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The Role of Glial Cells in Brain Aging: Mechanisms of Cognitive Protection and Repair

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Abstract
The concept of cognitive reserve was developed to elucidate the distinction between the clinical and intellectual consequences of brain injury and the objective level of pathology. The structural (brain reserve) and functional (brain maintenance, resilience, and compensation) components of neural tissue constitute the cognitive reserve. These components reflect exposome-driven lifelong plasticity, which determines the brain's resilience to aging and disease. Adaptive alterations in neurons and brain networks constituted the primary focus of this concept's molecular foundation. We advocate for a more comprehensive perspective by presenting evidence that neuroglia play a significant role in defining the cognitive reserve through homeostatic, neuroprotective, and neurodegenerative processes. The development of the brain connectome and synaptically connected neuronal circuits, which determine cognitive reserve, depends on neuroglia throughout life. While neuroglia's regenerative capacities are essential for brain compensation in disease, neuroglial homeostatic and protective physiological responses characterize brain maintenance and resilience. An innovative strategy to extend cognitive lifespan may be to target neuroglia.

Keywords: Cognitive reserve, neuroglia, astrocytes, oligodendrocytes, microglia, brain resilience, brain maintenance, brain compensation, neuroplasticity, ageing, neuroprotection

Introduction

Increasing crowded population, the stress situation, plastic pollution, and environmental damage amplify frequencies of many maladies including neuroglia like Alzheimer diseases or dementia affected 55 million people in the world (Taddei & Duff 2025).

It is widely acknowledged that the functional consequences of brain damage vary substantially across individuals and are not strictly proportional to the extent of anatomical injury. Patients with identical structural brain lesions often exhibit markedly different neurological and cognitive outcomes (Goodglass et al., 1979). This variability is observed across a range of etiologies, including autoimmune disorders, systemic and localized infections, mechanical trauma, ischemia or stroke, and other pathologies affecting the central nervous system (CNS). Likewise, susceptibility to chronic conditions—such as age-related neurodegeneration and stress-induced psychiatric disorders—also shows significant interindividual variability.

At the turn of the 21st century, Yaakov Stern introduced the concept of cognitive reserve, which addresses the discrepancy between the clinical manifestations and the underlying neuropathology of brain injury. This framework posits that structural brain damage does not uniformly translate into cognitive decline, due to differences in the brain's capacity for functional compensation.

Alzheimer's disease (AD), the most prevalent form of dementia, exemplifies this variability. While AD pathogenesis is driven by a combination of genetic predispositions and biological mechanisms, environmental exposures and life-course factors significantly influence disease progression and cognitive deterioration. Early investigations suggested a moderate correlation between post-mortem amyloid plaque burden and premortem cognitive decline. However, these same studies documented individuals with extensive plaque deposition who maintained intact memory and cognitive performance (Love et al., 2004). A notable study of individuals aged 70–103 found that nearly one-third of cognitively intact participants met full neuropathological criteria for AD upon autopsy, underscoring the brain's differential capacity to tolerate pathological insult.

These findings highlight the role of both genetic and environmental factors in shaping brain development and resilience. Cognitive reserve is bolstered by factors such as education,

intellectual stimulation, physical activity, and social engagement—each promoting neuroplasticity and systemic resilience. Over time, the cumulative impact of diseases and psychosocial stressors increases the pathological burden, reducing the brain's ability to withstand future insults (De Kloet et al., 2005). Life experiences thus play a dual role: they may either enhance or diminish cognitive reserve, ultimately modulating the brain's vulnerability to neurodegenerative processes.

The cognitive reserve's intricacy

The brain's exceptional capacity to sustain cognitive function beyond the expected decline associated with aging or neurodegenerative disease is defined as "cognitive reserve." The history of how each individual brain has interacted with environmental factors throughout life—known as exposomes—is at the heart of cognitive reserve (Finch et al., 2019). These interactions can have either positive effects, such as lifelong learning and plasticity, or negative effects, like the buildup of pathogenic alterations. The genes that encode each person's brain also have an impact on these relationships. The closest relationship exists between these systemic and nerve tissue-specific alterations.

