

Persistent Inflammation and Cannabis Use Modulate the Endocannabinoid System

September 23

Tuesday, 12:30 pm

Billings Building—Rosedale Room

SPEAKER:



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Host: Dianna E. Willis, Ph.D.

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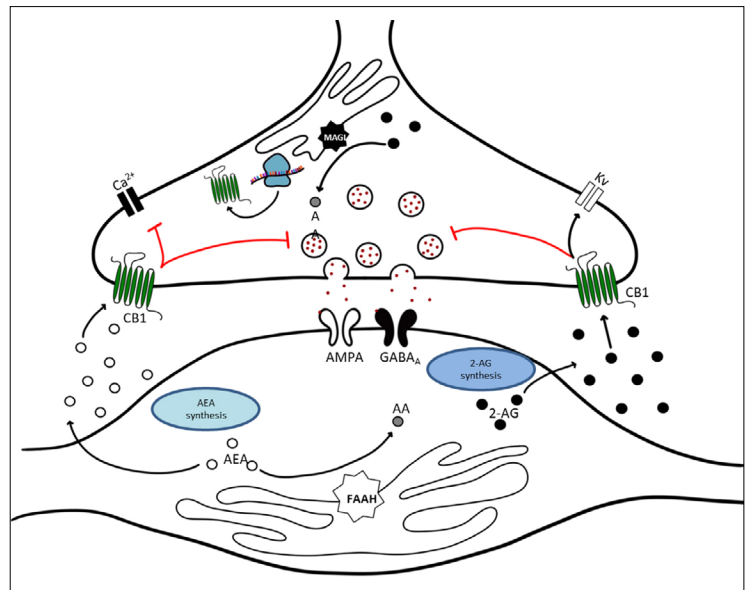
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Abstract

The Ingram Lab research aims to elucidate properties of ion channels, neurotransmitter transporters, G protein-coupled receptors (GPCRs), and cellular mechanisms of synaptic plasticity associated with acute and chronic pain and drug addiction. The lab focuses predominantly on adaptations within the ventrolateral periaqueductal gray (vlPAG), a key integration site for the descending pain modulatory circuit. The

fundamental observation driving the Ingram Lab research is that GPCRs are regulated via different mechanisms depending on their pre-vs. postsynaptic locations. Given that presynaptic GPCRs

govern the amount of neurotransmitter release throughout the nervous system, understanding how these receptors are regulated and how these mechanisms may be targeted by therapeutics is an important strategy for the development of novel drugs. Ongoing studies in the lab investigate (1) adaptations to GPCR signaling mechanisms and regulation, particularly opioid and cannabinoid receptors, (2) how those adaptations influence signal integration within the vlPAG and resulting output to the downstream rostroventromedial medulla, and (3) circuit specificity amongst the heterogeneous vlPAG subpopulations with respect to pain, addiction, and aversion behaviors.



Publications

1. Coutens B, Bouchet CA, Patti LC, McPherson KB, Boston BS, Jewett DC, Ingram SL. Corticosterone stimulates synthesis of 2-arachidonoylglycerol via putative membrane-bound glucocorticoid receptors and inhibits GABA release via CB1 cannabinoid receptors in the ventrolateral periaqueductal gray. *Mol Pharmacol*. 2025 Jun 21;107(8):100058. doi: 10.1016/j.molpha.2025.100058. Epub ahead of print. PMID: 40729946.
2. Bouchet CA, McPherson KB, Coutens B, Janowsky A, Ingram SL. Monoacylglycerol Lipase Protects the Presynaptic Cannabinoid 1 Receptor from Desensitization after Persistent Inflammation. *J Neurosci*. 2023 Jul 26;43(30):5458-5467. doi: 10.1523/JNEUROSCI.0037-23.2023. Epub 2023 Jul 6. PMID: 37414560; PMCID: PMC10376933.
3. Rios SM, Mootz JRK, Phillips TJ, Ingram SL. Absence of TAAR1 function increases methamphetamine-induced excitability of dorsal raphe serotonin neurons and drives binge-level methamphetamine intake. *Neuropsychopharmacology*. 2025 Jun;50(7):1136-1144. doi: 10.1038/s41386-025-02063-w. Epub 2025 Feb 11. PMID: 39934409; PMCID: PMC12089393.