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Chronic Stress and Neuroinflammation: Insights from Rodent Models

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Introduction

Chronic stress has long been recognized as a significant contributor to various physical and mental health disorders, exerting profound effects on the Central Nervous System (CNS) and neuroimmune function. In recent years, research using rodent models has provided invaluable insights into the intricate interplay between chronic stress and neuroinflammation, shedding light on the underlying mechanisms and potential therapeutic targets [1]. This comprehensive discussion aims to explore the multifaceted relationship between chronic stress and neuroinflammation, encompassing an introduction to chronic stress and its physiological effects, the role of neuroinflammation in stress-related disorders, experimental approaches using rodent models, key findings and implications for future research and therapeutic interventions. Chronic stress, characterized by prolonged activation of the Hypothalamic-Pituitary-Adrenal (HPA) axis and dysregulation of the stress response system, is a pervasive phenomenon in modern society, contributing to a myriad of adverse health outcomes, including psychiatric disorders (e.g., depression, anxiety), cardiovascular disease, immune dysfunction and neurodegeneration. The physiological response to stress involves the release of glucocorticoids (e.g., cortisol in humans, corticosterone in rodents) from the adrenal glands, which exert widespread effects on various organ systems, including the brain. Chronic exposure to stressors disrupts homeostasis within the CNS, leading to structural and functional alterations in key brain regions implicated in emotion regulation, cognition and stress modulation, such as the prefrontal cortex, hippocampus and amygdala [2].

Description

Neuroinflammation, a dynamic immune response within the CNS orchestrated by resident microglia and infiltrating immune cells, represents a fundamental component of the brain's innate immune defense system. While acute neuroinflammation plays a crucial role in the clearance of pathogens, resolution of injury and maintenance of tissue homeostasis, dysregulated or chronic neuroinflammatory processes have been implicated in the pathogenesis of various neuropsychiatric and neurodegenerative disorders, including Alzheimer's disease, Parkinson's disease and mood disorders [3]. Chronic stress serves as a potent trigger for neuroinflammatory activation, eliciting a cascade of pro-inflammatory cytokine release, microglial activation, oxidative stress and synaptic dysfunction, which contribute to neuronal damage and cognitive impairments. Experimental models using rodents, particularly mice and rats, have been instrumental in elucidating the complex interactions between chronic stress and neuroinflammation and unraveling the underlying mechanisms driving stress-induced neuropathology. These models encompass a diverse array of stress paradigms, including restraint stress, social defeat stress, chronic unpredictable stress and early-life stress, each designed to replicate different aspects of the human stress experience and recapitulate specific features of stress-related disorders. Rodent models enable researchers to interrogate the temporal dynamics, cellular and molecular pathways and behavioral correlates of stress-induced neuroinflammation, providing valuable insights into disease pathogenesis and identifying novel therapeutic targets. Key findings from rodent studies have highlighted the pivotal role of neuroinflammatory signaling pathways, including the Nuclear Factor-kappa B (NF-κB) pathway, the Mitogen-Activated Protein Kinase (MAPK) pathway and the inflammasome complex, in mediating the effects of chronic stress on CNS function and behavior. Chronic stress induces a state of "primed" microglia, characterized by enhanced reactivity and exaggerated pro-inflammatory cytokine production, which perpetuates neuroinflammatory cascades and exacerbates neuronal damage. Moreover, chronic stress-induced neuroinflammation has been implicated in synaptic remodeling, neurogenesis deficits and dysregulation of neurotransmitter systems, contributing to mood disturbances, cognitive deficits and maladaptive stress responses [4,5].

Conclusion

In conclusion, research utilizing rodent models has significantly advanced our understanding of the complex relationship between chronic stress and neuroinflammation and provided valuable insights into the pathophysiology of stress-related disorders. Leveraging these preclinical models holds promise for the identification of novel therapeutic targets and interventions aimed at mitigating the detrimental effects of chronic stress on brain health and resilience. However, further research is needed to elucidate the precise mechanisms linking chronic stress to neuroinflammation, unravel the heterogeneity of stress responses across different brain regions and cell types and translate preclinical findings into effective treatments for stress-related neuropsychiatric disorders. Collaborative efforts between basic scientists, clinicians and translational researchers are essential for bridging the gap between bench and bedside and advancing our understanding of the neurobiology of stress and resilience.

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Conflict of Interest

None.

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