

In Alzheimer's Disease, a protective signal is sent between the brain's supporting cells.

Shawn Kruger*

Editorial Office, Journal of Neuroinformatics and Neuroimaging, London, United Kingdom

Accepted January 18, 2021

Commentary

Alzheimer's Disease (AD) is a serious and common neurodegenerative disorder in which brain cells and synaptic connections between neuronal cells are lost, resulting in progressive cognitive deterioration. The existence of disease associated aggregates of diverse proteins in the brain, such as plaques formed of the protein amyloid-(A) and neurofibrillary tangles produced of the protein tau, is one of the hallmarks of Alzheimer disease. The most frequent kind of dementia in elderly adults is Alzheimer's Disease (AD) [1].

Dementia is a brain disease that severely limits a person's capacity to do daily tasks. The onset of AD is gradual. The regions of the brain that control cognition, memory, and language are the first to be affected. People with Alzheimer's disease may have problems recalling recent events or names of people they know [2]. Mild Cognitive Impairment (MCI), a related issue, produces more memory impairments than typical for adults of the same age. Many persons with MCI will acquire AD, but not all. Alzheimer's disease was first identified in 1906 by Alois Alzheimer, who used criteria such as gradual memory loss, disorientation, and pathology indicators to identify it (senile plaques and neurofibrillary tangles).

Symptoms of Alzheimer's disease worsen over time. Alzheimer's disease is characterised by the shrinking of the cerebral cortex and the medial temporal lobe, as well as the swelling of the brain ventricles. It's possible that family members will go unnoticed [3]. They may have difficulty communicating, reading, or writing. Disturbances in memory and language, as well as visuospatial orientation and higher executive function, are all clinical indications of Alzheimer's disease. Personality changes, impaired judgement ability, wandering, psychosis, mood disturbance, agitation, and sleep disorders are examples of noncognitive alterations. After the age of 60, Alzheimer's disease commonly sets in. As you become older, the risk increases. If a family member has experienced the condition, your chances are even higher. There is no cure for the condition. For a brief time, though, some medications may help keep symptoms from getting worse.

Within the brain, a network of interactions exists between cells, ensuring brain homeostasis and functional plasticity. Astrocytes, oligodendrocytes, and microglia are among the cells involved, in addition to neurons. In Alzheimer's disease, a protective

signal is sent between the brain's supporting cells. Monocytes and lymphocytes, which are present in the peripheral immune system, have been discovered to play a crucial role in keeping the brain healthy and assisting in its restoration [4]. The various immune-specific mechanisms of cellular communication among cells within the mammalian brain, as well as their crosstalk with the periphery in both health and disease, are described here. We also believe that therapies aimed at strengthening the peripheral immune response can help to restore the brain-immune system balance and rewire their communication in order to treat chronic neurodegenerative disorders. Neurodegenerative patterns are heterogeneous due to genetic diversity and varied environmental exposures [5]. Clinically, finding and identifying these patterns (clusters) using advanced computer aided tools and thereby putting the participants into more homogeneous groups can be beneficial.

References

1. Price TO, Eranki V, Banks WA, et al. Topiramate treatment protects blood-brain barrier pericytes from hyperglycemia-induced oxidative damage in diabetic mice. *Endocrinology*. 2012;153:362-372.
2. Reese TS, Karnovsky MJ. Fine structural localization of a blood-brain barrier to exogenous peroxidase. *J Cell Biol*. 1967;34:207-217.
3. Rhea EM, Rask MC, Banks WA. Insulin transport across the blood-brain barrier can occur independently of the insulin receptor. *J Physiol*. 2018;596:4753-4765.
4. Sagare AP, Bell RD, Zhao Z, et al. Pericyte loss influences Alzheimer-like neurodegeneration in mice. *Nat Commun*. 2013;4:2932.
5. May AA, Bedel ND, Shen L, et al. Estrogen and insulin transport through the blood-brain barrier. *Physiol Behav*. 2016;163:312-321.

*Correspondence to:

Shawn Kruger
Editorial Office
Journal of Neuroinformatics and Neuroimaging
London
United Kingdom
E-mail: i_kruger51@gmail.com