Healthy Aging Research

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Perspective

Mitochondrial Theory of Aging

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DESCRIPTION

The free radical and non-free radical versions of the mitochondrial theory of aging exist. One of the versions of the free radical hypothesis of aging is the first. It was created by J. Miquel and associates and expanded upon by Linnane and associates. A. N. Lobachev suggested the second.

According to the mitochondrial free radical theory of aging, cellular components are harmed by free radicals generated by mitochondrial action, which causes cellular aging. Cell organelles known as mitochondria work to produce ATP, which gives the cell energy. Reactive oxygen species, or ROS for short, are created when electrons from the mitochondrion escape and interact with water during ATP synthesis. ROS have the ability to damage macromolecules including lipids, proteins, and DNA, which is thought to speed up the aging process.

Molecular basis

It is believed that mitochondria are organelles that evolved from endocytosed bacteria that discovered how to coexist inside prehistoric cells. The mitochondrial DNA, which codes for elements of the electron transport chain, was kept alive by these bacteria. The inner mitochondrial membrane contains the electron transport chain (ETC), which generates ATP molecules as a type of energy. Because ATP is created from ADP *via* a sequence of redox reactions, the procedure is known as oxidative phosphorylation.

ROS: Superoxide, hydrogen peroxide, and hydroxyl radical are only a few of the chemical species that make up ROS highly reactive, oxygen-containing species. If the ETC complexes are not working properly, electrons may leak and interact with water to produce ROS. Normal ROS levels are maintained with minimum leakage, serving physiological functions in signaling and homeostasis. In fact, their presence at low levels lengthens life by activating transcription factors and aging-related metabolic pathways.

Mitochondrial metabolites: Different metabolites are frequently restricted to the mitochondria because the tricarboxylic acid cycle (TCA) occurs in the mitochondrial matrix. As we age, mitochondrial function deteriorates, allowing these metabolites to escape and cause age-related epigenetic changes.

In order to produce energy, acetyl-coenzyme A enters the mitochondrial matrix's TCA cycle and is oxidized. It can serve as a substrate for histone acetylation once it enters the nucleus after departing the mitochondria. Gene activation is brought on by the epigenetic alteration of histone acetylation. Acetyl-CoA levels are higher in the nucleus and cytoplasm at a young age and its transfer to the nucleus can lengthen worm lifetime.

DAMPs: When a cell is under stress, chemicals called damage-associated molecular patterns are released. A DAMP that is only made accessible by mitochondrial damage is mitochondrial DNA. Age-related increases in blood mitochondrial DNA levels contribute to inflame-aging, a chronic inflammatory condition that is a hallmark of old age.

Mitochondrial-derived peptides: 13 proteins have been found to be encoded by mitochondrial DNA. Other recently discovered short protein coding sequences are known as mitochondria-derived peptides, and their byproducts.

Alzheimer's disease, which is regarded as an age-associated disease, is protected from by the peptide humanin, which is generated from mitochondria. Age-related insulin resistance, which is a major contributor to type 2 diabetes, is prevented by MOTS-c. As people get older, their levels of humanin and MOTS-c drop, while their activity seems to lengthen lives.

Mitochondrial membrane: Almaida-Pagan and colleagues discovered that the lipid composition of the mitochondrial membrane changes with aging. With aging, the percentage of polyunsaturated fatty acids increased and the percentage of monounsaturated fatty acids dropped. With aging, the total amount of phospholipids also reduced.

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