

人參皂苷 Rg1 在 Cuprizone 誘導多發性硬化症模型中的雙重機制：調控

膠質細胞反應與促進再髓鞘化

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大綱

一、前言

二、人參皂苷 Rg1 透過抑制 CXCL10 介導的膠質細胞反應，在 CPZ 誘導的脫髓鞘小鼠模型中發揮保護作用

三、人參皂苷 Rg1 透過促進少突膠質前驅細胞介導的髓鞘修復，增強脫髓鞘疾病中的再髓鞘化與功能恢復

四、結論

摘要

多發性硬化症 (multiple sclerosis, MS) 是一種中樞神經系統自體免疫與神經退化性疾病，以脫髓鞘及軸突損傷為主要特徵，導致感覺、運動與認知功能受損。現有疾病改善療法雖能降低復發率，卻伴隨副作用與無法促進再髓鞘化的限制。人參皂苷 Rg1 具多重藥理活性，包括抗發炎、抗氧化與免疫調控，但其在多發性硬化症治療中的作用機制仍未完全釐清。故本篇報告旨在探討 Rg1 對於 Cuprizone (CPZ) 誘導之脫髓鞘動物的作用與機制。第一篇研究顯示，Rg1 可透過抑制 NF- κ B/CXCL10 信號路徑，降低小膠質細胞與星狀膠質細胞過度活化，下調 TNF- α 、IL-1 β 並提升 IL-10 表現，並減少脫髓鞘與軸突損傷。第二篇研究則指出，Rg1 能雙相調控 GSK3 β / β -Catenin 信號路徑：早期抑制 GSK3 β 促進 oligodendrocyte precursor cell (OPC) 增殖，晚期恢復其活性以推動分化，最終提升 myelin basic protein (MBP) 與 myelin oligodendrocyte glycoprotein (MOG) 表現並促進再髓鞘化。綜合而言，Rg1 兼具抗發炎與促進神經修復之作用，為多發性硬化症具潛力的候選治療物。

Dual mechanisms of ginsenoside rg1 in a cuprizone -induced model of multiple sclerosis: modulating glial responses and promoting remyelination

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Outline

1. Introduction
2. Rg1 exerts protective effect in CPZ-induced demyelination mouse model via inhibiting CXCL10-mediated glial response
3. Ginsenoside Rg1 promotes remyelination and functional recovery in demyelinating disease by enhancing oligodendrocyte precursor cells-mediated myelin repair
4. Conclusion

Abstract

Multiple sclerosis (MS) is an autoimmune and neurodegenerative disease of the central nervous system, characterized by demyelination and axonal damage, leading to sensory, motor, and cognitive dysfunction. Although current disease-modifying therapies can reduce relapse rates, they are associated with adverse effects and fail to promote remyelination. Ginsenoside Rg1 possesses multiple pharmacological activities, including anti-inflammatory, antioxidant, and immunomodulatory effects; however, its mechanisms in MS treatment remain incompletely understood. This report aims to investigate the effects and mechanisms of Rg1 in a cuprizone (CPZ)-induced demyelination model. The first study demonstrated that Rg1 suppressed the NF- κ B/CXCL10 signaling pathway, reduced overactivation of microglia and astrocytes, downregulated TNF- α and IL-1 β , upregulated IL-10, and attenuated demyelination and axonal injury. The second study revealed that Rg1 exerted a biphasic regulation of the GSK3 β / β -Catenin pathway: early inhibition of GSK3 β promoted oligodendrocyte precursor cell (OPC) proliferation, while later restoration of its activity facilitated differentiation, ultimately enhancing the expression of myelin basic protein (MBP) and myelin oligodendrocyte glycoprotein (MOG) and promoting remyelination. Collectively, Rg1 exhibits both anti-inflammatory and neuro-repairing properties, highlighting its potential as a therapeutic candidate for MS.

Reference

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