Development or disease: duality of the mitochondrial permeability transition pore

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Mitochondria is not only a dynamic organelle that produces ATP, but is also an important contributor to cell functions in both development and cell death processes. These paradoxical functions of mitochondria are partially regulated by the mitochondrial permeability transition pore (mPTP), a high-conductance channel that can induce loss of mitochondrial membrane potential, impairment of cellular calcium homeostasis, oxidative stress, and a decrease in ATP production upon pathological activation. Interestingly, despite their different etiologies, several neurodegenerative diseases and heart ischemic injuries share mitochondrial dysfunction as a common element. Generally, mitochondrial impairment is triggered by calcium deregulation that could lead to mPTP opening and cell death. Several studies have shown that opening of the mPTP not only induces mitochondrial damage and cell death, but is also a physiological mechanism involved in different cellular functions. The mPTP participates in regular calcium-release mechanisms that are required for proper metabolic regulation; it is hypothesized that the transient opening of this structure could be the principal mediator of cardiac and brain development. The mPTP also plays a role in protecting against different brain and cardiac disorders in the elderly population. Therefore, the aim of this work was to discuss different studies that show this controversial characteristic of the mPTP; although mPTP is normally associated with several pathological events, new critical findings suggest its importance in mitochondrial function and cell development.

Calcium

Cardiac development

Mitochondria

Mitochondrial permeability transition pore

Neuronal development

Oxidative stress

mitochondrial permeability transition pore

adenosine triphosphate

calcium

carrier protein

mitochondrial permeability transition pore

Alzheimer disease

excitotoxicity

heart development

heart muscle ischemia

Huntington chorea

nervous system development

nonhuman

Parkinson disease

priority journal

protein function

Review

animal

biosynthesis

cardiac muscle cell

cardiomyopathy

cytology

degenerative disease

embryology

growth, development and aging

heart

human

metabolism

mitochondrial membrane potential

mitochondrion

mouse

oxidative stress

pathology

physiology

Adenosine Triphosphate

Animals

Calcium

Cardiomyopathies

Heart

Humans

Membrane Potential, Mitochondrial

Mice

Mitochondria

Mitochondrial Membrane Transport Proteins

Myocytes, Cardiac

Neurodegenerative Diseases

Oxidative Stress