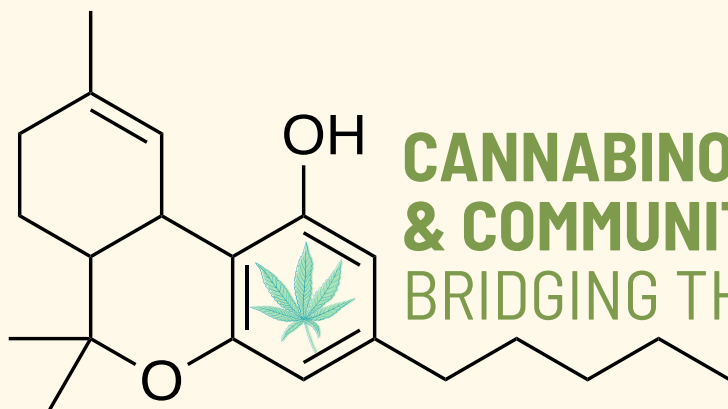


Welcome to the

• **INAUGURAL UC-WIDE** •



**CANNABINOID SCIENCE
& COMMUNITY HEALTH:
BRIDGING THE GAP**

Symposium

May 3rd, 2024

Welcome to the Inaugural UC-wide Symposium UCR Center for Cannabinoid Research

We are delighted to welcome you to the inaugural University of California-wide symposium, “Cannabinoid Science and Community Health: Bridging the Gap”. This event includes a series of talks from leaders in cannabinoid research in the University of California system, poster sessions for displaying studies conducted by our trainees and researchers, as well as a panel to discuss opportunities for our collective cannabinoid research to improve community health.

It's an exciting time for cannabinoid research. Cannabis is rapidly becoming legalized across the United States for medicinal and recreational purposes. There are large gaps, however, in our understanding of how cannabis use impacts our health and the biological mechanisms involved. This symposium will provide a platform to showcase the latest cannabinoid science and to lay the foundation for future collaborations among California's cannabinoid scientists and institutions.

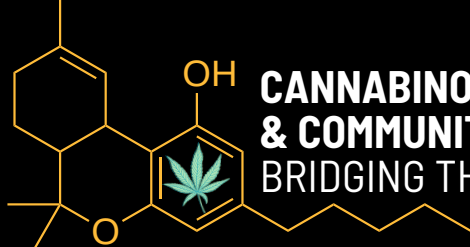
“Cannabinoid Science and Community Health: Bridging the Gap” is sponsored by the University of California-Riverside Center for Cannabinoid Research (UCRCCR). UCRCCR was created to serve as a community of diverse scientists and clinicians with common goals aimed at advancing our understanding of roles for the endocannabinoid system in health and disease, and the impact that cannabis use has on these processes. We aim to promote cannabinoid research in an unbiased manner unaffected by dogmas and stigmas that have plagued cannabinoid research for decades.

We thank Pica Preston and the entire team of staff and student volunteers for their invaluable and tireless support in organizing this event.

Hope you have a wonderful day celebrating cannabinoid science!

Nick DiPatrizio, Ph.D., Founding Director of the UCR Center for Cannabinoid Research





Inaugural UC-wide Symposium UCR Center for Cannabinoid Research

- MAY 3RD, 2024 | 12:30 PM - 6:00 PM -

UC Riverside, School of Medicine Education II Building, Room 205

Program:

12:30pm **Registration**

12:45-12:50pm **Welcome and Introduction**

*Nicholas DiPatrizio, Ph.D., Director, UCR Center for Cannabinoid Research,
Professor of Biomedical Sciences | University of California, Riverside*

AFTERNOON SESSION I, MODERATOR: NATALIE ZLEBNIK, PHD.

Keynote Speaker

1:00-1:45pm **“Systemic long-term effects of adolescent THC exposure.”**

*Daniele Piomelli, Ph.D., Distinguished Professor of Anatomy & Neurobiology School of Medicine,
Director of the Center for the Study of Cannabis | University of California, Irvine*

1:50-2:15pm **“UCLA Center for Cannabis and Cannabinoids: Opportunities for collaborations across disciplines and institutions.”**

Ziva Cooper, Ph.D., Director of the Center for Cannabis and Cannabinoids in the Jane & Terry Semel Institute for Neuroscience and Human Behavior, Professor of Psychiatry and Biobehavioral Sciences and Anesthesiology | University of California, Los Angeles

2:20-2:45pm **“Driving Under the Influence of Cannabis: Evaluation of the Field Sobriety Tests.”**