Brain reserve: an inactive idea

Brain reserve—previously referred to as neural or functional reserve—was initially conceptualized from an anatomical perspective, defined by the structural attributes of an individual's brain at the time of injury. Early theories posited that brain reserve was determined by metrics such as overall brain volume, neuronal count, and synaptic density. Notably, individuals with larger brains have been shown to exhibit a lower risk of developing dementia (Allen et al., 2005). According to Beveridge et al., (2025) education level has more impact on neuroglia hygiene than brain reserve.In this framework, greater anatomical capacity confers a higher threshold for damage tolerance, suggesting that more extensive neurodegeneration is required before clinical symptoms of cognitive decline emerge. Accordingly, brain reserve is considered a "passive" model, reflecting a fixed structural buffer against pathology. However, it is now recognized that lifelong neuroplasticity continuously reshapes neuronal connectivity and brain morphology, blurring the distinction between passive structural reserve and adaptive functional capacity.

Cognitive reserve: a principle of activity

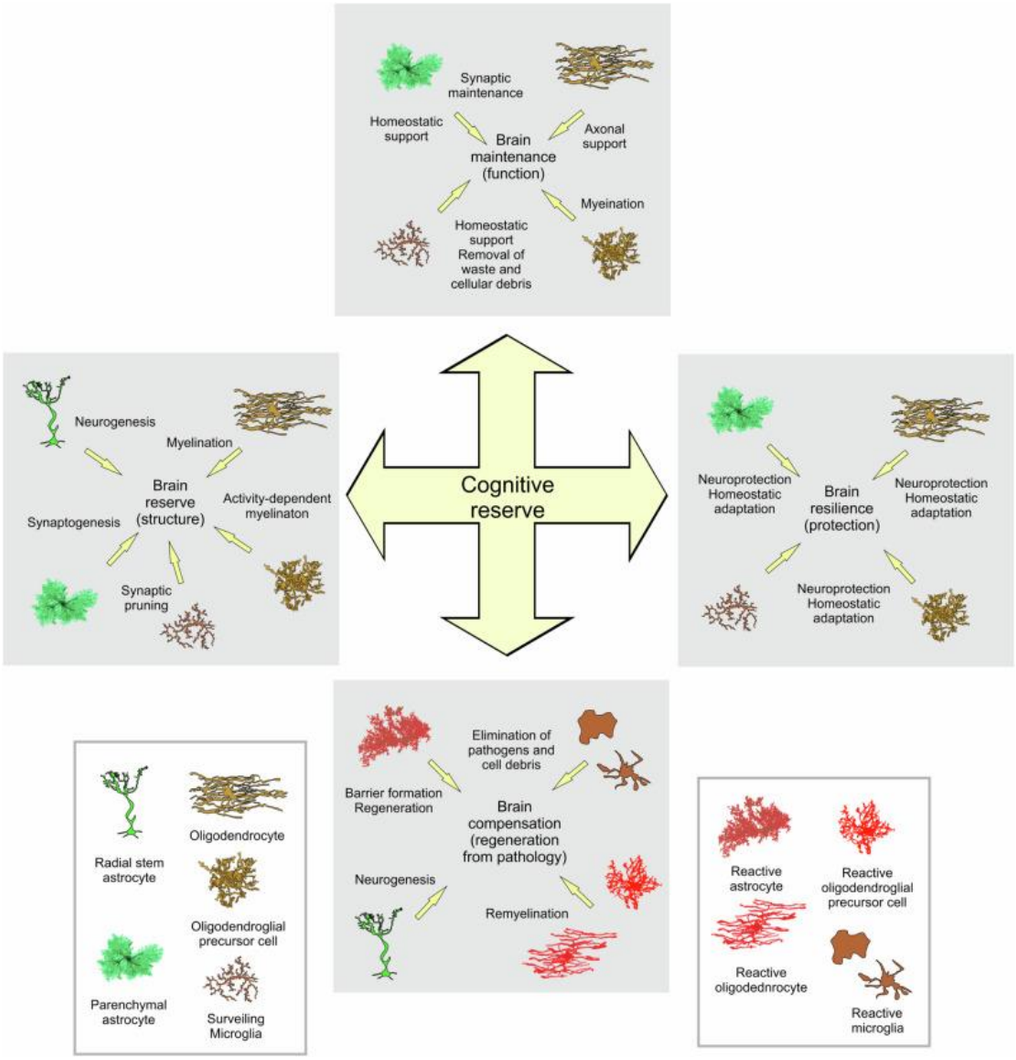
Cognitive reserve is a dynamic construct that reflects the brain's capacity to adapt to life's ongoing challenges. Learning, memory, and the continuous response to environmental demands induce structural and functional modifications in neural networks over time. As such, cognitive reserve represents an individual trait shaped by cumulative neural plasticity—morphological and functional—arising from lifelong interactions between genetic predispositions, environmental exposures, and accrued pathological burden. The structural state of the brain at the time of injury, together with the efficiency of compensatory and homeostatic mechanisms, influences both the extent of cognitive decline and the potential for recovery. Ultimately, this interplay determines an individual's vulnerability or resilience to neurological disease (Lomeli et al., 2017). Brain resilience is defined as the brain's capacity to withstand injury without developing pathological symptoms, is similarly characterized by these factors. Additional components that define the comprehensive reserve and resilience capacity, such as brain maintenance and brain compensation, are encompassed within cognitive reserve. Specifically, brain maintenance employs diverse strategies to maintain brain physiology, thereby influencing cognitive lifespan and physiological brain aging. Finally, brain compensation describes a range of defense mechanisms that restore brain health and cognitive function by repairing and regenerating damaged neural circuits or neural tissue.

