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## Neuroinflammation and Neurodegenerative disease

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Dear Editor,

Age-related neurodegenerative diseases refer to the gradual and oncoming loss of neurons in the central nervous system (CNS) (1). More than 7 million Americans are involved with neurodegenerative diseases. In addition to the importance of addressing these diseases in terms of the increasing numbers of patients, the costs incurred for these diseases also make the matter more significant; More than 500 billion dollars is spent annually on neurodegenerative diseases' medical issues in the US (2).

Neurodegenerative diseases are characterized by pathological protein accumulation. Each neurodegenerative disease describes by a particular protein; In comparison to neural inclusions present in viral infections the protein is alien, these protein accumulations are made up of inherent neuronal proteins and other cellular parts (1). Amyloid- $\beta$ , prion protein, tau,  $\alpha$ -synuclein, TAR-DNA-binding protein 43 kDa, and fused-in sarcoma protein are the most common proteins that are getting involved in neurodegeneration (3).

Over time, these accumulations of protein propagate to white matter fibers and lead to brain dysfunction. Also, the body's inflammatory system is affected by these aggregations' toxicity. So, Selective neuronal loss occurs in the brain and in peripheral organs that cause cognitive and behavioral deficits (1, 4, 5).

Aging is a leading risk factor for the incidence and progression of neurodegenerative disorders. During the aging process, cellular and functional alterations affect the brain. Normal aging is accompanied by cellular senescence, which is caused by irreversible DNA damage. As people get older, senescent

cells increase in number and evidence indicates that their aggregation may impart to the pathogenesis of age-related diseases such as neurodegenerative diseases (6). Some of the most common neurodegenerative diseases are Alzheimer's disease (AD), Parkinson's disease (PD), Multiple Sclerosis (MS), and Amyotrophic lateral sclerosis (ALS) (1-4).

The actualization of therapies produced in preclinical cases to patients in the clinic is one of the major challenges in the neuroscience field. Contrary to multiple compounds being formulated in preclinical models, no successful disease-improving drugs are existing clinically for neurodegenerative disorders. Various reasons can be introduced for this inability but one of the most significant reasons is the failure to define reliable medication targets for these diseases (7).

The brain is an immunologically privileged organ since the peripheral immune cells are limited to penetrating the blood-brain barrier. But the glial cells consisting of microglia and astrocytes, are the primary constituents of a dedicated neuroimmune system (8, 9). The glial cells have roles in the production of pro-and anti-inflammatory and cooperate in different activities under normal and pathological conditions, including phagocytosis, steroid release, cytokine release, free radical reduction, and cellular repair. The activity of glia cells may lead to the damage of healthy neurons, synaptic dysfunction, loss of synapses, and neuronal death (9). So, an imbalance between proinflammatory and reparatory functions of neuroimmune cells can contribute to CNS injury. As has been mentioned above, neuroinflammation and activation of the neuroimmune cells including microglia and astrocytes have been suggested to have a role in the pathology of several neurodegenerative diseases (10). According to the

matter of the major role of inflammation in neurodegenerative diseases, modulating neuroinflammation can be a potential treatment target.

**Keywords:** Neurodegenerative disease, neuroinflammation, Alzheimer's disease, Multiple sclerosis

### Declaration

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### Conflict of interest

The authors declare no conflict of interest regarding the publication of this paper.

### Ethical approval

No need

### Consent for publication

This manuscript has been approved for publication by all authors.

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