*Thomas Marcotte, Ph.D., Co-Director of the Center for Medicinal Cannabis Research,
Professor of Psychiatry | University of California, San Diego*

2:50-3:15pm **Afternoon Break 1 and Poster Session**

3:20-3:45pm **“Cannabis in Cancer Care.”**

Donald Abrams, M.D., Professor Emeritus of Medicine | University of California, San Francisco

3:50-4:15pm **“Advancing Cannabis Safety: Research Agenda & Strategies for Policy Implementation.”**

Arturo Durazo Ph.D., Acting Director, Nicotine & Cannabis Policy Center, Community-Engaged Health Scientist, Health Sciences Research Institute | University of California, Merced

AFTERNOON MODERATOR II, CHRISTOPHER FICHTNER, MD.

4:20-4:45pm **Afternoon Break II and Poster Session**

4:50-5:25pm **“A Tour of Cannabinoid Research at UCR.”**

*Nicholas DiPatrizio, Ph.D., Director, UCR Center for Cannabinoid Research,
Professor of Biomedical Sciences | University of California, Riverside*

5:30-6:00pm **Discussion Panel**

6:00pm **Closing remarks and expression of gratitude**

*Nicholas DiPatrizio, Ph.D., Director, UCR Center for Cannabinoid Research,
Professor of Biomedical Sciences | University of California, Riverside*

Keynote Presentation:

“Systemic long-term effects of adolescent THC exposure.”



Daniele Piomelli, Ph.D.

Director, Center for the Study of Cannabis
Distinguished Professor of Anatomy & Neurobiology, School of Medicine
University of California, Irvine

Piomelli's Lab: <https://www.law.uci.edu/centers/csc/>

Daniele Piomelli studied pharmacology and neuroscience with James H. Schwartz and Eric Kandel at Columbia University (1983-1988), and with Paul Greengard at the Rockefeller University (1988-1990). In 2000, two of his mentors (Kandel and Greengard) were awarded the Nobel Prize for their contributions to physiology and medicine. After working at the INSERM in Paris (France) and at the Neurosciences Institute in San Diego, with Nobel Laureate Gerald Edelman, Daniele joined the University of California, Irvine, where he is now Louise Turner Arnold Chair in Neurosciences and Distinguished Professor of Anatomy and Neurobiology, Pharmacology and Biological Chemistry. Daniele is an author of >400 peer-reviewed articles in journals such as Nature, Science, Nature Medicine, PNAS and Nature Neuroscience, three full-length books, and 34 patents. He founded the department of drug discovery and development (D3) at the Italian Institute of Technology in Genoa (Italy), which he directed from 2007 to 2016, and three biopharmaceutical start-ups based on discoveries made in his lab. He is director of the UCI's Center for the Study of Cannabis and Editor-in-Chief of Cannabis and Cannabinoid Research, the first peer-reviewed journal entirely dedicated to the study of cannabis, its derivatives, and their endogenous counterparts in the human body.

Presentation:

“UCLA Center for Cannabis and Cannabinoids: Opportunities for collaborations across disciplines and institutions.”



Ziva Cooper, Ph.D.

Director, UCLA Center for Cannabis and Cannabinoids in the Jane and Terry Semel Institute for Neuroscience and Human Behavior
Professor in the UCLA Departments of Psychiatry and Biobehavioral Sciences and Anesthesiology

Cooper's Lab: <https://cannabis.semel.ucla.edu/ziva-cooper/>

Ziva Cooper, Ph.D., is the Director of the UCLA Center for Cannabis and Cannabinoids in the Jane and Terry Semel Institute for Neuroscience and Human Behavior and Professor in the UCLA Departments of Psychiatry and Biobehavioral Sciences and Anesthesiology. Her research involves understanding variables that influence both the therapeutic potential and adverse effects of cannabis and cannabinoids, the chemicals in the cannabis plant. Dr. Cooper received her PhD from the University of Michigan in Biopsychology in 2007 in the field of preclinical psychopharmacology, experience that informs her focus on translating preclinical studies of cannabinoids to the clinic using placebo-controlled human drug-administration studies. Ziva served on the National Academies of Sciences, Engineering, and Medicine Committee on the Health Effects of Cannabis that published a comprehensive report of the health effects of cannabis and cannabinoids in 2017. She is also serving on the current National Academies of Sciences, Engineering, and Medicine Committee on the public health consequences of changes in the cannabis policy landscape. Her current projects funded by the NIH and California State include understanding the potential for cannabis constituents to reduce reliance on opioids, differences between men and women in their response to the pain-relieving effects of cannabis, effects of cannabis as a function of age, and therapeutic effects of cannabinoids in patient populations. She is the President of the International Cannabinoid Research Society, a past Board Director for the College on Problems of Drug Dependence, an Associate Editor of Neuropsychopharmacology and is on several Editorial Boards of journals including Cannabis and Cannabinoid Research.

