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Multiple neuroprotective mechanisms of minocycline in autoimmune CNS inflammation.

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Abstract

Axonal destruction and neuronal loss occur early during multiple sclerosis, an autoimmune inflammatory CNS disease that frequently manifests with acute optic neuritis. Available therapies mainly target the inflammatory component of the disease but fail to prevent neurodegeneration. To investigate the effect of minocycline on the survival of retinal ganglion cells (RGCs), the neurons that form the axons of the optic nerve, we used a rat model of myelin oligodendrocyte glycoprotein (MOG)-induced experimental autoimmune encephalomyelitis. Optic neuritis in this model was diagnosed by recording visual evoked potentials and RGC function was monitored by measuring electroretinograms. Functional and histopathological data of RGCs and optic nerves revealed neuronal and axonal protection when minocycline treatment was started on the day of immunization. Furthermore, we demonstrate that minocycline-induced neuroprotection is related to a direct antagonism of multiple mechanisms leading to neuronal cell death such as the induction of anti-apoptotic intracellular signalling pathways and a decrease in glutamate excitotoxicity. From these observations, we conclude that minocycline exerts neuroprotective effects independent of its anti-inflammatory properties. This hypothesis was confirmed in a non-inflammatory disease model leading to degeneration of RGCs, the surgical transection of the optic nerve.

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