Cellular mechanisms of the cognitive reserve and the role of neuroglia

For over a century, neurons have been the primary focus of research on nervous system function in health and disease (Mehler et al., 2008). Consequently, the neuronocentric approach in psychology, psychiatry, neurophysiology, and neurology posits that cognitive reserve resides in neurons. However, the nervous tissue also comprises non-neuronal components, such as vascular cells and neuroglia. The nervous system evolved by dividing its functions between neurons—which rapidly process and transport information—and neuroglia—which maintain homeostasis and safeguard the nervous tissue. All cellular constituents of the nervous tissue, including neurons, neuroglia, and vascular cells, collaborate to form the brain's active milieu, characterized by intricate feedback signaling. Despite distinct gene sets and time-domain-specific actions, these cell types collectively contribute to cognitive reserve. The connectome, synaptic connections, and learning are shaped by neural plasticity, which also influences cognitive reserve. Similarly, cognitive reserve depends on preserving the massive brain vascularization—the human brain's

blood vessels span approximately 600 kilometers (Stone et al., 2023). Age-dependent remodeling of the brain's vasculature, which regulates blood flow and metabolic health, is facilitated by pericytes and endothelial cells (5–10 billion in total). Pericytes, in particular, are gaining attention for their roles in brain repair and compensation. In this context, we highlight **neuroglia**—the homeostatic and defensive cells of the nervous system—as critical mediators of lifelong neural adaptation. Within the central nervous system (CNS), astrocytes, microglia, and oligodendrocytes execute complex signaling cascades that maintain neural tissue homeostasis under physiological conditions and orchestrate protective responses during pathology. By shaping the neural microenvironment, modulating immune responses, and supporting synaptic and structural plasticity, neuroglia play a pivotal role in both the progression and resolution of neurological disorders. Importantly, these glial cells operate through distinct, cell-type-specific mechanisms that contribute substantively to **cognitive reserve**, reinforcing the brain's resilience to age- and disease-related decline. (Fig. 1).

Fig. 1. The contribution of neuroglia to cognitive reserve.



