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Cannabinoids and traumatic stress modulation of contextual fear extinction and GR expression in the amygdala-hippocampal-prefrontal circuit.

Ganon-Elazar E[1], Akirav I.

Author information

Abstract

Considerable evidence suggests that cannabinoids modulate the behavioral and physiological response to stressful events. We have recently shown that activating the cannabinoid system using the CB1/CB2 receptor agonist WIN55,212-2 (WIN) in proximity to exposure to single-prolonged stress (SPS), a rat model of emotional trauma, prevented the stress-induced enhancement of acoustic startle response, the impairment in avoidance extinction and the enhanced negative feedback on the hypothalamic-pituitary-adrenal (HPA) axis (Ganon-Elazar and Akirav, 2012). Some of the effects were found to be mediated by CB1 receptors in the basolateral amygdala (BLA). Here we examined whether cannabinoid receptor activation in a putative brain circuit that includes the BLA, hippocampus and prefrontal cortex (PFC), could prevent the effects of traumatic stress on contextual fear extinction and alterations in glucocorticoid receptor (GR) protein levels. We found that: (i) SPS impaired contextual fear extinction tested one week after trauma exposure and that WIN prevented the stress-induced impairment of extinction when microinjected immediately after trauma exposure into the BLA or hippocampus (5 μg), but not when microinjected into the PFC, (ii) the ameliorating effects of WIN on contextual extinction were prevented by blocking GRs in the BLA and hippocampus, and (iii) SPS up regulated GRs in the BLA, PFC and hippocampus and systemic WIN administration (0.5 mg/kg) after trauma exposure normalized GR levels in the BLA and hippocampus, but not in the PFC. Cannabinoid receptor activation in the aftermath of trauma exposure may regulate the emotional response to the trauma and prevent stress-induced impairment of extinction and GR up regulation through the mediation of CB1 receptors in the BLA and hippocampus. Taken together, the findings suggest that the interaction between the cannabinoid and glucocorticoid systems is crucial in the modulation of emotional trauma.

KEYWORDS: CB1 receptors; Cannabinoids; Contextual fear extinction; Glucocorticoid receptors; Post traumatic stress disorder; Single prolonged stress

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