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Autophagy and Neurodegeneration: Unraveling the Molecular Links

Yuan Klochkov*

Department of Translational Research and Surgery, University of Pisa, 56126 Pisa, Italy

Introduction

Autophagy is a fundamental cellular process involved in the degradation and recycling of cellular components, crucial for maintaining cellular homeostasis and preventing the accumulation of damaged or dysfunctional proteins and organelles. Dysregulation of autophagy has been implicated in various neurodegenerative diseases, including Alzheimer's disease, Parkinson's disease, and Huntington's disease. Understanding the molecular links between autophagy and neurodegeneration is essential for elucidating disease mechanisms and developing potential therapeutic interventions. This review explores the intricate molecular pathways connecting autophagy and neurodegeneration, shedding light on potential targets for disease modulation and treatment.

Description

Autophagy, a fundamental cellular process involved in maintaining cellular homeostasis, is intricately linked to neurodegeneration, contributing significantly to the pathogenesis of various neurological disorders. In neurodegenerative diseases such as Alzheimer's, Parkinson's, and Huntington's disease, dysregulation of autophagy disrupts the clearance of misfolded proteins and protein aggregates, leading to their accumulation and subsequent neuronal dysfunction. Furthermore, autophagy plays a crucial role in maintaining mitochondrial homeostasis through selective degradation of damaged mitochondria, preventing the generation of reactive oxygen species (ROS) that can damage neurons. Dysfunctional autophagy can also disrupt neuronal survival and death pathways, exacerbating neurodegeneration by promoting neuronal cell death or apoptosis in response to stressors. Understanding the molecular links between autophagy and neurodegeneration provides critical insights into disease mechanisms and potential therapeutic targets for mitigating neurodegenerative processes and preserving neuronal function.

The molecular links between autophagy and neurodegeneration are complex and multifaceted, involving various signaling pathways and cellular processes. Dysregulation of autophagy can contribute to the pathogenesis of neurodegenerative diseases by promoting the accumulation of protein aggregates, impairing mitochondrial function, and disrupting neuronal homeostasis. Conversely, enhancing autophagy through pharmacological or genetic interventions has emerged as a potential therapeutic strategy for mitigating neurodegeneration and preserving neuronal function. However, challenges remain in targeting autophagy specifically in affected brain regions and cell types without disrupting essential cellular functions [1-5].

Conclusion

Autophagy plays a crucial role in the pathogenesis of neurodegenerative diseases, serving as a double-edged sword by both protecting against and contributing to neuronal dysfunction and cell death. Elucidating the molecular links between autophagy and neurodegeneration offers insights into disease mechanisms and potential therapeutic targets. Future research efforts aimed at modulating autophagy in a precise and controlled manner hold promise for developing novel treatments for neurodegenerative diseases, ultimately improving the quality of life for affected individuals.

Acknowledgement

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Conflict of Interest

None.

References

1. Sardiello, Marco, Michela Palmieri, Alberto Di Ronza and Diego Luis Medina, et al. "A gene network regulating lysosomal biogenesis and function." *Sci* 325 (2009): 473-477.
2. Settembre, Carmine, Chiara Di Malta, Vinicia Assunta Polito and Moises Garcia Arencibia, et al. "TFEB links autophagy to lysosomal biogenesis." *Sci* 332 (2011): 1429-1433.
3. Harris, Harry and David C. Rubinsztein. "Control of autophagy as a therapy for neurodegenerative disease." *Nat Rev Neurol* 8 (2012): 108-117.
4. Mizushima, Noboru and Masaaki Komatsu. "Autophagy: Renovation of cells and tissues." *Cell* 147 (2011): 728-741.
5. Decressac, Mickael, Bengt Mattsson, Pia Weikop and Martin Lundblad, et al. "TFEB-mediated autophagy rescues midbrain dopamine neurons from α-synuclein toxicity." Proc Natl Acad Sci 110 (2013): E1817-E1826.

***Address for Correspondence:** Yuan Klochkov, Department of Translational Research and Surgery, University of Pisa, 56126 Pisa, Italy; E-mail: yuan@klochkov.it*

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