All facets of cognitive reserve fundamentally depend on neuroglial function. The brain's cytoarchitecture and structural integrity are shaped by dynamic interactions between neurons and neuroglia. During development, prenatal radial glia and postnatal radial astrocyte-derived neural stem cells play pivotal roles in promoting neurogenesis and guiding neuronal migration, thereby establishing the foundational architecture necessary for cognitive resilience (Mori et al., 2005). Astrocytes facilitate synaptogenesis, regulate synaptic development, and provide building blocks for the adaptive remodeling of neuronal membranes through the release of various chemicals, such as cholesterol, glypicans, hevins, and trombospondins. Microglia generates synaptically connected neuronal ensembles via synaptic pruning and trogocytosis, while oligodendroglial cells maintain the connectome and enable activity-dependent lifelong myelination. Neuroglial cells sustain CNS homeostasis and define brain resilience through numerous metabolic cascades and protective

pathways. Neuroglia is indispensable for brain defense and regeneration; reactive glial cells construct perilesional barriers, eliminate infections and cellular waste, and orchestrate repair.

The reserve of the brain and neuroglia

As previously stated, a diversity of neurons and interneuronal connections, defined by the connectome (synapses and axonal projections), constitute the brain reserve. Neuroglia is responsible for maintaining all these components (Verkhratsky et al., 2019). However, neural stem cells, also known as radial stem astrocytes, support adult neurogenesis, while radial glia is involved in embryonic neurogenesis. These cells share the primary traits of astroglia. Microglia employ synaptic pruning to remove quiet, dysfunctional, or superfluous connections, thereby reshaping neuronal ensembles, and astrocytes release a range of chemicals that regulate synaptogenesis, synaptic maturation, and synaptic extinction, both influencing synaptic connectivity. Oligodendroglia is responsible for activity-dependent myelination and support the entire connectome of the brain. White matter, which constitutes approximately 50% of the adult human brain compared to 10% in rodents, is a major component affecting the processing and cognitive abilities of the human brain.

Neuroglia and brain maintenance

All organisms are maintained in a state of near-equilibrium stability through brain maintenance, which Claude Bernard referred to as interior milieu stability and Walter Cannon as homeostasis (Cooper et al., 2008). Allostasis, also known as adaptive homeostasis (αλλο − changeable and στασισ - standing still), means "maintaining homeostasis through change" or "remaining stable by being variable." By continuously adapting to external obstacles, the central nervous system (CNS), maintains homeostasis, as do all other tissues, organs, and even the body as a whole. The maintenance of the brain is mostly the responsibility of astrocytes, the main homeostatic cells in the central nervous system (CNS). Astrocytes are essential for the dynamic control of ionostasis, or the ionic composition of the CNS interstitium, using a range of specialist pumps and transporters. They regulatetrace metals and the four main biological ions (Na^+, Ca^{2+}, K^+, and Cl^-). By aiding in neurotransmitter catabolism (astrocytes break down glutamate into glutamine and break down catecholamines and adenosine) and neurotransmitter clearance (astrocytes express transporters that remove glutamate, GABA, catecholamines, and adenosine), astrocytes are indispensable for neurotransmission and neuronal delivery of essential neurotransmitter precursors like glutamine or L-serine. Astrocytes are the primary antioxidant mechanism in the central nervous system and mediate neuroprotection in various ways (Chen et al., 2020). When the glycogen in astrocytes is broken down, L-lactate is created, which can be used as fuel by neurons. Throughout life, oligodendroglial precursor cells (OPCs) support de novo activity-dependent myelination and remyelination and stimulate overall central nervous system homeostasis, much like oligodendrocytes support axons. Finally, microglia carries out ongoing repairs, eliminate debris, and back the immune homeostasis of the nerve tissue.

Neuroglia and the resilience of the brain

The brain's resistance to insults depends on the neural tissue's capacity to sustain a certain level of harm withoutdeveloping pathological symptoms. While neuroglia is increasingly recognized as central to this resilience, the underlying mechanisms remain incompletely understood. Each insult perturbs the homeostatic functions that glial cells, particularly astrocytes, mobilize in response to environmental stressors (Woodburn et al., 2021). Failure or attenuation of these responses can precipitate neuropathology. For instance, prolonged exposure to stress can induce astrocytic atrophy, a phenomenon linked to impaired neurotransmission and the emergence of depressive-like symptoms. Notably, a subset of animals exposed to identical stressors showed no astrocytic alterations—an outcome correlated with greater emotional resilience and an enhanced capacity to cope with adversity.

