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Sleep disturbances and severe stress as glial activators: key targets for treating central sensitization in chronic pain patients?

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Abstract

The mechanism of sensitization of the central nervous system partly explains the chronic pain experience in many patients, but the etiological mechanisms of this central nervous system dysfunction are poorly understood. Recently, an increasing number of studies suggest that aberrant glial activation takes part in the establishment and/or maintenance of central sensitization. Areas covered: This review focused on preclinical work and mostly on the neurobiochemistry studied in animals, with limited human studies available. Glial overactivation results in a low-grade neuroinflammatory state, characterized by high levels of BDNF, IL-1β, TNF-α, which in turn increases the excitability of the central nervous system neurons through mechanisms like long-term potentiation and increased synaptic efficiency. Aberrant glial activity in chronic pain might have been triggered by severe stress exposure, and/or sleeping disturbances, each of which are established initiating factors for chronic pain development. Expert opinion: Potential treatment avenues include several pharmacological options for diminishing glial activity, as well as conservative interventions like sleep management, stress management and exercise therapy. Pharmacological options include propentofylline, minocycline, β -adrenergic receptor antagonists, and cannabidiol. Before translating these findings from basic science to clinical settings, more human studies exploring the outlined mechanisms in chronic pain patients are needed.

Keywords: Pain; fibromyalgia; low back pain; neuroinflammation; sleep; stress.

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