Neurocellular metabolism: Energy production and utilization in the brain.

Yu Parkhomenko*

Department of Biochemistry, National Academy of Sciences of Ukraine, Kyiv, Ukraine

Introduction

Neurocellular metabolism, the intricate network of biochemical processes within neurons, plays a fundamental role in sustaining the energy demands of the brain. Despite comprising only a small fraction of the body's mass, the brain consumes a disproportionate amount of energy, highlighting the importance of efficient metabolic pathways in neuronal function. In this article, we delve into the complexities of neurocellular metabolism, exploring its role in brain homeostasis, synaptic activity, and neurological disorders [1].

Energy production in neurons

Neurons, like all cells, rely on Adenosine Triphosphate (ATP) as a primary source of energy. Glucose serves as the main fuel for ATP production in the brain, undergoing glycolysis and oxidative phosphorylation within neuronal mitochondria [2]. Glucose transporters facilitate the uptake of glucose into neurons, where it is metabolized to pyruvate through glycolysis. Pyruvate enters the tricarboxylic acid (TCA) cycle within mitochondria, generating reducing equivalents that fuel ATP synthesis through oxidative phosphorylation [3].

Astrocyte-neuron metabolic coupling: Astrocytes, the supportive cells of the brain, play a crucial role in neurocellular metabolism through metabolic coupling with neurons. Glucose taken up by astrocytes undergoes glycolysis, with lactate being produced as a byproduct. Lactate is then shuttled to neurons, where it serves as an additional energy substrate [4]. This astrocyte-neuron lactate shuttle (ANLS) represents a key metabolic interaction between astrocytes and neurons, ensuring the efficient utilization of energy substrates during neuronal activity [5].

Metabolic regulation of synaptic transmission: Neurocellular metabolism is tightly linked to synaptic activity, with metabolic pathways dynamically modulating neurotransmitter release and synaptic plasticity. ATP generated through oxidative phosphorylation fuels the maintenance of ion gradients essential for synaptic transmission. Moreover, metabolic intermediates, such as α-ketoglutarate and acetyl-CoA, serve as substrates for neurotransmitter synthesis and epigenetic modifications, respectively, influencing synaptic function and plasticity [6].

Neurocellular metabolism in neurological disorders

Dysregulation of neurocellular metabolism contributes to the pathogenesis of various neurological disorders, including Alzheimer's disease, Parkinson's disease, and epilepsy [7]. Metabolic impairments, such as mitochondrial dysfunction and glucose hypometabolism, disrupt neuronal energy production, leading to synaptic dysfunction and neurodegeneration. Targeting metabolic pathways represents a promising approach for therapeutic intervention in these disorders, aiming to restore energy homeostasis and mitigate neuronal damage [8].

Emerging insights and future directions: Recent advances in neuroimaging techniques and metabolic profiling have provided novel insights into neurocellular metabolism, allowing researchers to elucidate the metabolic alterations associated with brain function and dysfunction [9]. Moreover, emerging technologies, such as optogenetics and metabolomics, offer powerful tools for studying the dynamic regulation of neurocellular metabolism in health and disease. Future research endeavors will likely focus on deciphering the molecular mechanisms underlying neurocellular metabolic regulation and developing targeted therapies for neurological disorders [10].

Conclusion

Neurocellular metabolism lies at the intersection of energy production, synaptic function, and brain health, playing a crucial role in sustaining neuronal activity and viability. By unraveling the complexities of neurocellular metabolism, researchers aim to elucidate the mechanisms underlying brain function and dysfunction, paving the way for innovative therapeutic strategies for neurological disorders. Through interdisciplinary collaborations and technological advancements, the exploration of neurocellular metabolism continues to expand, offering new avenues for understanding and treating brain-related conditions.

References

1. Gesell R, Hansen ET, Siskel J. On the electrotonic nature of stimulation, inhibition, summation and after-discharge of nerve centers. Am J Physiol Cell Physiol. 1947;148(2):515-29.
2. Pavlova O, Stepanenko S, Chehivska L, et al. Thiamine deficiency in rats affects thiamine metabolism possibly through the formation of oxidized thiamine pyrophosphate. Biochimica et Biophysica Acta (BBA)-General Subjects. 2021;1865(11):129980.

***Correspondence to:** Yu Parkhomenko, Department of Biochemistry, National Academy of Sciences of Ukraine, Kyiv, Ukraine, E-mail: Yu@biochem.kiev.ua

***Received:** 04-Feb-2024, Manuscript No. AACBM-24-130177; **Editor assigned:** 06-Feb-2024, PreQC No. AACBM-24-1301775(PQ); **Reviewed:** 20-Feb-2024, QC No AACBM-24-1301775; **Revised:** 23-Feb-2024, Manuscript No. AACBM-24-1301775(R); **Published:** 28-Feb-2024, DOI:10.35841/aacbm-6.1.187*

***Citation:** Parkhomenko Y. Neurocellular metabolism: Energy production and utilization in the brain. J Cell Biol Metab. 2024;6(1):187*

3. Gómez Á, Cerdán S, Pérez-Laso C, et al. Effects of adult male rat feminization treatments on brain morphology and metabolomic profile. Horm Behav. 2020;125:104839.
4. Lipkin LE. Cytoplasmic inclusions in ganglion cells associated with parkinsonian states: a neurocellular change studied in 53 cases and 206 controls. Am J Pathol. 1959;35(6):1117.
5. Wakutani Y, Kowa H, Kusumi M, et al. Genetic analysis of vascular factors in Alzheimer's disease. Ann NY Acad Sci. 2002;977(1):232-8.
6. Li Y, Wang M, Wang S. Effect of inhibiting mitochondrial fission on energy metabolism in rat hippocampal neurons during ischemia/reperfusion injury. Neurol Res. 2016;38(11):1027-34.
7. Klein RC, de Jong BM, de Vries JJ, et al. Direct comparison between regional cerebral metabolism in progressive supranuclear palsy and Parkinson's disease. J Mov Disord. 2005;20(8):1021-30.
8. Stoppe G, Vedder H. The role of estrogens in the prevention and therapy of dementia. Reproduktionsmedizin. 2000;16:310-8.
9. Sloand JA, Shelly MA, Feigin A, et al. A double-blind, placebo-controlled trial of intravenous iron dextran therapy in patients with ESRD and restless legs syndrome. Am J Kidney Dis. 2004;43(4):663-70.
10. Trope I, Lopez-Villegas D, Lenkinski RE. Magnetic resonance imaging and spectroscopy of regional brain structure in a 10-year-old boy with elevated blood lead levels. Pediatrics. 1998;101(6):e7.

Citation: *Parkhomenko Y. Neurocellular metabolism: Energy production and utilization in the brain. J Cell Biol Metab. 2024;6(1):187*