The brain'scompensatory neuroglia

Neuroglia constitute the central nervous system’s principal line of defense, mounting highly regulated responses to pathological insults through **reactive gliosis**—an evolutionarily conserved hallmark of glial activation. This complex process is shaped by disease context, environmental stimuli, and the degree of tissue disruption. A key feature of reactive gliosis is **proliferative anisomorphic gliosis**, characterized by the expansion and accumulation of astrocytes, microglia, and oligodendrocyte precursor cells (OPCs) in the vicinity of lesions. These cells collectively form a **protective glial barrier** that isolates damaged regions, mitigates further injury, and contributes to the restoration of tissue homeostasis (Traiffort et al., 2020). This condition is brought on by brain trauma that can be mechanical, viral, ischaemic, or autoimmune. Anastrocytes lose their

complex arborisations and become barrier-reactive astrocytes, while certain microglia differentiates into phagocytic macrophage-like cells that remove material from inside the lesion core. Significant morphological and metabolic changes also occur in these glial cells. Glial barriers are crucial for postlesional regeneration, aid in wound closure, and effectively protect the surrounding nerve tissue. Brain regeneration also benefits from neurogenesis, which is provided by radial stem astrocytes. Reactive astrocytes and reactive microglia surround senile plaques to protect neurons from damage, and astrocytes undergo isomorphic, non-proliferative gliosis in AD. This protective role diminishes with age and the severity of AD; glial paralysis promotes neuronal death in the later stages of the illness, leading to clinical dementia. Finally, in many illnesses, OPCs serve as a source of regenerative remyelination. For brain compensation, neuroglial cells are frequently necessary (Franklin et al., 2015)

Age, neuroglia, and cognitive decline associationwith ageing

Age is the primary risk factor for neurodegenerative disorders and is associated with cognitive impairment. Physiological ageing is characterized by a relative maintenance of neuronal numbers and a notable loss of white matter linked to oligodendrocyte decline and the halt of OPC proliferation. As the brain ages, both astrocytes and microglia show substantial shrinkage, which reduces the brain's ability to defend itself and maintain homeostasis. Tau astrogliopathy, the root cause of several neurodegenerative illnesses linked to cognitive decline, is also associated with ageing (McCann et al., 2021). It was demonstratedthat the brains of "super-centenarians," or individuals over110, exhibit age-dependent tau astrogliopathy. Thus, neuroglial degeneration and paralysis are at least partially associated with age-dependent cognitive decline; asTian et al. (2016) observed this during their research on electro acupuncture inhibited neuronal apoptosis.

Lifestyle

Lifestyle factors—such as diet, physical activity, social engagement, and intellectual stimulation—have been shown to significantly influence cognitive reserve and lifespan. Studies in aged animals and diverse experimental models indicate that the cognitive benefits of physical exercise and enriched environments are mediated, at least in part, by enhanced homeostatic support and improved neuroglial architecture. Ageing and chronic neurodegenerative conditions are frequently associated with glial asthenia and atrophy of astrocytes and microglia—deficits that can be partially reversed through dietary and environmental interventions. Notably, experimental replacement of senescent astrocytes and microglia with their younger counterparts in aged rats has been shown to restore cognitive function, highlighting the therapeutic potential of targeting glial health to mitigate age-related cognitive decline. (Verkhratsky et al., 2019). In AD rats and miceneurogenesis is likewise stimulated by exercise and exposure to enriched environments; the polyunsaturated fatty acid 2-hydroxy-docosahexaenoic acid supplementation has a comparable effect (Wu et al. 2008, Wu et al. 2005). Calories restriction increases synaptic coverage, which improves K^+ buffering, glutamate clearance, and synaptic plasticity. Additionally, it results in the development of astroglial perisynaptic leaflets. Mental exercises such as playing card game bridge develop both cognitive and social skills that together curtail the risk of neurodegenerative diseases. For example, bridge players live longer than chess players, both with relatively low risk and frequency of occurring Alzheimer disease or other similar brain malfunctions (Yu et al. 2025; Makri et al. 2021).

