How Lysosomes Protect Themselves from Corrosion in High Acid Environments

Jinglin Lu(✉)

University of Washington, Seattle, WA 98105, US
lujinglin2020@outlook.com

Abstract. Lysosomes are important metabolic sensors for many substances' cell growth and metabolic processes are also the center of explanation of substances in cells. Mitochondria, likewise, serve the main metabolic center of the cell. These two things interact with each other to regulate cellular metabolism and are inextricably linked to neurodegenerative diseases and lysosomal storage disorders. With current knowledge and investigations, we know that the membrane of lysosomes is very susceptible to damage. They can be affected by oxidative stress and similar environmental factors, so they need to protect themselves from corrosion in a highly acidic environment by some specific means. Judging from the current research, there is still a blank in this field. The current study proposes the use of glycosylation to protect lysosomes, but the disadvantage of this method is that it has a certain instability, and under certain circumstances, lysosomes will still be corroded by acid. Through experiment, this paper discusses how lipocalin apolipoprotein D (ApoD) is protected in an acidic environment of the cell membrane of the lysosome, and through research and investigation, this study found that a protein called lipocalin apolipoprotein D (ApoD) that can effectively prevent lysosomes from being corroded by acidity.

Keywords: Glycosylation · lysosome · mitochondria · Apo D

1 Introduction

Lysosomes are organelles in animal cells, which contain a variety of high concentrations of acid hydrolases. There are basically 60 hydrolases that have been confirmed so far, among which the marker enzymes are acid phosphatase (ACP) [1] and three phosphatase-phosphatases (TMP), the main function is to play a role in digestion in cells, also have a defense function for the body, and can remove senescent cells and redundant organelles.

Lysosome is an indispensable thing for people. It not only bears the function of catabolism, but also controls the lifespan of cells. Lysosome can be positioned as the central cellular center that controls aging. Lysosomes are not only the place where cellular waste is processed, but also as regulators that stabilize cells. Lysosomes are also fragile, and they are very susceptible to corrosion in a high acid environment. Therefore, if the lysosome is dysfunctional, it will cause many diseases, including some neurological diseases, such as Alzheimer's disease and Parkinson's disease. Studies have shown that

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the pathogenesis of these two diseases is also related to lysosome damage, which may be caused by their corrosion in a high acid environment.

What is more worthy of our attention is that lysosomes are closely related to the mechanism of cell self-digestion. They can adjust the magazine or some damaged substances produced by cells in the process of aging, so lysosomes plays an important role in causing cell senescence.

This paper primarily investigates how lysosomes are protected from water thawing under the action of Apo D, and demonstrates the importance of ApoD in the intervention of some diseases. The importance of Apo D for the treatment of some diseases and their protective effect on lysosomes were proved by Immunol blot analysis, image analysis variety of diseases caused by abnormal lysosomal enzymes have been discovered, and some diseases have also been found to lead to an increase in the number of lysosomes, so the research on lysosomes has received increasing attention in recent years [2].

Alzheimer's disease is a complex polygenic type of disease, most the Alzheimer's disease is hereditary, which shows that under the action of environmental factors and other genetic factors, Alzheimer's disease There is a great chance that mutism will be passed on to the next generation [3]. Recent studies have shown that there is a strong relationship between Alzheimer's disease and lysosomes. In the absence of lysosomes, Alzheimer's disease is more likely to occur and may lead to worsening. In some extreme environments, such as water-soluble or highly acidic conditions, the lysosome is corroded by these conditions and disappears, leading to Alzheimer's disease. But there is a protein called Apod D, which can protect lysosomes under certain circumstances, thereby reducing the occurrence of disease.

2 Methodology

This experiment will observe the effect of Apod D on the lysosome and the final effect and change on Alzheimer's disease through the test PH value and the method of a genetic test. Whether Apod D played a decisive role was determined by studying mice aged 12 and 24 weeks, measuring their amyloid protein and the size of their brain masses. And in order to exclude genetic and environmental factors, the same variable group was set to avoid an experimental error.

2.1 Analysis of the Mechanism of Lysosome

Since the lysosome is full of hydrolases, why is its own membrane "survived"?

For this problem, the membrane proteins of lysosomes have been modified by glycosylation, and the disguised biofilms will successfully escape cannibalism and ultimately save themselves from danger. But even if the lysosome can take precautions, it still cannot change the fact that it is the most dangerous organelle in the cell. Once the lysosome leaks accidentally, the membrane of the lysosome can continuously transport the hydrogen ions in the cytoplasm into the lysosome, which creates an acidic environment inside, and the pH in the cytoplasm is close to neutral, so even if the lysosome can take precautions, it still cannot change the fact that it is the most dangerous organelle in the

cell. Once the lysosome leaks accidentally, the membrane of the lysosome can continuously transport the hydrogen ions in the cytoplasm into the lysosome, which creates an acidic environment inside, and the pH in the cytoplasm [4] is close to neutral, even if the acid hydrolase of the lysosome can successfully escape to the cytoplasm, it will lose its destructive power due to denaturation and inactivation. Therefore, the protective mechanism of glycosylation is still defective, so we need to find new ways to make up for such defects. Through research and investigation, there is a protein called Apo D, which shows a very stable structure in a high acid environment, and it is itself an antioxidant. This important discovery and research can be used for some lysosome-related diseases and provide patients with better treatment opportunities.

