Short Communication
Volume 07:05, 2024

Journal of Clinical Neurology and Neurosurgery

ISSN: 2684-6012 Open Access

Age-related Changes are Prevented by Short Peptides for Fibroblast-derived Induced Neurons

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Introduction

Age-related cognitive decline and neurodegenerative disorders are among the most pressing challenges in medicine, with aging being a primary risk factor. Research into reversing or mitigating these changes has gained momentum, focusing on innovative approaches like the use of short peptides to influence the cellular and molecular mechanisms underlying neuronal health. Short peptides have emerged as promising therapeutic agents capable of modulating biological processes, particularly in preventing age-related changes in fibroblast-derived induced Neurons (iNs). These neurons, created through reprogramming somatic cells, provide a groundbreaking model for studying neuronal aging and potential interventions. By leveraging short peptides, researchers are uncovering ways to preserve neuronal function, opening avenues for combating age-related neurological decline [1].

Description

Induced Neurons (iNs) are generated by reprogramming fibroblasts, bypassing the pluripotent stem cell stage. This process allows for the direct conversion of somatic cells into functional neurons, offering a valuable platform for studying age-related changes. However, the aging signature of the donor fibroblasts can persist, influencing the functionality and health of the resulting iNs. As these cells retain markers of aging, including altered gene expression, reduced mitochondrial function and accumulation of oxidative damage, they serve as ideal models to explore therapeutic strategies. This is where short peptides become relevant, as they offer a means to modulate these age-associated characteristics and enhance neuronal resilience.

Short peptides are sequences of amino acids that exert specific biological effects, often mimicking or enhancing the activity of naturally occurring proteins. In the context of age-related changes in neurons, these peptides target cellular pathways critical for maintaining neuronal health. One significant pathway involves the regulation of mitochondrial function. Aging neurons often exhibit mitochondrial dysfunction, characterized by reduced energy production and increased oxidative stress. Short peptides can enhance mitochondrial activity by promoting biogenesis, improving electron transport chain efficiency and reducing Reactive Oxygen Species (ROS) accumulation. This mitigation of mitochondrial decline is crucial for preserving the energy demands of neurons and preventing functional impairments [2,3].

Additionally, short peptides influence the epigenetic landscape of iNs. Aging is associated with epigenetic drift, including altered DNA methylation and histone modifications, which can disrupt gene expression and cellular function. Peptides targeting epigenetic regulators can restore a more youthful transcriptional profile, promoting the expression of genes associated with neuronal survival, plasticity and repair. By reversing age-associated epigenetic changes, short peptides help maintain the functional integrity of iNs derived from aged fibroblasts.

The accumulation of misfolded proteins and impaired proteostasis is another hallmark of neuronal aging. Short peptides can enhance proteasomal and autophagic activity, facilitating the clearance of damaged proteins and preventing their aggregation. This is particularly relevant for neurodegenerative diseases, where protein misfolding plays a central role. By boosting cellular mechanisms to handle protein quality control, peptides offer a therapeutic avenue for both preventing age-related changes and addressing disease-specific pathologies. Inflammation is another critical aspect of neuronal aging, with chronic low-grade inflammation contributing to cellular dysfunction and cognitive decline. Short peptides can modulate inflammatory pathways, reducing the production of pro-inflammatory cytokines and enhancing anti-inflammatory responses. This immunomodulatory effect protects iNs from inflammation-induced damage, promoting their long-term health and function [4,5].

Conclusion

Despite their promise, the application of short peptides in combating age-related changes is not without challenges. Delivering peptides effectively to target cells and ensuring their stability in vivo remain significant hurdles. However, advances in drug delivery systems, such as nanoparticles and hydrogels, are paving the way for overcoming these obstacles. Additionally, understanding the precise mechanisms of peptide action and optimizing their design for specific pathways will enhance their therapeutic efficacy.

Age-related changes in fibroblast-derived induced neurons pose a significant challenge to neuronal health, with implications for cognitive decline and neurodegenerative diseases. Short peptides offer a powerful tool to prevent or reverse these changes by targeting key pathways involved in mitochondrial function, epigenetic regulation, proteostasis, inflammation and synaptic integrity. Through their multifaceted actions, peptides address the complex interplay of factors contributing to neuronal aging, providing a comprehensive strategy for preserving cognitive function. While challenges remain in translating these findings into clinical applications, the progress made thus far underscores the potential of short peptides as a cornerstone in the fight against age-related neurological decline. As research continues to refine and expand their use, short peptides may become integral to therapies aimed at enhancing neuronal health and combating the effects of aging.

Acknowledgement

None.

Conflict of Interest

None.

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Received: 01 October, 2024, Manuscript No. jcnn-24-154610; **Editor Assigned:** 03 October, 2024, Pre QC No. P-154610; **Reviewed:** 17 October, 2024, QC No. Q-154610; **Revised:** 22 October, 2024, Manuscript No. R-154610; **Published:** 29 October, 2024, DOI: 10.37421/2684-6012.2024.7.260

References

1. Zhang, Hua and Ilya Bezprozvanny. ""Dirty Dancing" of calcium and autophagy in Alzheimer's Disease." *Life* 13 (2023): 1187.
2. Longo, Frank M. and Stephen M. Massa. "Neuroprotective strategies in Alzheimer's disease." *NeuroRx* 1 (2004): 117-127.
3. Ilina, A. R., I. G. Popovich, G. A. Ryzhak and V. K. Khavinson. "Prospects for use of short peptides in pharmacotherapeutic correction of Alzheimer's disease." *Adv Gerontol* 37 (2024): 10-20.
4. Kraskovskaya, N. A., E. O. Kukanova, N. S. Lin'kova and E. A. Popugaeva, et al. "Tripeptides restore the number of neuronal spines under conditions of *in vitro* modeled Alzheimer's disease." *Bull Exp Biol Med* 163 (2017): 550-553.
5. Chen, Jian-Hua, C. Nicholes Hales and Susan E. Ozanne. "DNA damage, cellular senescence and organismal ageing: Causal or correlative?." *Nucleic Acids Res* 35 (2007): 7417-7428.

How to cite this article: Boltes, Amoruso. "Age-related Changes are Prevented by Short Peptides for Fibroblast-derived Induced Neurons." *J Clin Neurol Neurosurg* 7 (2024): 260.