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Minocycline injection in the ventral posterolateral thalamus reverses microglial reactivity and thermal hyperalgesia secondary to sciatic neuropathy.

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Abstract

We hypothesized that microglia in the ventral posterolateral (VPL) nucleus of the thalamus are reactive following peripheral nerve injury, and that inhibition of microglia by minocycline injection in the VPL attenuates thermal hyperalgesia. Our results show increased expression of OX-42 co-localized with phosphorylated p38MAPK (P-p38) in the VPL seven days after chronic constriction injury (CCI) of the sciatic nerve. However, astrocytic GFAP expression in the VPL is unchanged 7 and 14 days after CCI. Microinjection of minocycline into the VPL contralateral to CCI reverses thermal hyperalgesia, whereas vehicle injection has no effect on paw withdrawal latency. Minocycline abrogates the increased expression of OX-42 in the VPL after CCI. Therefore, peripheral nerve injury favors a hyperactive microglial phenotype in the VPL, suggesting remote neuroimmune signaling from the damaged nerve to the brain, concomitant with neuropathic behavior that is reversed by local intervention in the VPL to inhibit microglia.

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