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Reelin Protein and mRNA Alterations in Neurodegenerative Disorders

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Introduction

Neurodegenerative diseases constitute a group of debilitating conditions characterized by the progressive degeneration of neurons, leading to cognitive, motor and functional impairments. These diseases, including Alzheimer's Disease (AD), Parkinson's Disease (PD), Huntington's Disease (HD) and Amyotrophic Lateral Sclerosis (ALS), share common pathological mechanisms involving protein misfolding, aggregation and cellular dysfunction [1]. Among the molecular players implicated in these processes, the Reelin protein and its associated mRNA have gained attention for their potential roles in modulating neuronal survival, synaptic plasticity and overall brain health. Reelin is an extracellular glycoprotein primarily known for its pivotal role in neuronal migration and cortical layer formation during embryonic brain development. It acts through binding to its receptors, primarily ApoER2 and VLDLR and triggers a downstream signaling cascade that influences cytoskeletal dynamics, dendritic spine formation and synaptic plasticity. Beyond its developmental functions, emerging evidence suggests that Reelin might also contribute to adult neuroplasticity and the maintenance of neuronal integrity [2].

Description

In the context of neurodegenerative diseases, there is a growing body of research indicating disease-specific alterations in Reelin protein levels and mRNA expression. In AD, for instance, post-mortem studies have shown a reduction in Reelin protein expression in brain regions crucial for memory and cognition, such as the hippocampus and entorhinal cortex. This downregulation of Reelin may contribute to synaptic dysfunction and cognitive decline observed in AD patients. Moreover, genetic variants in the Reelin gene (RELN) have been associated with an increased risk of late-onset AD, underscoring the potential relevance of Reelin in disease pathogenesis [3]. Similar changes have been observed in PD, albeit with distinct patterns. The substantia nigra, a brain region primarily affected in PD, exhibits altered Reelin expression. Interestingly, in PD, Reelin expression seems to be upregulated in the early stages of the disease, potentially reflecting a compensatory response to the ongoing neurodegeneration.

However, as the disease progresses, Reelin levels might decline, contributing to the worsening motor and non-motor symptoms. This dynamic modulation of Reelin expression highlights its complex involvement in PD pathophysiology. HD, characterized by the expansion of CAG repeats in the huntingtin gene, presents yet another facet of Reelin's role. Studies have shown that mutant huntingtin protein interferes with Reelin signaling, disrupting its normal function in maintaining neuronal connectivity [4]. This disruption might contribute to the cognitive decline observed in HD, adding another layer of complexity to the interplay between Reelin and neurodegenerative processes. In ALS, a disease primarily affecting motor neurons, altered Reelin expression has also been documented. While research in this area is relatively limited, findings suggest that Reelin might be involved in the regulation of motor neuron survival and axonal integrity. Given the multifaceted nature of ALS pathogenesis, the potential contribution of Reelin to the disease warrants further investigation [5,6].

Conclusion

The Reelin protein and mRNA appear to play intricate roles in the pathogenesis of various neurodegenerative diseases. The alterations in Reelin expression are disease-specific, contributing to the diverse clinical manifestations observed across these conditions. However, many questions remain unanswered. The exact mechanisms underlying Reelin's involvement in neurodegeneration, its interactions with other pathological proteins and its potential as a therapeutic target necessitate in-depth exploration. Understanding the precise dynamics of Reelin alterations in different stages of disease progression could provide valuable insights into disease mechanisms and open avenues for novel therapeutic strategies. Modulating Reelin signaling, either through gene therapy, small molecules, or other interventions, might offer a means to restore synaptic function, enhance neuroplasticity and ultimately slow down the degenerative processes in these devastating disorders. Further interdisciplinary research efforts, combining molecular biology, neuroscience and clinical studies, are essential to unravel the complex interplay between Reelin and neurodegenerative diseases, bringing us closer to effective treatments and improved quality of life for affected individuals.

Acknowledgement

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Conflict of Interest

There are no conflicts of interest by author.

References

1. Frotscher, Michael. "Role for Reelin in stabilizing cortical architecture." *Trends Neurosci* 33 (2010): 407-414.
2. Pujadas, Lluís, Agnès Gruart, Carles Bosch and Lídia Delgado, et al. "Reelin regulates postnatal neurogenesis and enhances spine hypertrophy and long-term potentiation." *J Neurosci* 30 (2010): 4636-4649.
3. Beffert, Uwe, Edwin J. Weeber, Gerardo Morfini and Jane Ko, et al. "Reelin and cyclin-dependent kinase 5-dependent signals cooperate in regulating neuronal migration and synaptic transmission." *J Neurosci* 24 (2004): 1897-1906.
4. De Rouvroit, Catherine Lambert, Béatrice Bernier, Inès Royaux and Vinciane de Bergeyck, et al. "Evolutionarily conserved, alternative splicing of reelin during brain development." *Exp Neurol* 156 (1999): 229-238.
5. Jossin, Yves, Lanrun Gui and André M. Goffinet. "Processing of Reelin by embryonic neurons is important for function in tissue but not in dissociated cultured neurons." *J Neurosci* 27 (2007): 4243-4252.

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6. Jossin, Yves, Nina Ignatova, Thomas Hiesberger and Joachim Herz, et al. "The central fragment of Reelin, generated by proteolytic processing *in vivo*, is critical to its function during cortical plate development." *J Neurosci* 24 (2004): 514-521.

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