Lysosome contains enzymes that degrade various biological macromolecules. It is an extremely complex and delicate digestive system in cells that digests and decomposes various biological molecules. In a starvation state, lysosomes can even degrade the biological macromolecules of cells themselves, and the digested products pass through lysosomes and enter the cytoplasm to participate in normal cellular metabolism, absorption and utilization. In recent years, the most popular topic in the field of cell biology is apoptosis [5], which is related to individual growth, development, deformity, aging, and the occurrence and prevention of diseases. There is a disease called silicosis, which is caused by the destruction of the lysosomal membrane and the release of a large amount of hydrolytic enzymes. If the lysosome cannot work properly, the lysosome will lack an enzyme that breaks down glycolipids [6]. Yes, the glycolipids that should be degraded will be stored in the cells in large quantities, even reaching 100–300 times the normal level, causing metabolic disorders, making patients with low IQ, blindness and eventually premature death. Therefore, the insufficiency of lysosomes can also cause serious harm to health. Under normal conditions, intracellular lysosomes rupture and hydrolytic enzymes are released, leading to a process known as autolysis. Under the action of some non-physiological factors, the stability of lysosome is low, and the release of hydrolase will lead to cell lysis or tissue lysis [7]. For example, a disease called silicosis is caused by the destruction of the lysosomal membrane rather than the release of lysosomes due to the phagocytosis of silica dust particles into cells.

So how to prove that Apod D protein can protect the cell membrane of lysosome? This is well demonstrated by an experiment on Astroglial ApoD traffics through clathrin- and caveolin-dependent endocytic pathways and concentrates prominently in the late endosomal-lysosomal compartment. The experimenters used the fly's retina as a system to detect neural variations. In the Drosophila nervous system, there is a secreted substance called Glaz, which is a homolog of Apod D and can aggregate proteins well. Through this experiment, it can basically be proved that Apod D can slow down the oxidation reaction, which is a good indication that it can protect the cell membrane of lysosomes.

2.2 Research Methods

2.2.1 Genomic DNA Extraction and Microscopy

By drawing the venous blood of the mice in the experimental group, and using the salting-out method [8] to determine the genetic variables. Secondly, under the measurement of

the microscope, the starch anti-protein precipitation produced by different experimental groups was determined.

2.2.2 Lysosomal pH Measurement

To study acidic organelles such as lysosomes, eosinophilic pH fluorescent needles [9] can be used to measure them. This fluorescent needle emits yellow fluorescence when the pH value is relatively low, blue fluorescence when the pH value is relatively high, and selectively marks the acidic organelles in living cells. By calculating the ratio of emitted light, the corresponding pH value can be measure.

2.2.3 Immune Analysis

In order to detect the effect of Apod D apolipoprotein on brain tonic tumors, we need to analyze whether it has any effect on it through images. Image analysis is a good method. It works by extracting proteins from mouse brains onto a membrane called PVDF. The membranes are not solubilized by reagents, so they can be recycled well to look at the state of the protein at different times.

2.3 Research Results

Recent studies have shown that abnormal lysosomal function may be one of the predisposing factors for Ason. APod D is also one of the more influential determinants. Previous in vitro studies have found that Apod E can enhance autophagy, that is, the function of lysosomes, indicating that Apod E may be involved in the regulation of lysosome-related degradation functions.

Neuropathological and behavioral differences were also observed, and the effects of exogenous and endogenous Apod E3 and E4 on biological effects such as autophagy-lysosomal function were also observed. This experiment also revealed the molecular mechanism of Apod E's involvement in AD pathogenesis and provided a new target for drugs from the perspective of lysosome autophagy.

As a disease that has been studied by many scholars, Alzheimer's disease has not yet found out what the final pathogenesis is. Based on the current data, amyloid [10] anti protein deposition is a key factor in the pathogenesis of Alzheimer's disease. And these deposits are caused by lysosome deletion. Under the action of Apod D, lysosomes can survive with a specific probability. But for environmental factors and mice with different genes. The uncertain factor of gene is the limitation of this experiment, because it does not rule out the possibility that Apod D will have differential effects on different genetics and races [11].

3 Conclusion

This paper mainly studies the connection and influence between Alzheimer's disease and APod D apolipoprotein, the subject disease related to lysosome. Through research, it was found that APod D apolipoprotein can intervene in the disease and play a great role in protecting the lysosome, thereby reducing the production of amyloid and reducing the size of brain tumors [12]. This shows that Apod D apolipoprotein has a certain contribution to the protection of lysosome.

For example, Leukodystrophies that may be caused by the lack of lysosomes can be compared in the future by comparing the effects of glycosylation and Apod D apolipoprotein on these diseases [13]. And analyze their respective strengths and weaknesses. For the method of glycosylation, it has great instability, but Apod D apolipoprotein can complement this very well, so if these two methods can be combined, it should be able to intervene some lysosome-related diseases.

Through the research and experiments of the whole article on how lysosomes are not corroded in a high acid environment, it is found that the use of glycosylation is a relatively perfect method. In this trial, Alzheimer's disease was mainly used as the main research disease and object. In the course of the experiment, Apod D played a very decisive role [14]. So it should be possible to combine Apod D and glycosylation to help lysosomes survive better in an extremely high acid environment. However, this article only studies the effect of Apod D on Alzheimer's disease, and does not study other diseases caused by the lack of lysosomes [15]. This paper believes that the impact and performance of Apod D in other diseases can be more widely used in future research.

There is a very inextricably close relationship between lysosomes and cellular senescence [16]. So in the future research on lysosomes, people can focus on how to slow down the aging of cells by changing the shape of lysosomes, such as diet or exercise habits. This should be of great help in extending people's lifespan and fighting aging.

Authors' Contributions

This paper is independently completed by Jinglin Lu.

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