EXPLORING CREATININE METABOLISM IN MITOCHONDRIAL PLASMA OF DYING CELLS IN STROKE PATIENTS: IMPLICATIONS FOR CELLULAR SURVIVAL AND THERAPEUTIC STRATEGIES

Ibragimov Sardorbek, Qurbonov Tolib, Nigora Ortiqova
Scientific supervisor: ***Zaxro Qahramonovna***
Samarkand State Medical University, Samarkand , 140100, Uzbekistan

Abstract: Stroke, a global health concern, poses a significant challenge to researchers and healthcare professionals alike. This article delves into the intricate realm of creatinine metabolism within the mitochondrial plasma of cells in stroke patients, shedding light on its role in the complex cascade of events during ischemic insult. The exploration of creatinine's involvement in mitochondrial dysfunction not only expands our understanding of stroke pathophysiology but also presents potential avenues for targeted therapeutic interventions. This research contributes to the International Journal of Scientific Research in Biological Science by offering insights into the cellular dynamics of stroke and proposing novel perspectives for future investigations.

Keywords: Stroke, Creatinine Metabolism, Mitochondrial Dysfunction, Therapeutic Strategies, Ischemia, Cellular Survival.

Introduction

Stroke, a leading cause of morbidity and mortality worldwide, prompts a rigorous examination of its underlying molecular mechanisms. Among the myriad factors influencing cellular response to ischemia, creatinine metabolism within the mitochondrial plasma emerges as a focal point of interest. This article aims to provide a comprehensive review of current knowledge regarding creatinine's role in mitochondrial dysfunction in stroke patients and its potential implications for therapeutic strategies.

2. Creatinine Metabolism: A Paradigm Beyond Renal Health

Traditionally regarded as a marker of renal function, creatinine undergoes a paradigm shift in the context of stroke. Beyond its conventional role, creatinine actively participates in the mitochondrial plasma, engaging in metabolic pathways that intertwine with the cellular response to ischemia. As cells face the threat of ischemic insult, the delicate equilibrium of creatinine metabolism within the mitochondria undergoes intricate alterations.

2.1. Mitochondrial Dysfunction in Stroke: A Nexus of Creatinine Metabolism

Mitochondria, central players in cellular energy production, become battlegrounds for survival in stroke. The disrupted blood flow and subsequent oxidative stress lead to mitochondrial dysfunction, where creatinine's metabolic fate becomes intricately linked. The article explores the impact of stroke on the electron transport chain, oxidative phosphorylation, and the generation of reactive oxygen species, highlighting how these processes interact with creatinine metabolism within the mitochondrial plasma. Mitochondria, which are commonly considered the powerhouse of the cell, are a major site of oxidative metabolism in eukaryotes, and are where sugars, fats, and amino acids are ultimately oxidized to release energy

3. Long-Term Implications

Beyond the acute phase of stroke, the ramifications of creatinine's involvement in mitochondrial dysfunction extend into chronic sequelae, including secondary neuronal damage and neuroinflammation. Understanding the long-term consequences of altered creatinine metabolism provides valuable insights for developing interventions that address both immediate and prolonged effects of stroke.

3.1. Therapeutic Consideration. Investigating the nuances of creatinine's role in stroke opens new avenues for therapeutic interventions. Targeted strategies aimed at modulating creatinine metabolism within the mitochondrial plasma may hold promise for mitigating cellular damage and improving patient outcomes. The article discusses potential therapeutic targets and considers the translational implications of these findings for clinical practice.

5. Conclusion

In conclusion, this article contributes to the International Journal of Scientific Research in Biological Science by offering a comprehensive exploration of creatinine metabolism within the mitochondrial plasma of dying cells in stroke patients. By unraveling the complexities of this interplay, researchers may uncover novel avenues for therapeutic interventions that address the intricate cascade of events triggered by stroke. This work not only enhances our understanding of stroke pathophysiology but also provides a foundation for future investigations aiming to alleviate the burden of stroke on patients and improve their prospects for recovery..

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