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An ultra-low dose of tetrahydrocannabinol provides cardioprotection.

Waldman M¹, Hochhauser E, Fishbein M, Aravot D, Shainberg A, Sarne Y.

Author information

Abstract

Tetrahydrocannabinol (THC), the major psychoactive component of marijuana, is a cannabinoid agonist that exerts its effects by activating at least two specific receptors (CB1 and CB2) that belong to the seven transmembrane G-protein coupled receptor (GPCR) family. Both CB1 and CB2 mRNA and proteins are present in the heart. THC treatment was beneficial against hypoxia in neonatal cardiomyocytes in vitro. We also observed a neuroprotective effect of an ultra low dose of THC when applied to mice before brain insults. The present study was aimed to test and characterize the cardioprotective effects of a very low dose (0.002mg/kg) of THC which is 3-4 orders of magnitude lower than the conventional doses, administered before myocardial infarction in mice in vivo. Three regimens of THC administration were tested: single THC application 2h or 48h before the induction of infarct, or 3 weeks continuous treatment before MI. All protocols of THC administration were found to be beneficial. In the case of THC treatment 2h before MI, fractional shortening was elevated (37±4% vs. 42±1%, p<0.04), troponin T leakage to the blood was reduced (14±3ng/ml vs. 10±4ng/ml, p<0.008), infarct size decreased (29±4% vs. 23±4%, p<0.02), and the accumulation of neutrophils to the infarct area declined (36±10cells/field vs. 19±4cells/field, p<0.007) in THC-compared to vehicle-pretreated mice, 24h after MI. ERK1/2 phosphorylation following infarct was also inhibited by pre-treatment with THC (p<0.01).

CONCLUSION: A single ultra low dose of THC before ischemia is a safe and effective treatment that reduces myocardial ischemic damage.

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