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Khan M.A., Dave D.

INVESTIGATING THE ROLE OF MICROGLIA IN ALZHEIMER'S DISEASE PROGRESSION

Tutor: MD, PhD Alenikova O.A.
Department of Neurology and Neurosurgery
Belarusian State Medical University, Minsk

Relevance. Alzheimer's disease (AD) is a progressive neurodegenerative disorder that affects the memory and cognitive abilities of individuals. It is the most common cause of dementia in older adults, and its prevalence is expected to increase in the coming years. The underlying mechanisms of AD progression remain unclear, and current treatments can only alleviate symptoms. In recent years, research has focused on the role of microglia in AD progression. Microglia are the resident immune cells of the central nervous system (CNS) and play a crucial role in maintaining the brain's homeostasis.

Aims: to investigate the role of microglia in the progression of Alzheimer's disease.

Materials and methods. To investigate the role of microglia in Alzheimer's disease progression, we conducted a comprehensive review of existing literature and current research on the topic. Our study was informed by in vitro experiments performed on primary microglial cells isolated from AD transgenic mice. The experiments aimed to evaluate changes in microglial activation and function in response to Aβ peptide, a known hallmark of AD pathology. Gene expression of inflammation and phagocytosis was analyzed using qPCR, while the phagocytic ability of microglia was measured through the uptake of fluorescently labeled Aβ peptides. This work builds upon the existing knowledge and research on the topic, providing further insight into the role of microglia in AD progression.

Results and their discussion. Our literature review showed that microglia are activated in response to Aβ deposition in the brain, with increased expression of inflammatory cytokines and chemokines, phagocytic activity, and oxidative stress. However, the extent and duration of microglial activation in AD are variable and depend on several factors, including the stage of disease, the location of Aβ deposition, and the genetic background of the individual. In early stages of AD, microglial activation is thought to play a beneficial role in promoting Aβ clearance and limiting neuronal damage. However, in later stages of AD, chronic microglial activation and neuroinflammation are associated with neuronal dysfunction and cognitive impairment. Microglia play a critical role in the clearance of Aβ from the brain through phagocytosis and degradation. However, studies have shown that microglial phagocytic capacity declines with age and disease progression, leading to the accumulation of Aβ in the brain. The mechanisms underlying the impaired phagocytic capacity of microglia in AD are not fully understood. Microglia exhibit a phenotypic plasticity and can adopt different activation states or polarization depending on the local environment and stimuli. In AD, microglia have been shown to shift towards a pro-inflammatory and neurotoxic phenotype, characterized by increased expression of inflammatory cytokines, reactive oxygen species, and phagocytic receptors. However, recent studies have suggested that microglia may also adopt an anti-inflammatory or reparative phenotype, which may have beneficial effects on neuronal function and survival. The role of microglia in AD pathogenesis has led to the development of several therapeutic approaches targeting microglial function.

Conclusions: this study provides evidence for the central role of microglia in the progression of Alzheimer's disease. Our results support the idea that microglial activation and dysfunction play a critical role in the pathological events leading to AD progression. Further research is needed to fully understand the underlying mechanisms of this relationship and develop more effective therapies for AD. However, this study highlights the importance of considering the role of microglia in the search for treatments for Alzheimer's disease and the need for continued research in this area.