Noradrenergic innervation

The noradrenergic pan-brain innervation, which is primarily supplied by neurons of the Locus ceruleans (LC), a small nucleuslocated at the fourth ventricle, is the primary source (~70%) of noradrenaline (NA) in the central nervous system. This innervation at least partially mediates the neurochemical effects of lifestyle choices and environmental stimulation on the brain. There have also been suggestions that AD may be caused by degeneration of the noradrenergic system. Degradation of LC neurons due to aging and chronic age-dependent illnesses reduces noradrenergic bioavailability (Zorec et al., 2025).

The primary cellular targets for NA are astrocytes due to their high levels of α- and β-adrenoceptor expression. Moreover, only astrocytes contain monoamine oxidase B (MAO-B), the essential enzyme for catecholaminergic catabolism. Ageing and neurodegeneration are associated with increased expression of monoamine oxidase B (MAO-B), which may further compromise noradrenergic innervation. In Alzheimer’s disease (AD) mouse models, reduced noradrenaline levels have been shown to induce aberrant astrocytic Ca^{2+} signalling. Targeting MAO-B with either reversible or irreversible inhibitors may offer a promising strategy for enhancing cognitive reserve.

In addition, transcranial direct current stimulation (tDCS) has been demonstrated to modulate astrocytic function via adrenergic receptors, leading to improvements in memory, facilitation of motor rehabilitation, alleviation of depressive symptoms, and attenuation of cognitive decline in patients with AD.(Choi et al., 2013). Following exposure to tDCS, either pharmacological inhibition of α1-adrenoceptors or ablation of noradrenergic neurons inhibits the large astrocytic Ca^{2+} signals.

Conclusion

In conclusion, cognitive reserve reflects a dynamic, lifelong interplay between genetic factors, environmental exposures, and complex cellular processes, extending well beyond traditional neuron-centric models. Neuroglial cells—particularly astrocytes, oligodendrocytes, and microglia—emerge as critical contributors to this reserve. These cells maintain neural homeostasis through ion regulation, neurotransmitter clearance, and metabolic support; they facilitate compensatory mechanisms by promoting regeneration and forming protective barriers following injury; and they shape structural resilience through processes such as neurogenesis, synaptic pruning, and myelination. Age-related declines in glial function—characterized by asthenia and atrophy—substantially increase the risk of cognitive impairment and neurodegeneration. However, growing evidence suggests that lifestyle interventions, pharmacological treatments, and neuromodulatory techniques such as transcranial stimulation can enhance neuroglial health, offering promising strategies to bolster cognitive reserve. Recognizing neuroglia as active, indispensable agents in maintaining brain resilience and cognitive longevity—rather than merely passive support cells—fundamentally reframes our understanding of aging, neurodegeneration, and mental health, and opens new therapeutic avenues for preserving cognitive function across the lifespan..

Reference

Allen JS, Bruss J, Damasio H. The aging brain: the cognitive reserve hypothesis and hominid evolution. American journal of human biology. 2005 Nov;17(6):673-89.

Beveridge, J., Sheth, P., Thakkar, S., Silverglate, B., & Grossberg, G. (2025). The impact of cognitive reserve relative to risk of Alzheimer's disease and rate of progression: an up-to-date review of the literature. Expert Review of Neurotherapeutics, 25(2), 175-187.

Choi J, Twamley EW. Cognitive rehabilitation therapies for Alzheimer's disease: a review of methods to improve treatment engagement and self-efficacy. Neuropsychology review. 2013 Mar;23:48-62.

Chen Y, Qin C, Huang J, Tang X, Liu C, Huang K, Xu J, Guo G, Tong A, Zhou L. The role of astrocytes in oxidative stress of central nervous system: A mixed blessing. Cell proliferation. 2020 Mar;53(3):e12781.

Cooper SJ. From Claude Bernard to Walter Cannon. Emergence of the concept of homeostasis. Appetite. 2008 Nov 1;51(3):419-27.

De Kloet ER, Joëls M, Holsboer F. Stress and the brain: from adaptation to disease. Nature reviews neuroscience. 2005 Jun 1;6(6):463-75.

Franklin RJ, Goldman SA. Glia disease and repair—remyelination. Cold Spring Harbor perspectives in biology. 2015 Jul 1;7(7):a020594.

