Infinity Journal of Medicine and Innovation
Volume 1, Issue 1, March 2025
website: https://journalscientific-journal.com/index.php/JIM

Letter to the Editor,

The Hidden Key to Neurorepair: Can the Centrosome Be Reactivated?

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Dear Editor,

Neurodegenerative diseases and nervous system injuries remain among the most challenging medical conditions due to the limited regenerative capacity of neurons. Unlike most somatic cells, neurons are post-mitotic, largely attributed to the absence or inactivation of the centrosome, a key organelle in cell division. Given recent advances in molecular genetics and cellular reprogramming, We propose that reactivating the centrosome in neurons could serve as a novel approach to neural repair, potentially opening new therapeutic pathways for currently incurable neurological disorders.

Keywords:
Neurorepair, Neurodegenerative, Centrosome, microbiome.

Article history:
Received: 29 February 2025.
Revised: 20 March 2025.
Accepted: 24 March 2025.
Published: 31 March 2025.

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The centrosome plays a crucial role in microtubule organization, mitotic spindle formation, and cell cycle progression. In neurons, the centrosome is known to lose its functional capacity early in development, rendering these cells unable to re-enter the cell cycle [1, 2]. Several studies have indicated that manipulating key regulators of the cell cycle, such as cyclins, CDKs (cyclin-dependent kinases), and tumor suppressor genes (e.g., p53 and Rb), can induce cell cycle re-entry in post-mitotic cells, albeit often leading to apoptosis [3]. However, a controlled and precise reactivation of the centrosome could potentially bypass apoptotic pathways and facilitate neurogenesis.

We hypothesize that targeted genetic and epigenetic modulation of centrosomal components could restore its function in neurons without triggering detrimental effects. Gene editing approaches, such as CRISPR/Cas9-mediated activation of key centrosomal genes (e.g., CEP192, PLK4, SAS-6), might reinstate centrosome function in neurons, promoting regenerative processes [4]. Epigenetic reprogramming by modifying DNA methylation and histone modifications associated with centrosomal gene silencing could reactivate the centrosome while maintaining neuronal identity. Additionally, engineering artificial centrosomal structures and introducing them into neurons could provide the necessary microtubule-organizing capabilities for neuronal repair. A controlled expression of cell cycle regulators in combination with centrosomal reactivation could allow neurons to undergo limited proliferation for regenerative purposes.

To test this hypothesis, several experimental strategies can be designed. Using induced pluripotent stem cell (iPSC)-derived neurons, targeted gene activation can be assessed for its effects on centrosomal function and cell cycle re-entry. Transgenic mice expressing inducible centrosomal proteins in neurons could be developed to observe neural regeneration in vivo [5]. Advanced imaging techniques could be utilized to monitor centrosome reactivation and its impact on neural architecture in real-time. Functional analysis of regenerated neurons could determine whether centrosome reactivation

restores proper neuronal activity and connectivity.

If successful, centrosome reactivation in neurons could revolutionize treatments for neurodegenerative disorders such as Alzheimer's, Parkinson's, and spinal cord injuries. It could provide a foundation for regenerative medicine strategies that do not rely solely on stem cell transplantation. However, challenges such as avoiding uncontrolled cell proliferation, ensuring neuronal functionality, and maintaining genomic stability must be addressed.

The potential implications of this approach extend beyond neuronal repair and into the fundamental understanding of cellular aging and biological timekeeping. The irreversible loss of centrosomal activity in neurons may be a critical factor in the aging process, where cellular senescence is not merely a consequence of genetic damage but rather a programmed limitation on regenerative potential. By unlocking this dormant capacity, we could redefine the very nature of biological constraints on lifespan and cognitive function. Furthermore, the concept of reintroducing controlled mitotic activity into neurons challenges long-standing dogma regarding neuroplasticity, suggesting that structural and functional rejuvenation may be possible without external cellular replacement. This could lead to groundbreaking therapies that not only halt neurodegeneration but also actively reverse its effects, allowing the nervous system to regain lost functions in ways previously deemed unattainable.

This hypothesis invites further research into the intricate relationship between centrosomal biology and neural regeneration. I encourage the scientific community to explore this concept, as it holds the potential to shift paradigms in neuroscience and regenerative medicine.

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To site this article: Al-Khikani FH, Abid-Alzahra KM. The Hidden Key to Neurorepair: Can the Centrosome Be Reactivated?. Infinity J. Med. Innov. 2025; 1(1): 18-19.