Presentation:

“Driving Under the Influence of Cannabis: Evaluation of the Field Sobriety Tests.”



Thomas Marcotte, Ph.D.

Professor of Psychiatry

Co-Director of the Center for Medicinal Cannabis Research

University of California, San Diego

Marcotte's info: <https://profiles.ucsd.edu/thomas.marcotte>

Dr. Marcotte is Professor of Psychiatry at the University of California, San Diego, and Co-Director of the Center for Medicinal Cannabis Research, which has conducted clinical trials of cannabis for almost 20 years, and has an active, ongoing portfolio exploring the effects of cannabinoids (plant-based, synthetic) in various medical/psychiatric conditions. He is the principal investigator on studies addressing the effects that cannabis (including commercially-available products) has on driving performance, as well as methods for detecting cannabis-related driving impairment. He previously was the principal investigator on an NIH-funded take-home study of cannabis for the treatment of pain, and a co-investigator on cannabis studies addressing pain, as well spasticity in multiple sclerosis. He has been the PI on numerous HIV studies over the years, including projects aimed at examining antiretroviral treatment initiation in India, the development of brief screening tools for HIV-associated neurocognitive disorders (HAND), and studies examining the impact of HAND on real-world functioning. Dr. Marcotte has developed techniques for assessing the most complex of everyday tasks, driving an automobile, and examined the types of deficits that may predispose an individual to impaired driving abilities. Dr. Marcotte previously served as the Center Manager of the HIV Neurobehavioral Research Center (1996-2018), and Co-Director of CHARTER (a national multi-site study of the CNS impact of treatments for HIV). He co-edited the book *Neuropsychology of Everyday Functioning* (currently in its 2nd edition) and has been on the editorial boards of *Neuropsychology* and the *Journal of the International Neuropsychological Society*.

Presentation:
“Cannabis in Cancer Care.”



Donald Abrams, M.D.

Professor, Emeritus of Medicine
University of California, San Francisco

Abram's info: <https://profiles.ucsf.edu/donald.abrams>

Donald I. Abrams, M.D., is a Professor Emeritus of Medicine at the University of California San Francisco. He has an Integrative Oncology consultation practice at the UCSF Osher Center for Integrative Medicine. He received an A.B. in Molecular Biology from Brown University in 1972 and graduated from the Stanford University School of Medicine in 1977. After completing an Internal Medicine residency at the Kaiser Foundation Hospital in San Francisco, he became a fellow in Hematology/Oncology at the Cancer Research Institute of the University of California, San Francisco in 1980. He was one of the original clinician/investigators to recognize and define many early AIDS-related conditions at San Francisco General Hospital where he also served as chief of Hematology-Oncology for 14 years. He has long been interested in clinical trials of complementary and alternative medicine interventions for HIV/AIDS and cancer, including evaluations of medicinal cannabis. In 1997 he received funding from the National Institute on Drug Abuse to conduct a clinical trial on the short-term safety of cannabinoids in HIV infection. Subsequently he was granted funds by the University of California Center for Medicinal Cannabis Research to continue studies of the effectiveness of cannabis in a number of clinical conditions. He completed a placebo-controlled study of smoked cannabis in patients with painful HIV-related peripheral neuropathy as well as a study evaluating vaporization as a smokeless delivery system for medicinal cannabis. He conducted a NIDA-funded trial investigating the pharmacokinetic interaction between vaporized cannabis and opioid analgesics in patients with chronic pain. His last study was an NIH-funded trial evaluating vaporized cannabis in patients with sickle cell disease. He co-authored the chapter on “Cannabinoids and Cancer” in the Oxford University Press Integrative Oncology text that he co-edited with Andrew Weil. He co-edits the NCI PDQ CAM Cannabinoids and Cancer website. He was a member of the National Academies of Sciences, Engineering and Medicine’s committee that published *The Health Effects of Cannabis and Cannabinoids: Current State of Evidence and Recommendations for Research* in January 2017.

