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Amyotrophic Lateral Sclerosis and Molecular Motor Proteins

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Introduction

Amyotrophic Lateral Sclerosis (ALS) is a devastating neurodegenerative disorder characterized by the progressive loss of motor neurons, resulting in muscle weakness, paralysis, and ultimately death. While the exact etiology of ALS remains elusive, recent research has implicated molecular motor proteins in the disease's pathogenesis. Molecular motor proteins, including dynein and kinesin, play crucial roles in intracellular transport, a process essential for neuronal survival and function. This review explores the intricate interplay between ALS and molecular motor proteins, shedding light on their potential involvement in disease onset and progression. Amyotrophic lateral sclerosis is a complex and heterogenous disorder with genetic and environmental contributions [1]. A growing body of evidence suggests that disturbances in intracellular transport processes mediated by molecular motor proteins may contribute to motor neuron degeneration. This review aims to delve into the intricate relationship between ALS and molecular motor proteins, highlighting their roles in neuronal homeostasis and exploring their potential implications for therapeutic interventions [2,3].

Description

Molecular motor proteins, particularly dynein and kinesin, are central to the transport of organelles, vesicles, and other cargo within neurons. Dynein, responsible for retrograde transport toward the cell body, and kinesin, responsible for anterograde transport toward the synaptic terminals, ensure proper distribution of cellular components critical for neuronal survival, such as mitochondria, RNA, and proteins [4]. Dysregulation of these transport processes can lead to energy deficits, impaired protein quality control, and axonal degeneration. Numerous studies have linked abnormalities in molecular motor proteins to ALS pathogenesis. Mutations in genes encoding these proteins or their regulators have been identified in ALS patients. For instance, mutations in the dynein-dynactin complex components have been associated with both familial and sporadic ALS cases. Altered levels or subcellular localization of motor proteins in ALS-afflicted neurons further contribute to transport defects, disrupted axonal transport, and eventual motor neuron degeneration [5]. In ALS, impaired axonal transport leads to the accumulation of misfolded proteins and organelles in axonal swellings, known as axonal spheroids. These aggregates contribute to oxidative stress, protein aggregation, and inflammation, which collectively lead to motor neuron dysfunction and demise. The ensuing loss of connectivity between neurons and their targets exacerbates the neurodegenerative cascade [6].

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Conclusion

The intricate relationship between amyotrophic lateral sclerosis and molecular motor proteins highlights the pivotal role of intracellular transport in maintaining motor neuron health. Dysregulation of molecular motor proteins contributes to the axonal transport deficits observed in ALS, ultimately driving motor neuron degeneration. Unraveling the precise mechanisms underlying this relationship will facilitate the development of novel therapeutic strategies to combat this devastating disease. Targeting molecular motor proteins and axonal transport pathways presents a promising avenue for ALS therapy. Strategies to enhance transport efficiency, clear protein aggregates, or modulate the activities of motor proteins are being explored. Small molecules, gene therapies, and RNA-based approaches are under investigation to restore intracellular transport and mitigate neurodegeneration.

Acknowledgement

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Conflict of Interest

None.

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