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Perspective

The Significance of Control of Metabolism in Acute Ischemic Stroke

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DESCRIPTION

In the pathophysiological mechanisms of acute ischemic stroke, mitochondria play a vital role. When cerebral blood flow is impaired during an acute ischemic stroke, oxygen and glucose delivery are disrupted, which impairs mitochondrial oxidative phosphorylation and causes cellular bioenergetic stress. The mitochondrial unfolded protein response, mitochondrial fission and fusion, mitophagy, mitochondrial biogenesis, and intercellular mitochondrial transfer are a few examples of the mitochondrial quality control systems that cells might activate in response to such stress. Collectively, these adaptive response techniques assist maintain the mitochondrial network's structure and functionality, restoring the neurovascular unit's homeostasis in the process. This study discusses about the mechanisms of mitochondrial quality control in acute ischemic stroke. When these systems fail in acute ischemic stroke, an improved understanding of how these regulatory pathways maintain mitochondrial homeostasis will give a justification for creating novel neuroprotectants.

The indication restoration of blood flow to save oxygen-starved tissues is the main treatment objective for individuals with Acute Ischemic Stroke (AIS). Numerous studies have shown that intravenous thrombolysis with recombinant tissue Plasminogen Activator (t-PA) is an efficient first-line therapy for AIS. The short time frame and poor rate of reperfusion, however, have significantly hampered its use in clinical settings. 2015 saw the beginning of the Endovascular Thrombectomy (ET) era of extremely successful reperfusion therapy for Acute Coronary Syndrome (AIS) with the release of five good clinical trials. Therefore, it has been suggested that a future approach to treating AIS may involve combining these reperfusion treatments with neuroprotective measures.

The phospholipid bilayer membrane of mitochondria isolates the cytosol from their internal conditions. Water, ions, food molecules, Adenosine Diphosphate (ADP), and ATP are only a few of the substances that may pass through the Outer Mitochondrial Membrane (OMM)'s many protein channels. The Inner Mitochondrial Membrane (IMM) is home to the mitochondrial Electron Transport Chain (ETC), whose primary job is to produce ATP from electron donors such reduced Nicotinamide Adenine Dinucleotide (NADH) through several redox processes, providing cells with useable energy. Proton pumps (complexes I, III, and IV) move protons out of the mitochondrial matrix and into the Inter Membrane Space (IMS) during these redox processes. This creates the proton motive force and membrane potential (m) needed by complex V to produce ATP. However, throughout these processes, a tiny portion of electrons, particularly from complexes I and III, may escape from the ETC and interact with O_2 to produce superoxide. When the body is functioning normally, endogenous antioxidant mechanisms can eliminate Reactive Oxygen Species (ROS) and keep within the usual range, where ATP synthesis is effective but ROS creation is modest.

Insufficient oxygen and glucose provide during brain ischemia prevents mitochondrial respiration. According to several studies, the activity of mitochondrial ETC is decreased by 45-60% in focal tissues and by 15-40% in perifocal tissues after 2 hours of cerebral ischemia leading in a reduction in ATP synthesis and probably an increase in ROS. The destiny of the mitochondria depends on how long the ischemia lasts; whereas mitochondrial respiration can recover after a brief period of ischemia, chronic ischemia results in irreversible mitochondrial damage. After a protracted period of cerebral ischemia, oxygen quickly enters and refills the brain tissue, but the resulting surge in ROS production further exacerbates oxidative brain damage. It was initially discovered that succinate buildup in ischemic tissues might activate complex I to facilitate reverse electron transport.

Excessive production of ROS causes lipid, protein, and DNA oxidative damage. The Apoptosis-Inducing Factor (AIF), which is bound by PAR polymers, triggers caspase-independent apoptosis when oxidative DNA damage activates poly (ADP-ribose) (PAR) polymerase-1. Additionally, oxidative stress might encourage the translocation of Bax, an anti-apoptotic Bcl-2 family member, to the mitochondria and start caspase-dependent apoptosis. It's interesting to note that oxidative signalling can alter the mitochondrial network and function, indicating that the MtQC and pathological stress have a close bidirectional interaction.

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