with CD28 for binding to B7 molecules on antigen-presenting cells (APCs), thereby inhibiting T cell activation. Tregs can also suppress immune responses through the consumption of IL-2, a growth factor essential for T cell proliferation [3]. By expressing high levels of the IL-2 receptor chain (CD25), Tregs can deprive other T cells of

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The Role of T Regulatory Cells in Modulating Neuroimmune Responses: Findings

John D*

Department of Neuroimmunology, Harvard Medical School, Boston, USA

Abstract

neurological disorders. Strategies aimed at enhancing Treg function, such as adoptive transfer of ex vivo expanded Tregs or administration of IL-2, have shown promise in preclinical studies and some clinical trials [9]. Other approaches focus on promoting Treg induction or enhancing their suppressive activity through pharmacological interventions. For instance, low-dose IL-2 therapy has been shown to expand Tregs and improve clinical outcomes in some autoimmune diseases [10].

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e ndings summarized in this review highlight the critical role of Tregs in modulating neuroimmune responses and maintaining CNS homeostasis. Tregs exert their immunosuppressive functions through multiple mechanisms, including the production of immunosuppressive cytokines, cell-to-cell contact, and consumption of IL-2. Dysregulation of Treg function can contribute to the pathogenesis of various neurological disorders.

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Tregs play a crucial role in controlling neuroin ammation and preventing autoimmunity within the CNS. Further research is needed to fully understand the complex mechanisms regulating Treg function in the context of neuroin ammation and to develop more targeted and e ective therapeutic strategies based on modulating Treg activity for the treatment of neurological disorders.

References