Short Communication
Volume 08:04, 2025

Journal of Brain Research

ISSN: 2684-4583

Open Access

Brain Energy Metabolism in Neurological Disorders

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Introduction

Altered brain energy metabolism, particularly in glucose utilization and mitochondrial function, is a significant contributor to the pathogenesis of various neurological disorders. Dysregulation in these processes can lead to neuronal dysfunction, neuroinflammation, and ultimately, neurodegeneration. Targeting these metabolic pathways offers promising therapeutic avenues for conditions like Alzheimer's disease, Parkinson's disease, and epilepsy [1].

Mitochondrial dysfunction, a key hallmark in many neurodegenerative diseases, directly impairs ATP production, exacerbates oxidative stress, and triggers apoptotic pathways in neurons. Research highlights specific mitochondrial protein dysregulations and the potential of pharmacological interventions aimed at restoring mitochondrial homeostasis [2].

Glucose hypometabolism in specific brain regions is a consistent finding in Alzheimer's disease, preceding overt neuronal loss. This metabolic deficit impacts synaptic function and neuronal viability, suggesting that enhancing glucose uptake and utilization could be a strategy to slow disease progression [3].

Epilepsy is characterized by recurrent seizures, and altered neuronal excitability often stems from disruptions in energy metabolism, particularly affecting ion homeostasis. Strategies to stabilize neuronal energy levels and buffer against metabolic stress show promise in reducing seizure frequency [4].

Neuroinflammation, a common factor in many neurological conditions, can significantly impact brain energy metabolism by altering glial cell function and energy substrate availability. Understanding this interplay is crucial for developing therapies that target both inflammation and metabolic deficits [5].

Parkinson's disease is associated with impaired mitochondrial complex I activity and reduced ATP production in dopaminergic neurons. Oxidative stress further exacerbates this metabolic vulnerability, making neurons susceptible to degeneration [6].

Therapeutic strategies focusing on metabolic interventions, such as ketogenic diets or specific nutrient supplementation, are being explored to support brain energy metabolism in neurodegenerative disorders. These approaches aim to provide alternative fuel sources and enhance cellular resilience [7].

Astrocytes play a critical role in brain energy homeostasis by providing neurons with lactate, a crucial energy substrate. Dysfunctional astrocyte-neuron lactate shuttling has been implicated in various neurological diseases, impacting neuronal function and survival [8].

The brain's high energy demand makes it particularly vulnerable to metabolic insults. Maintaining a stable supply of glucose and oxygen, along with efficient mitochondrial function, is paramount for neuronal health and cognitive function. Disruptions at any stage of this complex process can trigger or exacerbate neurological disorders [9].

Investigating novel biomarkers for brain energy metabolism could lead to earlier and more accurate diagnosis of neurological disorders. Positron emission tomography (PET) imaging of glucose and oxygen metabolism, along with analysis of specific metabolic enzymes and metabolites, are promising avenues [10].

Description

Altered brain energy metabolism, particularly in glucose utilization and mitochondrial function, is a significant contributor to the pathogenesis of various neurological disorders. Dysregulation in these processes can lead to neuronal dysfunction, neuroinflammation, and ultimately, neurodegeneration. Targeting these metabolic pathways offers promising therapeutic avenues for conditions like Alzheimer's disease, Parkinson's disease, and epilepsy [1].

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Conclusion

Neurological disorders are significantly influenced by altered brain energy metabolism, involving glucose utilization and mitochondrial function. Disruptions in these processes lead to neuronal dysfunction, neuroinflammation, and degeneration, presenting therapeutic targets for diseases like Alzheimer's, Parkinson's, and epilepsy. Mitochondrial dysfunction impairs ATP production and increases oxidative stress, while glucose hypometabolism is a marker in Alzheimer's disease, affecting synaptic function. Epilepsy involves energy metabolism disruptions impacting neuronal excitability, and neuroinflammation exacerbates metabolic deficits. Parkinson's disease is linked to impaired mitochondrial complex I activity and reduced ATP in dopaminergic neurons. Therapeutic strategies include metabolic interventions like ketogenic diets and nutrient supplementation. Astrocytes are vital for energy homeostasis through lactate shuttling, and its dysfunction contributes to disease. Maintaining stable energy supply and efficient mitochondrial function is crucial for brain health, with disruptions leading to neurological issues. Novel biomarkers for brain energy metabolism, such as PET imaging, are being explored for early diagnosis.

Acknowledgement

None.

Conflict of Interest

None.

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How to cite this article: Al-Zahra, Fatima. "Brain Energy Metabolism in Neurological Disorders." *J Brain Res* 08 (2025):329.

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Received: 01-Aug-2025, Manuscript No. jbr-26-182902; **Editor assigned:** 04-Aug-2025, PreQC No. P-182902; **Reviewed:** 18-Aug-2025, QC No. Q-182902; **Revised:** 22-Aug-2025, Manuscript No. R-182902; **Published:** 29-Aug-2025, DOI: 10.38421/2684-4583.2025.8.329