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## Prevention and diminished expression of experimental autoimmune encephalomyelitis by low dose naltrexone (LDN) or opioid growth factor (OGF) for an extended period: Therapeutic implications for multiple sclerosis.

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### Abstract

Endogenous opioids inhibit the onset and progression of experimental autoimmune encephalomyelitis (EAE) with 30days of treatment. This study examined the long term effects of the opioid growth factor (OGF, [Met(5)]-enkephalin) and a low dose of the opioid antagonist **naltrexone** (LDN) on expression of myelin oligodendrocyte glycoprotein (MOG)-induced EAE. C57BL/6 mice began receiving daily injections of 10mg/kg OGF (MOG+OGF), 0.1mg/kg **naltrexone** (MOG+LDN), or saline (MOG+Vehicle) at the time of EAE induction and continuing for 60days. In contrast to 100% of the MOG+Vehicle group with behavioral symptoms of EAE, 63% and 68% of the MOG+OGF and MOG+LDN mice expressed disease. Both severity and disease indices of EAE in OGF- and LDN-treated mice were notably decreased from MOG+Vehicle cohorts. By day 60, 6- and 3-fold more animals in the MOG+OGF and MOG+LDN groups, respectively, had a remission compared to MOG+Vehicle mice. Neuropathological studies revealed i) astrocyte activation and neuronal damage as early as day 10 (prior to behavioral symptoms) in all MOG-injected groups, ii) a significant reduction of activated astrocytes in MOG+OGF and MOG+LDN groups compared to MOG+Vehicle mice at day 30, and iii) no demyelination on day 60 in mice treated with OGF or LDN and not displaying disease symptoms. These results indicate that treatment with OGF or LDN had no deleterious long-term repercussions and did not exacerbate EAE, but i) halted progression of disease, ii) reversed neurological deficits, and iii) prevented the onset of neurological dysfunction across a considerable span of time.

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