Presentation:

“Advancing Cannabis Safety: Research Agenda & Strategies for Policy Implementation.”



Arturo Durazo, Ph.D.

Acting Director, Nicotine & Cannabis Policy Center
Community-Engaged Health Scientist, Health Sciences Research Institute
University of California, Merced

Durazo's info: <https://hsri.ucmerced.edu/content/arturo-durazo-phd>

Arturo Durazo, Ph.D., is the Acting Director of the Nicotine and Cannabis Policy Center and a Community-Engaged Health Scientist at the University of California, Merced. He directs mentorship programs fostering undergraduate and graduate research in tobacco control with CSU Fresno and Stanislaus.

Dr. Durazo advances cancer prevention, tobacco/cannabis control, and health equity-related health science. His collaborative research aims to mitigate diseases that impact marginalized and under-resourced populations with community-academic partnerships. With two decades of health research and advocacy, he translates patient-reported outcomes into effective multi-lingual health interventions, especially within Latinx communities. As Chair of the Central Valley's Out with Big Tobacco Coalition, Dr. Durazo's leadership extends to shaping health policies in the San Joaquin Valley and the Sierra Nevada foothills.

Presentation:
“A Tour of Cannabinoid Research at UCR.”



Nicholas DiPatrizio, Ph.D.

Director, UCR Center for Cannabinoid Research
Professor of Biomedical Sciences
University of California, Riverside

DiPatrizio's info: <https://profiles.ucr.edu/app/home/profile/ndipatri>

Nicholas DiPatrizio, Ph.D., is associate professor of Biomedical Sciences in the UCR School of Medicine and founding director of the UCR Center for Cannabinoid Research (UCRCCR). <https://cannabinoid.ucr.edu>

The DiPatrizio laboratory is dedicated to elucidating the integrative neurobiology and physiology that controls food reward, sensory processing, and energy homeostasis, and dysregulation of these pathways in metabolic disorders. A combination of state-of-the-art analytical chemistry (i.e., ultra-performance liquid chromatography/tandem mass spectrometry), genetic (i.e., first-of kind genetic mutant mice with conditional organ-selective knockout of endocannabinoid system components), surgical, biochemical, molecular, pharmacological, and behavioral tools are employed to achieve these goals.

One area of focus in the DiPatrizio Lab is on identifying roles for the endocannabinoid system in food intake and reward, and dysregulation of these pathways in metabolic disease. Moreover, the impact of cannabis exposure on these systems in health and disease is investigated. Collectively, this knowledge will support the discovery and development of novel therapeutic strategies to safely treat metabolic and related disorders.

Poster Abstracts

1. Effects of cannabinoids on impulsive choice in rats

Andrew Villa, Leslie Estrada, Saigayathri Bhaskar, Kathlyn Mai, Chlinton Kuang, Yesenia Alvarenga, Natalie Zlebnik, Samantha Miller, Joseph Cheer, Jennifer Wenzel

University of California, Riverside

University of Maryland

University of California, San Diego

Impulsive choice is characterized by preference for smaller immediate rewards over larger delayed rewards, and impulsivity is a determining factor in the development and persistence of substance use disorders (SUDs). Endocannabinoid signaling is a powerful modulator of ventral striatal reward-processing circuitry that is critical to decision-making in impulsive choice. However, the role of both endogenous and exogenous cannabinoids in impulsive decision-making is yet to be examined. We employed targeted intra-NAc pharmacology to interrogate the endocannabinoid system during performance of a delay-discounting task and followed up with investigations of the effects of long-term exposure to delta-9-tetrahydrocannabinol (THC), a cannabinoid-1 (CB1) receptor agonist and the primary psychoactive constituent of cannabis, on impulsive decision-making. Results demonstrate that blocking accumbal CB1 receptor signaling potentiated impulsive choice, whereas enhancing endocannabinoid signaling attenuated impulsive choice. Long-term exposure to THC recapitulated the behavioral effects of promoting CB1 receptor signaling by decreasing impulsive decision-making. Together, these findings highlight new ways in which cannabinoid signaling contributes to impulsive decision-making. Results such as these may have important implications for the pathophysiology and treatment of SUDs and impulse control disorders such as attention deficit/hyperactivity disorder.

