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INVESTIGATION OF MITOCHONDRIAL ALTERATIONS BY ELECTRON MICROSCOPY

Rokhatoy Abdullaevna Kamoldinova

Andijan State Medical Institute, Uzbekistan

Abstract: Mitochondria are essential organelles responsible for cellular energy production, regulation of apoptosis, and metabolic homeostasis. Their structural integrity directly reflects cellular health and function. This article presents a histological and ultrastructural analysis of mitochondrial alterations observed through transmission electron microscopy (TEM) under physiological and pathological conditions. The study highlights the morphological features of mitochondrial injury, including swelling, cristae disruption, and membrane rupture, which are characteristic of degenerative, hypoxic, and toxic cellular damage.

Keywords: mitochondria, electron microscopy, ultrastructure, oxidative stress, cristae, apoptosis, cellular pathology.

Introduction

Mitochondria are dynamic double-membrane-bound organelles that play a fundamental role in energy metabolism through oxidative phosphorylation. Beyond ATP production, mitochondria are involved in calcium signaling, apoptosis regulation, and the generation of reactive oxygen species (ROS). Structural integrity of mitochondria is critical for maintaining cellular viability; even minor alterations in mitochondrial morphology can reflect profound metabolic dysfunction.

Electron microscopy provides an unparalleled resolution for studying mitochondrial ultrastructure, allowing direct visualization of the inner and outer membranes, cristae organization, and matrix density. The analysis of mitochondrial morphology under various physiological and pathological conditions contributes to understanding the mechanisms of cell injury and death. Mitochondrial alterations have been documented in a wide range of diseases, including ischemia-reperfusion injury, diabetes mellitus, neurodegenerative disorders, and cancer.

This study aims to describe the morphological patterns of mitochondrial alteration as revealed by transmission electron microscopy and to interpret these findings in the context of cellular degeneration and adaptive response.

Materials and Methods

Ultrastructural examination was performed on tissue samples obtained from the liver, cardiac muscle, and brain cortex of adult laboratory animals. Specimens were fixed in 2.5% glutaraldehyde in phosphate buffer (pH 7.4) and post-fixed in 1% osmium tetroxide. Following dehydration in graded ethanol, samples were embedded in epoxy resin. Ultrathin sections (60–90 nm) were stained with uranyl acetate and lead citrate and examined using a **JEOL 1400 transmission electron microscope**.

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Morphological parameters analyzed included mitochondrial size, shape, matrix density, cristae integrity, and the presence of vacuolization or inclusion bodies. Comparative assessment was performed between control and experimentally induced hypoxic and oxidative stress groups.

Results

Under physiological conditions, mitochondria displayed uniform morphology with clearly defined double membranes, densely packed cristae, and homogeneous matrix density. In hepatocytes, mitochondria were oval with well-organized cristae extending perpendicularly to the inner membrane. Cardiac myocytes demonstrated elongated mitochondria aligned parallel to myofibrils, reflecting high energy demand.

In hypoxic and degenerative states, electron microscopy revealed significant structural deviations:

- **Swelling of mitochondria** with distended outer membranes and rarefied matrix;
- **Cristae fragmentation and disorganization**, leading to vacuolization;
- **Formation of dense granules** and inclusion bodies within the mitochondrial matrix;
- **Outer membrane rupture**, indicating irreversible damage and initiation of apoptotic pathways.

In severe oxidative stress, **megamitocondria**—abnormally enlarged mitochondria with disrupted internal structure—were frequently observed. Such alterations correlated with decreased ATP synthesis and increased ROS production.

Discussion

The morphological changes observed under electron microscopy correspond to functional impairment of mitochondria. Swelling and cristae loss reflect disruption of the inner membrane potential, while outer membrane rupture indicates the release of pro-apoptotic factors such as cytochrome c.
These structural transformations are closely linked to bioenergetic failure, oxidative stress, and calcium overload. The presence of megamitochondria suggests an adaptive, compensatory mechanism where organelles enlarge to maintain energy output, though this adaptation is often temporary and followed by degeneration.

Mitochondrial alterations are central in the pathogenesis of degenerative diseases. In cardiac tissue, mitochondrial dysfunction contributes to ischemic injury and heart failure; in neural tissues, it is associated with Parkinson's and Alzheimer's diseases. Similarly, hepatic mitochondria are particularly susceptible to toxic damage from xenobiotics and free radicals.

Electron microscopy remains the gold standard for identifying early and ultrastructural signs of mitochondrial damage. Correlating morphological observations with biochemical and molecular findings enables a comprehensive understanding of mitochondrial pathophysiology.

Conclusion

Electron microscopy provides crucial insights into the structural dynamics of mitochondria under both physiological and pathological conditions. The characteristic ultrastructural alterations—swelling, cristae disruption, and membrane rupture—serve as hallmarks of mitochondrial dysfunction and cell injury.

Understanding these morphological signatures enhances our ability to diagnose, monitor, and interpret cellular responses to hypoxia, oxidative stress, and toxic insults. Future studies integrating **electron microscopy with immunocytochemistry and molecular imaging techniques** will deepen our knowledge of mitochondrial pathology and open new perspectives for targeted therapeutic intervention. Preservation of mitochondrial integrity should be recognized as a key factor in maintaining overall cellular and tissue health.

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