Finch CE, Kulminski AM. The Alzheimer's disease exposome. Alzheimer's & Dementia. 2019 Sep 1;15(9):1123-32.

Goodglass H, Kaplan E. Assessment of cognitive deficit in the brain-injured patient. Inneuropsychology 1979 (pp. 3-22). Boston, MA: Springer US.

Lomeli N, Bota DA, Davies KJ. Diminished stress resistance and defective adaptive homeostasis in age-related diseases. Clinical Science. 2017 Oct 25;131(21):2573-99.

Love S. Post mortem sampling of the brain and other tissues in neurodegenerative disease. Histopathology. 2004 Apr;44(4):309-17.

Makri, M., Iakovou, K. V., Dasygenis, M., Baras, N., Brouzou, A., Brouzos, I., ... & Tsolaki, M. (2021). The "Bridge" project: α European innovative intergenerational approach using serious games for people with dementia. Psychology, 12(9), 1434-1456.

McCann H, Durand B, Shepherd CE. Aging-related tau astrogliopathy in aging and neurodegeneration. Brain Sciences. 2021 Jul 13;11(7):927.

Mori T, Buffo A, Götz M. The novel roles of glial cells revisited: the contribution of radial glia and astrocytes to neurogenesis. Current topics in developmental biology. 2005 Jan 1;69:67-99.

Mehler MF. Epigenetic principles and mechanisms underlying nervous system functions in health and disease. Progress in neurobiology. 2008 Dec 11;86(4):305-41.

Stone J, Mitrofanis J, Johnstone DM, Robinson SR. Twelve protections evolved for the brain, and their roles in extending its functional life. Frontiers in Neuroanatomy. 2023 Nov 6;17:1280275.

Taddei, R. N., & Duff, K. E. (2025). Synapse vulnerability and resilience underlying Alzheimer's disease. EBioMedicine, 112.

Tian, G. H., Tao, S. S., Chen, M. T., Li, Y. S., Li, Y. P., Shang, H. C., ... & Tang, H. B. (2016). Electroacupuncture treatment alleviates central poststroke pain by inhibiting brain neuronal apoptosis and aberrant astrocyte activation. Neural Plasticity, 2016(1), 1437148.

Traiffort E, Kassoussi A, Zahaf A, Laouarem Y. Astrocytes and microglia as major players of myelin production in normal and pathological conditions. Frontiers in Cellular Neuroscience. 2020 Apr 7;14:79.

Verkhratsky A, Parpura V, Rodriguez-Arellano JJ, Zorec R. Astroglia in Alzheimer's disease. Neuroglia in neurodegenerative diseases. 2019:273-324.

Verkhratsky A, Ho MS, Zorec R, Parpura V. The concept of neuroglia. Neuroglia in Neurodegenerative Diseases. 2019:1-3.

Woodburn SC, Bollinger JL, Wohleb ES. The semantics of microglia activation: neuroinflammation, homeostasis, and stress. Journal of neuroinflammation. 2021 Dec;18:1-6.

WU, Z., LI, M., LI, Y., ZHANG, Y., JIA, X., & CHEN, M. (2005). Quantitative study of tiantai I on superoxidative dismutase and lipofuscin in relevant cerebral areas of spontaneous Alzheimer disease in mice. Chinese Journal of Tissue Engineering Research, 178-181.

Wu, Zz., Huang, A.C.J., de Vellis, J. et al. Effect of Tiantai No.1 (天泰1号) on β-amyloid-induced neurotoxicity and NF-κ B and cAMP responsive element-binding protein. Chin. J. Integr. Med. **14**, 286–292 (2008). https://doi.org/10.1007/s11655-008-0286-y

You, D., Ramli, S. H., Ibrahim, R., Romli, M. H., Chu, Q., Yu, X., & Li, Z. (2025). Rehabilitation playthings design to promote positive emotions among people living with Alzheimer's Disease: a conceptual synthesis. Disability and Rehabilitation: Assistive Technology, 1–12. https://doi.org/10.1080/17483107.2025.2484624

Zorec R. Noradrenergic system in the pathogenesis of age-dependent neurodegeneration. Ageing and longevity. 2025 Jan 28;6(1):33-9.