang F., *et al*. "Myelin degeneration and diminished myelin renewal contribute to age-related deficits in memory". *Nature Neuroscience* 23 (2020): 481-486.

11. McKenzie IA., *et al*. "Motor skill learning requires active central myelination". *Science* 346 (2014): 318-322.

12. Zhang B., *et al*. "Targeting MAPK Pathways by Naringenin Modulates Microglia M1/M2 Polarization in Lipopolysaccharide-Stimulated Cultures". *Frontiers in Cell Neuroscience* 12 (2018): 531.

13. Song G J., *et al*. "A novel small-molecule agonist of PPAR-γ potentiates an anti-inflammatory M2 glial phenotype". *Neuropharmacology* 109 (2016): 159-169.