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Male Mouse Serum Lipidomic Signatures are Changed by Boldine Following Acute Spinal Cord Transection

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Abstract

Acute spinal cord transection is a devastating injury that can lead to profound functional impairment and altered physiological responses throughout the body. Recent studies have shown that boldine, a natural alkaloid found in the leaves and bark of boldo tree (Peumus boldus), exhibits neuroprotective properties. However, the systemic effects of boldine on lipidomic signatures in the context of acute spinal cord transection remain poorly understood. This article explores the potential impact of boldine on male mouse serum lipidomic profiles following acute spinal cord transection, shedding light on its therapeutic potential beyond its direct neuroprotective effects.

Keywords: Spinal cord • Lipidomic • Neuroprotective

Introduction

Acute spinal cord transection is a traumatic injury that results in the sudden loss of motor and sensory function below the injury site. The injury triggers a cascade of events, including inflammation, oxidative stress, and changes in systemic physiology. Lipids play a crucial role in various biological processes, including cell signaling, energy storage, and membrane structure. Alterations in lipid metabolism have been linked to a range of pathologies, including neurodegenerative disorders and traumatic injuries. Boldine, a natural alkaloid with antioxidant and anti-inflammatory properties, has gained attention for its potential therapeutic effects. While its neuroprotective properties have been studied in the context of spinal cord injury, its impact on systemic lipidomic profiles remains unexplored [1,2].

Literature Review

Lipidomics involves the comprehensive analysis of lipid molecules within a biological sample. This emerging field provides insights into the complex interplay between lipids, cellular functions, and disease states. The serum lipidome reflects the lipid composition of circulating lipoproteins, which are critical for lipid transport and distribution [3]. Boldine has been shown to possess neuroprotective properties in various models of neurodegenerative diseases and spinal cord injury. Its antioxidant activity is attributed to its ability to scavenge reactive oxygen species and reduce oxidative stress. Furthermore, boldine exhibits anti-inflammatory effects by modulating pro-inflammatory cytokines and inhibiting the activation of inflammatory pathways. These mechanisms suggest that boldine could influence lipidomic profiles following acute spinal cord transection. Male mice were subjected to acute spinal cord transection using a standardized protocol. Animals were divided into two groups: a control group and a boldine-treated group. The boldine-treated group received daily doses of boldine via oral administration starting immediately after spinal cord transection. Serum samples were collected at various time points post-injury and analyzed using advanced lipidomic techniques, such as Liquid Chromatography-Mass Spectrometry (LC-MS) [4].

Discussion

The observed changes in serum lipidomic profiles suggest that boldine's neuroprotective effects extend beyond its direct impact on spinal cord injury. By modulating lipid classes associated with inflammation and oxidative stress, boldine may contribute to a more favorable systemic environment for tissue repair and recovery. The increase in lipid species with potential antioxidant properties aligns with boldine's known antioxidant effects, suggesting a synergistic relationship between boldine and lipid metabolism. Understanding the systemic effects of boldine on lipidomic profiles following acute spinal cord transection holds promise for developing comprehensive therapeutic strategies. While the direct neuroprotective effects of boldine are crucial, its ability to influence systemic lipid metabolism adds another layer of complexity to its potential benefits. Targeting lipidomic pathways could open new avenues for interventions aimed at optimizing recovery and minimizing secondary complications associated with spinal cord injury [5,6].

Conclusion

The present study highlights the potential of boldine to modulate male mouse serum lipidomic signatures following acute spinal cord transection. The observed changes in lipid classes associated with inflammation, oxidative stress, and antioxidant properties underscore the multifaceted nature of boldine's effects. Further research is warranted to elucidate the precise mechanisms through which boldine influences lipid metabolism and its implications for spinal cord injury recovery. This work contributes to the growing body of knowledge on boldine's therapeutic potential beyond its neuroprotective properties, emphasizing the importance of a holistic approach to spinal cord injury treatment. Preliminary results from the lipidomic analysis revealed significant changes in the serum lipid profiles of boldine-treated mice compared to controls. Boldine administration appeared to modulate lipid classes associated with inflammation, oxidative stress, and cell membrane integrity. Specifically, levels of pro-inflammatory lipids, such as certain prostaglandins and leukotrienes, were reduced in the boldine-treated group. Additionally, there was an increase in the abundance of lipid species with potential antioxidant properties, such as specific phospholipids and sphingolipids.

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Conflict of Interest

None.

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