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Rage and its Ligands, a Receptor for Advanced Glycation End Products, Attention to Spinal Cord Injury

Jyungr Aesd*

Department of Anatomy, Yonsei University College of Medicine, Seoul 120-752, Korea

Introduction

Spinal Cord Injury (SCI) is a complex pathological condition characterized by immediate mechanical damage and subsequent neuroinflammatory processes that contribute to secondary damage. The Receptor for Advanced Glycation End Products (RAGE) and its ligands play a critical role in mediating inflammation and oxidative stress, which are key contributors to secondary injury following SCI [1,2]. This review delves into the significance of RAGE activation and its ligands in the context of SCI, shedding light on potential therapeutic strategies targeting this pathway to mitigate neuroinflammation and enhance recovery. Spinal cord injury leads to profound functional deficits and challenges the neuroscientific community to develop effective treatments [3]. A growing body of evidence suggests that the RAGE pathway, comprising the receptor RAGE and its diverse ligands, is implicated in the pathophysiology of SCI. This review focuses on elucidating the roles of RAGE and its ligands in neuroinflammation, oxidative stress, and secondary injury cascades following SCI. Spinal cord injury is a complex trauma leading to irreversible neurological deficits. Mounting evidence suggests that RAGE, a multiligand receptor, and its ligands are implicated in various neurodegenerative disorders. This review aims to elucidate the contribution of the RAGE axis to spinal cord injury, emphasizing its role in inflammation, oxidative stress, and neuronal dysfunction [4].

Description

RAGE, a member of the immunoglobulin superfamily, binds to a variety of ligands, including Advanced Glycation End Products (AGEs), S100 proteins, and High-Mobility Group Box 1 (HMGB1). Activation of RAGE initiates proinflammatory signaling cascades, leading to increased oxidative stress, cytokine release, and cell death. The broad range of RAGE ligands reflects its multifaceted involvement in various pathological conditions, including SCI. Neuroinflammation is a hallmark of SCI and contributes to tissue damage and functional impairment. RAGE activation triggers the release of proinflammatory cytokines and chemokines, attracting immune cells to the site of injury. This exacerbates the inflammatory response and creates a cycle of damage and inflammation that worsens secondary injury [5]. Oxidative stress, resulting from an imbalance between Reactive Oxygen Species (ROS) production and antioxidant defenses, is a major driver of secondary injury following SCI. RAGE activation induces the generation of ROS, leading to lipid peroxidation, DNA damage, and protein oxidation. This oxidative burden further propagates inflammation and cellular damage. In SCI, RAGE activation triggers a robust inflammatory response, recruiting immune cells and exacerbating tissue damage. AGE-RAGE interactions stimulate microglial activation and astrocyte reactivity, fostering a proinflammatory milieu that contributes to secondary injury cascades. This chronic inflammation exacerbates neuronal damage and impairs tissue repair mechanisms [6].

Conclusion

Given the pivotal roles of RAGE and its ligands in neuroinflammation and oxidative stress, targeting this pathway holds promise for SCI treatment. Strategies to modulate RAGE expression or block its interaction with ligands, either through pharmacological agents or genetic interventions, offer potential therapeutic avenues. Inhibition of RAGE-mediated signaling could mitigate inflammation, reduce oxidative stress, and promote tissue repair. Translating the insights from preclinical studies to clinical applications is a critical challenge. Developing RAGE-targeted therapies requires rigorous evaluation of safety, efficacy, and long-term effects. Combining RAGE inhibition with other emerging therapeutic strategies, such as cell transplantation or neuroprotective agents, may offer synergistic benefits for SCI patients. The involvement of RAGE and its ligands in neuroinflammation and oxidative stress amplifies secondary injury mechanisms in spinal cord injury. Understanding the intricate roles of RAGE signaling offers new opportunities for targeted therapeutic interventions aimed at mitigating inflammation and improving functional outcomes for individuals affected by SCI. Interventions targeting the RAGE axis hold promise in mitigating SCI-associated pathologies. Inhibitors of RAGE activation, such as soluble RAGE or small molecules, could potentially attenuate inflammation, oxidative stress, and apoptosis. Additionally, targeting RAGE ligands, including AGEs and HMGB1, may offer therapeutic strategies to mitigate the consequences of SCI.

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Conflict of Interest

None.

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***Address for Correspondence:** Jyungr Aesd, Department of Anatomy, Yonsei University College of Medicine, Seoul 120-752, Korea, E-mail: jyungra@gmail.com*

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