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## Targeting descending dopaminergic signalling to treat trigeminal neuropathic pain

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rigeminal neuropathic pain is a debilitating condition and represents a challenge to clinicians. In the present study, we employed optogenetic manipulation to investigate the role of the dopaminergic pathway from hypothalamic A11 nucleus to spinal trigeminal nucleus caudalis (Sp5C) in orofacial neuropathic pain. Dopamine receptor D1-Cre and D2-Cre male mice were used in this study. A chronic constriction injury-infraorbital nerve (CCI-ION) mouse model was prepared as described previously. Optogenetic manipulation was conducted to examine the effect of activation or inhibition of D1/2-mediated dopaminergic signaling on trigeminal neuropathic pain in the CCI-ION model. Conditional place preference (CPP) and von Frey filaments were used to measure the emotional and sensory components of the CCI-IONinduced pain behaviors. Immunohistochemistry staining was used to assess the expression of D1/2 dopamine receptor and A11 lesion. Pain behavioral testing showed that D1-Cre mice injected with the inhibitory virus AAV5-EF1a-DIO-eNpHR3.0-EYFP exhibited a decrease in pain behaviors when stimulated with green light (532 nm), but D1-Cre mice injected with the excitatory virus AAV5-EF1a-DIO-ChR2 (E123A)-EYFP exhibited an increase in pain behaviors when stimulated with blue light (473 nm). Interestingly, we observed an opposite effects in D2-Cre mice when the same optogenetic manipulation was carried out. Moreover, 6-hydroxy-dopamine hydrobromide (6-OHDA)-produced specific lesion of A11 dopaminergic neurons blocked the effect of optogenetic manipulation of the Sp5C D1/2 dopamine receptor on trigeminal neuropathic pain in this CCI-ION model. In addition, D1 and D2 dopamine receptors were highly expressed in the Sp5C. Therefore, the descending dopaminergic pathway from A11 to Sp5C could be a critical target for the treatment of trigeminal neuropathic pain.

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