OPEN ACCESS Freely available online

ISSN: 2161-1009
Biochemistry & Analytical Biochemistry

Opinion Article

Role of Lysosomes in Neurodegenerative Disease

Nuone Paulo*

Department of Cellular Biochemistry, University Medical Center Goettingen, Goettingen, Germany

DESCRIPTION

Rare genetic disorders only impact a small number of humans, but because their etiologies are frequently understood, it is possible to build accurate animal models and conduct physiopathology research. A set of rare diseases known as lysosomal storage disorders are caused by a fundamental change in lysosome activity. The frequent occurrence of neurological signs in these illnesses brought attention to the role played by lysosomes in neurodegeneration. The same is true for other subgroups of uncommon neurodegenerative disorders. Thanks to hydrolytic enzymes, lysosomes join with autophagosomes and endosomes to enable the breakdown of their contents. Recently, it has become clear that modification of the autophagy-lysosome pathway may be a key factor in the death of neurons in a variety of neurodegenerative illnesses [1].

They demonstrate that a variety of modifications to the autophagy-lysosome pathway are related to neuronal death using a number of rare neurodegenerative illnesses. However, the majority of the time, it is still unknown why altering this pathway can cause neurodegeneration. Macroautophagy, also known as autophagy in this context, is a conserved mechanism that enables the mass destruction of macromolecules or organelles by delivering them to lysosomes. An autophagosome is formed when cellular substrates that need to be broken down are absorbed by a double-membrane compartment. Following its union with the degradative organelles, lysosomes, the latter's content is then broken down by hydrolytic enzymes. This cellular mechanism is crucial for the development of novel treatments because it has been linked to numerous neurodegenerative disorders [2].

Although lysosomes are essential to this system, it is yet unclear how autophagy is altered in many neurodegenerative disorders. Numerous rare neurodegenerative disorders with known genetic alterations are among the illnesses that exhibit lysosome function impairment. Developing treatments for each pathology is difficult due to the diversity of diseases and underlying mutations. However, the understanding of the genetic causes of these illnesses offers a chance to look into the factors that affect the autophagy-lysosome pathway. In this regard, Lysosomal Storage Disorders (LSDs) are a class of illnesses that primarily disrupt lysosomal function and are frequently accompanied by defective autophagy [3].

Neurological symptoms are frequently seen by LSD patients, emphasising the critical function of lysosomes in maintaining neuronal viability. Lysosomal malfunction and reduced autophagy, though, are also symptoms of other uncommon neurodegenerative illnesses, either as a fundamental failure or as a result of changes to other relevant pathways. They don't want to give a comprehensive list of rare disorders with lysosomal dysfunction in this study; instead, we've chosen a few cases to illustrate the range of changed functions and the numerous lysosome-autophagy pathway components that can be affected [4].

Lysosome dysfunction's effects on the autophagy pathway

Since they destroy the autophagic material after fusion with autophagosomes, lysosomes are essential components of the autophagy system. Once more, LSD aided in understanding the processes connecting lysosome malfunction to a dysfunctional autophagy pathway. A decrease in the autophagic flux, which measures the rate at which the substance is destroyed through autophagy, as well as an increase of autophagosomes and the autophagic substrate p62 are all symptoms of most LSD caused by lysosomal dysfunction. Similar changes are also seen in the majority of uncommon neurodegenerative disorders. Nevertheless, despite these similarities, defects in autophagy can result from dysfunction at various stages of the process, such as the formation of autophagosomes, the fusion of autophagosomes with lysosomes, the degradation of the autophagosomes, or the recovery of the lysosome membrane following the completion of autophagy [5].

With definition, only a tiny number of patients are affected by rare neurodegenerative illnesses, making it difficult to design a specific therapy strategy for each condition. Instead, it would make more sense to create a limited number of therapeutic

Correspondence to: Nuone Paulo, Department of Cellular Biochemistry, University Medical Center Goettingen, Goettingen, Germany, E-mail: nuonepaulo@cel.de

Received: 23-Nov-2022, Manuscript No. BABCR-22-19348; **Editor assigned:** 28-Nov-2022, Pre QC No. BABCR-22-19348 (PQ); **Reviewed:** 13-Dec-2022, QC No. BABCR-22-19348; **Revised:** 21-Dec-2022, Manuscript No. BABCR-22-19348 (R); **Published:** 29-Dec-2022, DOI: 10.35248/2161-1009.22.11.470.

Citation: N Paulo (2022) Role of Lysosomes in Neurodegenerative Disease. Biochem Anal Biochem. 11:470.

approaches that can be used to treat certain subgroups of rare diseases that have at least some shared physiopathology. Numerous uncommon neurodegenerative illnesses are characterized by lysosome malfunction, which frequently impairs autophagy.

As was discussed here, a few changes, such as the buildup of autophagosomes or autolysosomes, are seen in a significant number of rare disorders. The mechanisms behind these cellular dysfunctions, though, are frequently obscure. Additionally, lysosomes are needed for more than only the destruction of autophagosomes. Additionally, they permit the breakdown of endocytosed macromolecules, and lysosome malfunction can hinder the endocytosis mechanism.

REFERENCES

1. Lammens W, Le Roy K, Van Laere A, Rabijns A, Van den Ende W. Crystal structures of Arabidopsis thaliana cell-wall invertase mutants in complex with sucrose. J Mol Biol. 2008;377(2):378-385.
2. Osiebe O, Adewale IO, Omafuvbe BO. Comparative studies on intracellular invertase from an hyperproducer (Abagboro yeast) and a commercial yeast (Saccharomyces carlsbergensis).
3. Sahin F. Using sand particles for the disruption of cell walls of gram-positive bacteria and mycobacteria. J Bacteriol Parasitol. 2016;7(290): 2.
4. Nelson N. A photometric adaptation of the somogyi method for the determination of glucose. J Biol Chem. 1944;153(2):375-380.
5. Bradford MM. A rapid and sensitive method for the quantitation of microgram quantities of protein utilizing the principle of protein-dye binding. Anal Biochem. 1976;72(1-2):248-254.