

Neuroglia—An Open Access Journal

James St John

Griffith Institute for Drug Discovery, Griffith University, 170 Kessels Road Nathan, Brisbane, QLD 4111, Australia;
j.stjohn@griffith.edu.au

Welcome to *Neuroglia*, a new Open Access MDPI journal which will report original research articles and reviews on studies of neuroglia. With a view of creating an impact for the community, we provide a platform for reporting and discussing glial biology, neuropathology, the role of the glia in neurological diseases, and the numerous strategies that can be developed for producing effective therapies.

Neuroglia were first described in 1856 by Rudolf Virchow, who wrote of the presence of a connective substance in which the nervous system elements are embedded, and which created a “Nervenkitt” or nervous system glue (neuroglia). Since then, the introduction of new identification methods and expression studies have enabled us to identify numerous different glial cells and it is clear that their roles are much more than merely a glue that holds the nervous system together. Today, we are aware of the important roles that glial cells have in guiding neurons to their targets, maintaining homeostasis, myelinating axons, and repairing and regenerating nerves by removing dead cells and destroying pathogens. We are also aware of the numerous different states of the different types of glial cells as they react to the changing neural environment and how glial cells contribute to disease and repair of the nervous system.

Within the developing peripheral and central nervous systems, glial cells differentiate and migrate to establish the neural networks while communicating with and supporting the neurons. The glial limitans creates protective barriers to the central nervous system, while most oligodendrocytes and Schwann cells produce the myelin sheaths that insulate axons. Complex signalling occurs across populations of glial cells, including waves of calcium signalling which can coordinate glial cell responses. Vascular–glia interactions form the neurovascular blood brain barrier and bidirectional signalling can regulate blood flow and astrocyte maturation [1]. Glial cells are also critical components of neuroplasticity and aid the continual remodelling of neural connections [2]. This is partly because glial cells are highly phagocytic and remove dead cells and cellular components that arise [3].

After injury such as ischemia, glial cells provide neuroprotection to limit neural damage with the glial cells acting as active immune cells that quickly react to clean up an injury site [4]. However, despite the wealth of functions, glial cells are also involved in disease and neurodegeneration. Bacteria and viruses can invade the nervous system via peripheral nerves, including the nasal cavity nerves (olfactory and trigeminal nerves) [5] with implications for neurodegeneration, and the gut biome interactions can regulate the gut–brain axis responses. While the glia can engulf and destroy pathogens to prevent many infections, the pathogens can also exploit the glial physiology to survive and spread into the nervous system. Pathogen infections can lead to demyelinating diseases which can have devastating effects on people. After injury and infection, reactive gliosis can exacerbate conditions and lead to chronic inflammation. Glioblastoma, which has multiple potential initiation factors, is just one example of uncontrolled cell growth that we need to understand better so that we can create effective therapies.

With our deep fundamental knowledge of glial biology, we can create therapies to treat and repair injuries and diseases of the nervous system. Glial cells are highly responsive to injuries and can drive neuroregeneration and repair [6]. For example, pharmacology can help us design drug therapies to tackle chronic pain [7]. Cell transplantation is a growing



Citation: St John, J. *Neuroglia*—An Open Access Journal. *Neuroglia* **2021**, *2*, 2–3. <https://doi.org/10.3390/neuroglia2010002>

Received: 31 May 2021

Accepted: 31 May 2021

Published: 4 June 2021

Publisher's Note: MDPI stays neutral with regard to jurisdictional claims in published maps and institutional affiliations.



Copyright: © 2021 by the author. Licensee MDPI, Basel, Switzerland. This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution (CC BY) license (<https://creativecommons.org/licenses/by/4.0/>).

area of interest and transplantation of glial cells is being trialled for treatment of various nerve injuries, including spinal cord injuries [8].

I have only mentioned a few aspects of glial function in this editorial. We still have a long way to go in understanding the biology and function of glial cells, and how we can create therapies for the vast array of neurological conditions that involve glial cells. Together we can share our discoveries and design therapies that will have positive impacts for society. To achieve this, we have brought together a diverse and experienced editorial board who will work with the publisher and dedicated administrative team to bring you the most important articles. It will be an exciting journey to see the new discoveries and advances that our scientific community will bring to this journal and we welcome your submissions.

Conflicts of Interest: The author declares no conflict of interest.

References

1. Presa, J.L.; Saravia, F.; Bagi, Z.; Filosa, J.A. Vasculo-Neuronal Coupling and Neurovascular Coupling at the Neurovascular Unit: Impact of Hypertension. *Front Physiol* **2020**, *11*, 584135. [[CrossRef](#)] [[PubMed](#)]
2. Stagni, F.; Giacomini, A.; Guidi, S.; Emili, M.; Uguagliati, B.; Salvalai, M.E.; Bortolotto, V.; Grilli, M.; Rimondini, R.; Bartesaghi, R. A flavonoid agonist of the TrkB receptor for BDNF improves hippocampal neurogenesis and hippocampus-dependent memory in the Ts65Dn mouse model of DS. *Exp. Neurol.* **2017**, *298*, 79–96. [[CrossRef](#)]
3. Crespo-Castrillo, A.; Garcia-Segura, L.M.; Arevalo, M.A. The synthetic steroid tibolone exerts sex-specific regulation of astrocyte phagocytosis under basal conditions and after an inflammatory challenge. *J. Neuroinflammation* **2020**, *17*, 37. [[CrossRef](#)] [[PubMed](#)]
4. Moretti, R.; Leger, P.L.; Besson, V.C.; Csaba, Z.; Pansiot, J.; Di Criscio, L.; Gentili, A.; Titomanlio, L.; Bonnin, P.; Baud, O.; et al. Sildenafil, a cyclic GMP phosphodiesterase inhibitor, induces microglial modulation after focal ischemia in the neonatal mouse brain. *J. Neuroinflammation* **2016**, *13*, 95. [[CrossRef](#)] [[PubMed](#)]
5. Nazareth, L.; Walkden, H.; Chacko, A.; Delbaz, A.; Shelper, T.; Armitage, C.W.; Reshamwala, R.; Trim, L.K.; St John, J.A.; Beagley, K.W.; et al. Chlamydia muridarum Can Invade the Central Nervous System via the Olfactory and Trigeminal Nerves and Infect Peripheral Nerve Glial Cells. *Front. Cell. Infect. Microbiol.* **2020**, *10*, 607779. [[CrossRef](#)] [[PubMed](#)]
6. Lindsey, B.W.; Aitken, G.E.; Tang, J.K.; Khabooshan, M.; Douek, A.M.; Vandestadt, C.; Kaslin, J. Midbrain tectal stem cells display diverse regenerative capacities in zebrafish. *Sci. Rep.* **2019**, *9*, 4420. [[CrossRef](#)] [[PubMed](#)]
7. Ceruti, S. From astrocytes to satellite glial cells and back: A 25 year-long journey through the purinergic modulation of glial functions in pain and more. *Biochem. Pharmacol.* **2021**, *187*, 114397. [[CrossRef](#)] [[PubMed](#)]
8. Gilmour, A.D.; Reshamwala, R.; Wright, A.A.; Ekberg, J.A.K.; St John, J.A. Optimizing Olfactory Ensheathing Cell Transplantation for Spinal Cord Injury Repair. *J. Neurotrauma* **2020**, *37*, 817–829. [[CrossRef](#)] [[PubMed](#)]