THE ARSENAL

Augusta University's Undergraduate Research Journal

ISSN 2380-5064 | The Arsenal is published by the Augusta University Libraries | http://guides.augusta.edu/arsenal

Volume 5, Issue 1 (2023)
Special Edition Issue

EXAMINING THE FUNCTION OF ELAVL GENES DURING NEURONAL REPROGRAMMING

Christine Williams, Natalie Mseis, Na Jiang, Mei Jiang, Xuanyu Chen, and Hedong Li

Citation
Williams, C., Mseis, N., Jiang, N., Jiang, M., Chen, X., & Li, H. (2023). Examining the function of elavl genes during neuronal reprogramming. *The Arsenal: The Undergraduate Research Journal of Augusta University*, *5*(1), 82-83.
http://doi.org/10.21633/issn.2380.5064/s.2021.05.01.82

Examining the Function of ELAVL Genes During Neuronal Reprogramming

Presenter(s): Christine Williams

Author(s): Christine Williams, Natalie Mseis, Na Jiang, Mei Jiang, Xuanyu Chen, and Hedong Li

Faculty Sponsor(s): Hedong Li, PhD

Affiliation(s): Department of Chemistry and Physics, Department of Neuroscience and Regenerative Medicine

ABSTRACT

Spinal cord injuries often impair a person's daily life by limiting mobility. While potential treatments such as stem cell transplantation are being investigated in animal models, these treatments have not translated in clinical trials and entail risks including triggering an immune response and tumorigenesis. Neuronal reprogramming has emerged as an alternative method of aiding spinal cord injuries to restore the mobility lost by patients. When an injury to the spinal cord occurs, reactive astrocytes surround the site of injury where neurons are dead. Neuronal reprogramming involves the reprogramming of those surrounding glial cells into functional neurons. When neuronal reprogramming is done in vivo, this potentially eradicates the chances of triggering an immune reaction which results in a less risky treatment. While neuronal reprogramming has great potential in treating neurological diseases, the underlying molecular mechanisms of this unique biological process are still not fully elucidated. Previous research has shown that when overexpressed, the transcription factor, NeuroD1 can convert reactive astrocytes into functional neurons at a high conversion efficiency. NeuroD1 is a critical neurogenic transcription factor that is expressed during the development of the central nervous system. The overexpression of NeuroD1 leads to upregulation of other genes including the two genes, ELAVL2 and ELAVL4. These two genes are known for their active role in neuronal development especially when it comes to neuronal differentiation. Here, we aim to determine if the two genes are capable of reprogramming on their own. Upon cloning these two genes into overexpression vectors, the vectors were virally infected into glial cells. Using techniques like western blot and immunocytochemistry, the cells

overexpressing these genes were then analyzed. The function of ELAVL2 and ELAVL4 in the reprogramming of glial cells (U251 glioblastoma cell line) was examined. Our studies have led us to conclude that the overexpression of the ELAVL genes does not induce the reprogramming of U251 glioblastoma cells to neurons on their own. During NeuroD1-mediated neuronal reprogramming, six days post-infection is enough time to start seeing DCX expression, indicative of the presence of young neurons. Both cells infected with the vectors containing the ELAVL2 or ELAVL4 plasmid did not have expression of DCX. As a result, it was concluded that there were no young neurons present and neuronal reprogramming did not occur by overexpressing the ELAVL genes themselves.

Received: 02/15/2023 Accepted: 03/29/2023

Correspondence: Christine Williams, Augusta University, 1120 15th St. Augusta, GA 30912, chwilliams4@augusta.edu