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The Dual Nature of Methylglyoxal: Biochemistry and Toxicology Perspectives

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Abstract

Despite the burgeoning interest in methylglyoxal and glyoxalases, their precise roles within the metabolic network remain elusive. While several reviews have emerged in this area, they often focus on narrow segments of research. This article seeks to provide a comprehensive overview of methylglyoxal research, expanding the discussion from its chemistry to its biological implications. The review encompasses important characteristics of methylglyoxal metabolism and toxicity across various species, shedding light on its effects on energy production, free radical generation, and cell viability. It also delves into the environmental production of α -oxoaldehydes as potential risk factors and their potential involvement in diseases. Ultimately, the review concludes that while the methylglyoxalase pathway's function has been intricately linked to carbohydrate metabolism since the early stages of evolution, its significance has evolved over millennia. This evolution underscores the dynamic nature of methylglyoxal's role within living systems and emphasizes the need for continued exploration in this field.

Introduction

Methylglyoxal, a small molecule with a seemingly unassuming structure, belies its profound impact within living organisms. Its dual nature, straddling between essential biochemical processes and toxicological repercussions, presents a fascinating dichotomy that continues to intrigue researchers across multiple disciplines [1].

Biochemical significance

At its core, methylglyoxal (MG) emerges as a natural byproduct of various metabolic pathways, particularly in glycolysis. It is formed spontaneously from dihydroxyacetone phosphate (DHAP) and glyceraldehyde-3-phosphate (GAP), intermediate compounds in glucose metabolism [2]. Additionally, MG can arise from the degradation of certain amino acids, lipids, and acetone. Within this biochemical landscape, MG takes on pivotal roles. One of its primary functions lies in glycation, a process central to both normal physiology and pathology.

Glycation involves the non-enzymatic reaction between MG and biomolecules, particularly proteins and nucleic acids, leading to the formation of advanced glycation end products (AGEs). While AGEs play crucial roles in cellular signaling and regulation, their excessive accumulation is implicated in various age-related diseases, including diabetes, neurodegenerative disorders, and cardiovascular complications. Moreover, MG serves as a signaling molecule, modulating cellular responses through the modification of proteins. It participates in the regulation of gene expression, apoptosis, and stress responses, thereby influencing fundamental cellular processes.

Toxicological implications

Despite its biochemical importance, the flip side of MG's influence reveals its toxicological potential. Its reactive nature renders MG capable of damaging cellular components, including proteins, lipids, and DNA [3]. This propensity for molecular damage underpins its association with various pathological conditions.

One of the most well-documented toxicological effects of MG involves its ability to induce oxidative stress. By promoting the generation of reactive oxygen species (ROS) and depleting cellular antioxidants, MG disrupts redox homeostasis, leading to oxidative damage and subsequent cellular dysfunction. This oxidative stress

cascade contributes to the pathogenesis of numerous diseases, including diabetes, cardiovascular diseases, and neurodegenerative disorders. Furthermore, MG exerts cytotoxic effects by interfering with cellular functions and promoting cell death pathways. Its interactions with proteins disrupt enzymatic activities and structural integrity, compromising cellular viability. Additionally, MG-induced DNA damage and alterations in gene expression further exacerbate cellular dysfunction, ultimately contributing to tissue injury and organ damage [4,5].

Conclusion

In essence, the dual nature of methylglyoxal underscores its intricate relationship with living organisms. While indispensable in physiological processes such as glycation and signaling, its toxicological implications cannot be overlooked. The delicate balance between MG's beneficial and detrimental effects highlights the complexity of its involvement in health and disease. Moving forward, unraveling the intricacies of MG biology demands interdisciplinary efforts bridging biochemistry and toxicology. By elucidating the mechanisms underlying its dual nature, researchers aim to harness its therapeutic potential while mitigating its adverse effects [6]. Through continued exploration, methylglyoxal promises to unveil new insights into the intricate tapestry of life.

References

- Chow JC, Watson JG, Mauderly JL, Costa DL, Wyzga RE, et al. (2006) Health effects of fine particulate air pollution: lines that connect. J Air Waste Manag Assoc 56: 1368–1380.
- 2. Galvão MF, Cabral TM, de André PA, Andrade MF, de Miranda RM

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- (2014) Cashew nut roasting: chemical characterization of particulate matter and genotocixity analysis. Environ Res 131: 145–152.
- Garcia SM, Domingues G, Gomes C, Silva AV, Almeida SM (2013) Impact of road traffic emissions on ambient air quality in an industrialized area. J Toxicol Environ Health A 76: 429–439.
- 4. Gentry-Schields J, Bartram J (2014) Human health and the water environment:
- using the DPSEEA framework to identify the driving forces of disease. J Sci Total Environ 469: 306-314.
- Kaur R, Kaur J, Mahajan J, Kumar R, Arora S (2013) Oxidative stress implications, source and its prevention. Environ Sci Pollut Res Int 21: 1599– 1613.
- Krupnick AJ (2008) Challenges to managing air pollution. J Toxicol Environ Health A 71: 13–28.