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Perspective

Mitochondrial Dynamics and Bioenergetics Role in Stimulating Neurogenesis in Hippocampal Progenitor Cells

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DESCRIPTION

Neurogenesis, the formation of new neurons from neural progenitor or stem cells, is an essential element of brain plasticity. The hippocampus, a region known for its essential role in learning, memory and emotion, retains the ability to generate neurons throughout life. Mitochondria, often referred to as the cell's powerhouse, serve an essential function in regulating cellular energy. In hippocampal progenitor cells, mitochondrial dynamics and bioenergetic processes play a vital role in promoting neurogenesis. Understanding the interaction between mitochondrial function and neurogenesis may offer insights into cognitive health and age-related neurological decline. Here, we examine how mitochondrial dynamics and bioenergetics contribute to the stimulation of neurogenesis in hippocampal progenitor cells.

Mitochondria are highly dynamic organelles, continuously undergoing processes of fusion and fission to maintain their functional integrity and adaptability. These processes are integral to cellular bioenergetics, supporting the energy requirements of cells. In hippocampal progenitor cells, the ability of mitochondria to meet energy demands affects their proliferation, differentiation and survival. During neurogenesis, mitochondrial bioenergetics must be well-regulated to support cellular processes, including the synthesis of macromolecules and membrane structures that are necessary for cell division and differentiation into mature neurons.

Hippocampal progenitor cells, which are precursors to neurons, rely on mitochondrial energy production in the form of Adenosine Triphosphate (ATP). ATP generation through oxidative phosphorylation provides the energy needed to support the biochemical reactions that govern neurogenesis. Additionally, mitochondria influence cellular calcium homeostasis and produce Reactive Oxygen Species (ROS), which can act as signaling molecules in moderate concentrations. The tight regulation of these mitochondrial functions allows progenitor cells to proceed through the stages of neurogenesis without experiencing oxidative damage.

The processes of mitochondrial fusion and fission are essential for maintaining the functional integrity and adaptability of mitochondria. Fusion allows mitochondria to combine their contents, mitigating damage by sharing mitochondrial DNA and proteins, while fission enables the removal of damaged mitochondria through mitophagy. The balance between fusion and fission is essential in hippocampal progenitor cells, where mitochondrial health directly impacts cell viability and proliferation.

Mitochondrial fusion is primarily facilitated by Mitofusins (Mfn1 and Mfn2) and Optic atrophy 1 (Opa1) proteins, which regulate the merging of mitochondrial membranes. Fusion supports neurogenesis by promoting a healthy mitochondrial network, enabling efficient ATP production. In contrast, mitochondrial fission, governed by Dynamin-related protein 1 (Drp1), divides mitochondria, which aids in the isolation and removal of damaged components. Elevated Drp1 activity is linked to increased mitochondrial fission, a process that may be essential for preparing progenitor cells for mitosis and subsequent differentiation.

An imbalance in fusion and fission processes can disrupt mitochondrial function and bioenergetics, ultimately impairing neurogenesis. Excessive fission, for instance, may lead to increased mitochondrial fragmentation, reducing ATP production efficiency and potentially affecting progenitor cell survival. Conversely, promoting fusion may enhance mitochondrial function, thereby supporting neurogenesis through improved energy availability.

Mitochondrial bioenergetics refers to the metabolic processes that mitochondria employ to generate ATP and meet cellular energy demands. In hippocampal progenitor cells, ATP demand is high due to the rapid cell division and differentiation that accompany neurogenesis. Glycolysis and oxidative phosphorylation are two primary pathways for ATP production, with progenitor cells

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relying on a shift in metabolic profile as they differentiate into neurons.

During the proliferation stage, hippocampal progenitor cells primarily utilize glycolysis for ATP production. This metabolic choice supports rapid cell division, as glycolysis is less reliant on oxygen and can quickly generate ATP, even if at a lower yield than oxidative phosphorylation. Upon differentiation, cells increase their reliance on oxidative phosphorylation, a shift that corresponds with the maturation of mitochondrial networks and the increasing energy demands of mature neurons. This metabolic shift enables cells to produce ATP more efficiently, supporting neuronal functions such as synaptic transmission and membrane potential maintenance.

Oxidative phosphorylation takes place within the mitochondrial inner membrane, where Electron Transport Chain (ETC) complexes generate ATP through a process that involves electron transfer and proton gradient establishment. For hippocampal progenitor cells to complete differentiation successfully, their mitochondria must function efficiently, producing ATP in sufficient quantities to meet increased energy requirements. Dysfunctional mitochondria that fail to meet these bioenergetic demands can compromise the neurogenesis process, potentially affecting cognitive function and memory formation in the hippocampus.

Beyond their bioenergetic function, mitochondria play a role in signaling pathways that influence neurogenesis. Mitochondria are involved in calcium signaling and ROS production, both of which can affect the expression of genes involved in cell cycle regulation, differentiation and survival. Calcium, a key second messenger, regulates the activity of several enzymes and transcription factors. Mitochondria help maintain cellular calcium balance, thereby affecting calcium-dependent signaling pathways.

Calcium influx into mitochondria occurs through the Mitochondrial Calcium Uniporter (MCU) complex, which responds to changes in cytosolic calcium levels. Controlled calcium uptake by mitochondria influences the activation of neurogenic transcription factors, thereby promoting gene expression necessary for differentiation. Dysregulation of mitochondrial calcium handling can impair the signaling pathways essential for neurogenesis, ultimately affecting hippocampal function.