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Neuropharmacology. 2017 Oct;125:220-230. doi: 10.1016/j.neuropharm.2017.07.024. Epub 2017 Jul 25.

Cannabidiol disrupts the consolidation of specific and generalized fear memories via dorsal hippocampus CB₁ and CB₂ receptors.

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Abstract

Pharmacological interventions able to modulate a fear memory while it is consolidated could have therapeutic value in tempering those maladaptively overconsolidated. Animal and human studies have shown the intensity of unconditioned stimulus delivered during fear conditioning influences qualitative and quantitative aspects of the memory to be established. By varying the shock intensity used for contextual pairing in rats, here we induced specific and more generalized long-term fear memories to investigate whether, how and where in the brain the cannabidiol (CBD; 3.0-30 mg/kg i.p.) could impair their consolidation and related outcomes. When given immediately after their acquisition, it reduced respectively the conditioned fear expression, and fear generalization, ultrasonic vocalizations at 22-kHz and the relative resistance to extinction. CBD had no effects on short-term fear memory, and its delayed treatment no longer affected the consolidation process. As the dorsal hippocampus (DH) modulates fear memory specificity and generalization, and cannabinoid type-1 (CB₁) and type-2 (CB₂) receptors contribute to consolidation, we investigated their involvement in CBD effects. Both systemic and intra-DH treatment with the CB₁ receptor antagonist/inverse agonist AM251 or the CB₂ receptor antagonist/inverse agonist AM630 prevented the disrupting CBD effects on consolidation. Since the CBD effects on the endocannabinoid transmission are probably indirect, we investigated and demonstrated the FAAH inhibitor URB597 induced effects similar to those of CBD when given systemically or intra-DH. Altogether, the present results suggest the CBD disrupts the consolidation of different fear memories via anandamide-mediated activation of DH CB₁ and CB₂ receptors.

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KEYWORDS: Cannabidiol; Consolidation; Extinction; Generalization; Hippocampus

PMID: 28754373 DOI: 10.1016/j.neuropharm.2017.07.024

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