Mini Review
Volume 12:1, 2023

Journal of Trauma & Treatment

ISSN: 2167-1222 Open Access

Adult Brain Repair of Neural Damage

Azhar Noor*

Department of Neurorehabiliation, University of Greifswald Greifswald, Germany

Abstract

The brain is always at risk of injury, whether it is acute or chronic. The capacity for regeneration in the adult mammalian brain has long been thought to be severely limited when compared to other tissues such as the skin, liver, or intestines. As a result, the mammalian brain is unable to rebuild structures that have been lost due to harmful events such as ischemic stroke or traumatic brain injury. With acute or chronic injury, however, there is significant functional restoration due to the ability of surviving brain structures to take up at least some of the functions of destroyed tissues. This is seen, for example, in patients who have left-hemispheric strokes and may initially exhibit motor or sensory aphasia.

Keywords: Brain damage • Chronic brain injury • Cell damage

Introduction

Acute or chronic brain damage in adults frequently results in significant loss of neuronal tissue and persistent functional disability. A number of treatments have been developed over the last two decades to harness the regenerative potential of neural stem cells and the current destiny flexibility of neural cells in the nervous system to avoid tissue loss or improve structural and functional regeneration following damage. We examine current obstacles and limitations in stem cell-associated neural healing in the adult brain, as well as potential approaches for advancing experimental stem cell therapies into the clinic [1,2].

Following extensive training and rehabilitation, a significant number of patients regain their ability to talk and communicate:

- Similarly, the brain can compensate effectively before the consequences of significant loss of neuronal tissue become apparent
- Before Parkinson's symptoms appear, more than 80% of dopaminergic neurons in the substantia nigra are thought to be damaged
- As a result, the adult mammalian brain's ability to repair itself—at least functionally—is obvious.
- These endogenous repair processes have obvious limitations, leaving a significant percentage of adult brain damage patients with lasting functional abnormalities. As a result, fresh ways for treating degenerative or traumatic brain illnesses must be devised. Exogenous cells have been used in the illness context to either recruit or augment endogenous healing processes or to improve brain function by supplying exogenous cells via transplantation.
- We concentrate solely on existing methodologies and concepts for enhancing brain healing using endogenous neural stem cells (NSCs) or other neural cells.

NSCs make new neurons in specific locations of the adult brain15, which can be used to target endogenous neurogenesis for brain repair [3-5].

***Address for Correspondence:** Azhar Noor, Department of Neurorehabiliation, University of Greifswald Greifswald, Germany, E-mail: noor.az@gmail.com*

Received: 11 December, 2022, Manuscript No. JTM-22-86940; **Editor assigned:** 13 December, 2022, PreQC No. P-86940; **Reviewed:** 26 December, 2022, QC No. Q-86940; **Revised:** 31 December, 2022, Manuscript No. R-86940; **Published:** 07 January, 2023, DOI: 10.37421/2167-1222.2022.11.547

Neurogenesis

One of these is the SVZ, which lines the lateral ventricles and is where NSCs give birth to newborn cells that migrate along the rostral migratory stream into the olfactory bulb (OB), where they differentiate into several types of olfactory neurons. In the rodent brain, SVZ/OB neurogenesis is relatively active, whereas in the human brain, the neurogenic activity of the SVZ appears to be very low or non-existent. In contrast, the hippocampus DG, where NSCs give rise to DG granule cells throughout life, is the second major neurogenic region.

Glial repair with neural stem cells

The destiny potential of endogenous NSCs also allows for targeting NSCs to assist glial cell replacement and eventual brain repair, in addition to techniques aimed at enhancing endogenous neurogenesis for neuronal repair in the context of acute or chronic illness. Demyelination in the mouse SVZ [6], for example, has been shown to result in increased NSC-derived oligodendrocyte production, which may aid in the remyelination of the wounded brain following a lesion76,77. Induced production of oligodendrocytes (which are not produced by DG NSCs under normal conditions) could be used to induce remyelination of the DG circuitry in a variety of demyelinating disorders, including multiple sclerosis and epilepsy78–81. However, possible therapeutic techniques aimed at using endogenous NSCs for glial repair are still being developed, and more research is needed. [7-9]

Acknowledgement

Not applicable.

Conflict of Interest

There is no conflict of interest by author.

References

1. Omalu, Bennet I., Steven T. DeKosky, Ryan L. Minsterand Cyril H. Wecht et al. “Chronic traumatic encephalopathy in a National Football League player.” *Neurosurgery* 57 (2005): 128-134
2. McKee AC. “The first NINDS/NIBIB consensus meeting to define neuropathological criteria for the diagnosis of chronic traumatic encephalopathy.” *Acta Neuropathol* 131 (2016):75–86.
3. Goldstein, Lee E., Andrew M. Fisher, Chad A. Tagge and Xiao-Lei Zhang et al. “Chronic traumatic encephalopathy in blast-exposed military veterans and a blast neurotrauma mouse model.” *Sci Transl Med* 4 (2012): 134ra60-134ra60.
4. Millspaugh, J. A. “Dementia pugilistica.” *US Nava Med Bull* 35(1937): e303.

5. Critchley, M. "Hommage a clovis vincent." *Paris: Maloine* (1949).

6. McKee AC. "The spectrum of disease in chronic traumatic encephalopathy." *Brain* 136 (2013):43–64.

7. Jordan BD. "The clinical spectrum of sport-related traumatic brain injury." *Nat Rev Neurol* 9 (2013):222–230.

8. McKee, Ann C., Thor D. Stein, Patrick T. Kiernan, and Victor E. Alvarez. "The neuropathology of chronic traumatic encephalopathy."*Brain Pathol* 25(2015): 350-364.

9. McKee, Ann C., Robert C. Cantu, Christopher J. Nowinski and E. Tessa Hedley-Whyte et al. "Chronic traumatic encephalopathy in athletes: progressive tauopathy after repetitive head injury." *J Neuropathol Exp Neurol* 68(2009): 709-735.

How to cite this article: Noor, Azhar. "Adult Brain Repair of Neural Damage." J Trauma Treat 12 (2023): 547.