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The role of the endocannabinoid system in a mouse model of ddC-Induced neuropathic pain

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Statement of the Problem: Nucleoside reverse transcriptase inhibitors (NRTIs) are the cornerstone in the treatment of HIV/AIDS. Sometimes, their use is limited by the development of a painful neuropathy, which does not respond well to drugs. However, some HIV patients with painful neuropathy report relief after using cannabis. The aim of this study is to evaluate whether the endocannabinoid system plays a role in NRTI-induced painful neuropathy.

Methodology & Theoretical Orientation: Female BALB/c mice were treated with 25 mg/kg of 2 , 3 -dideoxycytidine (ddC). The expression of the endocannabinoid system molecules was evaluated by real-time RT-PCR in the brain, spinal cord and paw skin at days two, six and nine post ddC administration. The effects of the endocannabinoids, N-arachidonoyl ethanolamine (AEA), 2-arachidonoyl glycerol (2-AG), cannabinoid receptor antagonists, AM 251 and AM 630 on ddC-induced thermal hyperalgesia were evaluated using the hot plate test.

Findings: Mice treated with ddC developed mechanical and cold allodynia, thermal hyperalgesia and chemical hyposensitivity. Mice are sacrificed at a time point when they had developed allodynia, hyperalgesia or hyposensitivity which had increased transcripts of phospholipase C-1beta and acylglycerol kinase in the paw skins and spinal cords but not in the brain. On the other hand, transcripts of fatty acid amide hydrolase and monoacyl glycerol were down regulated in the paw skins and brains but not in the spinal cord. AEA and 2-AG had antihyperalgesic effects against ddC-induced thermal hyperalgesia, but had no effect in naïve mice. The antihyperalgesic activity of AEA was antagonized by AM251 and AM630, whereas the activity of 2-AG was antagonized by AM251 but not by AM630.

Conclusion & Significance: Our results show that ddC induces painful neuropathy, which is associated with dysregulation of the endocannabinoid system. Agonists of cannabinoid receptors could be useful therapeutic agents for the management of NRTI-induced painful sensory neuropathy.

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