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Antiallodynic effect of β-caryophyllene on paclitaxel-induced peripheral neuropathy in mice.

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Abstract

Painful peripheral neuropathy is a common side effect of paclitaxel (PTX). The use of analgesics is an important component for management of PTX-induced peripheral neuropathy (PINP). However, currently employed analgesics have several side effects and are poorly effective. β-caryophyllene (BCP), a dietary selective CB_2 agonist, has shown analgesic effect in neuropathic pain models, but its role in chemotherapy-induced neuropathic pain has not yet been investigated. Herein, we used the mouse model of PINP to show the therapeutic effects of BCP in this neuropathy. Male Swiss mice receiving PTX (2 mg kg^{-1}, ip, four alternate days) were treated with BCP (25 mg kg^{-1}, po, twice a day) either during or after PTX administration. Some groups were also pretreated with AM630 (CB_2 antagonist, 3 mg kg^{-1}, ip) or AM251 (CB_1 antagonist, 1 mg kg^{-1}, ip). Spinal cord samples were collected in different time points to perform immunohistochemical analysis. BCP attenuated the established mechanical allodynia induced by PTX ($p < 0.0001$) in a CB_2-dependent manner. Of note, when given concomitantly with PTX, BCP was able to attenuate the development of PINP ($p < 0.0001$). Spinal cord immunohistochemistry revealed that preventive treatment with BCP reduced p38 MAPK and NF-κB activation, as well as the increased Iba-1 and IL-1β immunoreactivity promoted by PTX. Our findings show that BCP effectively attenuated PINP, possibly through CB_2-activation in the CNS and posterior inhibition of p38 MAPK/NF-κB activation and cytokine release. Taken together, our results suggest that BCP could be used to attenuate the establishment and/or treat PINP.

KEYWORDS: Cannabinoid; Neuropathic pain; Paclitaxel; β-caryophyllene

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