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Cannabinoids prevent the effects of a footshock followed by situational reminders on emotional processing.

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Abstract

Posttraumatic stress disorder (PTSD) can develop following exposure to a traumatic event. Hence, what we do in the first few hours after trauma exposure may alter the trajectory of PTSD. We examined whether cannabinoids can prevent the effects of a single footshock followed by situational reminders (SRs) on emotional processing. Rats were exposed to a footshock (1.5 mA, 10 s) on day 1 followed by exposure to SRs of the shock on days 3 and 5. The CB1/2 receptor agonist WIN55,212-2 or vehicle were injected intraperitoneally 2 h after the shock. After 1 week, PTSD-like symptoms were examined. Exposure to SRs exacerbated the effects of the shock as rats exposed to shock and SRs, but not shock alone, showed impaired extinction of the traumatic event, impaired plasticity in the hippocmapal-accumbens pathway, enhanced latency to startle, and altered expression of CB1 receptors (CB1r) and glucocorticoid receptors (GRs) in the CA1, basolateral amygdala (BLA) and prefrontal cortex (PFC). WIN55,212-2 prevented the effects of the shock and SRs on extinction, plasticity, and startle response. WIN55,212-2 normalized the shock/SR-induced upregulation in CB1r in the PFC, and CA1 and GRs in the CA1, with no effect on BLA downregulation of CB1r and GRs. Shock and SRs caused lasting (1 week) alterations in emotional processing associated with changes in GR and CB1r expression in brain areas related to PTSD. WIN55,212-2 administered after trauma exposure prevented these alterations via PFC- and CA1-CB1r and CA1-GRs.

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