2. Enhanced endocannabinoid tone in the striatum alters the dopaminergic substrates of cognitive flexibility

Brandon Oliver, Heathe Bago, Alexandra Arcenas, Harrison Lin, Natalie Zlebnik

University of California, Riverside

Dopaminergic (DA) signaling in the striatum is essential for reward-based learning, with mesolimbic pathways to the ventral striatum critical for early learning and nigrostriatal pathways to the dorsal striatum relevant for habitual behaviors. The role of phasic DA release in updating associations during reversal learning remains unclear. Additionally, the endocannabinoid system, particularly through cannabinoids like 2-AG, modulates midbrain DA neurons influencing striatal DA release ultimately impacting reward learning. Striatal CB1R modulation alters habitual action control necessary for learning-induced plasticity. Further, extracellular DA in the NAc is decreased following CB1 antagonism and increased following CB1 agonism. Therefore, endocannabinoid signaling may play a role in reward processing and performance on a reward-motivated task via regulation of striatal DA release. The current experiment used fiber photometry to measure transient DA signals in the NAc (N = 54) and DLS (N = 16) via the genetically-encoded dopamine sensor, GrabDA, during an operant probabilistic reversal learning (PRL) task. Mice were trained to discriminate between two levers of differing reinforcement probabilities (80% vs. 20%); followed by a reversal phase where the reinforcement probabilities were inverted across levers. During the reversal session, mice were pre-treated with a monoacylglycerol lipase (MAGL) inhibitor, JZL-184 to increase synaptic 2-AG. Results demonstrate that systemic manipulations of endocannabinoid signaling impair reversal learning performance and dysregulate associated NAc and DLS DA release. These findings give critical insight into the role of the endocannabinoid system in flexible reward-based learning and may have significant implications for the use of cannabinoids for recreational or therapeutic purposes.

3. Cholinergic neurotransmission controls orexigenic endocannabinoid signaling in the gut in diet-induced obesity

Courtney P. Wood, Camila Alvarez, and Nicholas V. DiPatrizio

University of California, Riverside
University of California, San Diego

The brain bidirectionally communicates with the gut to control food intake and energy balance, which becomes dysregulated in obesity. For example, endocannabinoid (eCB) signaling in the small-intestinal epithelium (SI) is upregulated in diet-induced obese mice (DIO) and promotes overeating by a mechanism that includes inhibiting gut-brain satiation signaling. Upstream neural and molecular mechanism(s) involved in overproduction of orexigenic gut eCBs in DIO, however, are unknown. We tested the hypothesis that overactive parasympathetic signaling at muscarinic acetylcholine receptors (mAChRs) in the SI increases biosynthesis of the eCB, 2-arachidonoyl-sn-glycerol (2-AG), which drives hyperphagia via local CB1Rs in DIO. Male mice were maintained on a high-fat/high-sucrose western-style diet for 60 days, then administered several mAChR antagonists 30 min prior tissue harvest or a food intake test. Levels of 2-AG and activity of its metabolic enzymes in the SI were quantitated. DIO mice, when compared to those fed a low-fat/no-sucrose diet, displayed increased expression of cFos protein in the dorsal motor nucleus of the vagus, which suggests increased activity of efferent cholinergic neurotransmission. These mice exhibited elevated levels of 2-AG biosynthesis in the SI, which was reduced to control levels by mAChR antagonists. Moreover, the peripherally-restricted mAChR antagonist, methylhomatropine bromide, and the peripherally-restricted CB1R antagonist, AM6545, reduced food intake in DIO mice for up to 24 h but had no effect in mice conditionally deficient in SI CB1Rs. These results suggest that hyperactivity at mAChRs in the periphery increases formation of 2-AG in the SI and activates local CB1Rs, which drives hyperphagia in DIO.

4. Role of the Gut-Brain Endocannabinoid System in Food Reward

Camila Alvarez, Brandon L. Oliver, Natalie E. Zlebnik, Nicholas V. DiPatrizio

University of California, Riverside

The obesity epidemic is largely a result of overeating high-fat and high-sugar diets combined with a sedentary lifestyle. Dysregulation of several neural and molecular pathways have been identified that contribute to obesity and overeating, including the gut-brain endocannabinoid system (eCBs). The eCBs is ubiquitously expressed throughout the CNS and peripheral tissues, including the gastrointestinal tract, and plays a major role in food intake, energy homeostasis, and reward. It is largely unknown if gut-brain eCBs signaling controls brain reward mechanisms, and elucidating these roles is the focus of this project. Our lab has reported that rodents display strong preferences, when given a choice, for a Western-style diet (WD, high fat/sucrose) versus standard rodent chow (low fat/no sucrose). Importantly, this preference was attenuated in mice treated with a cannabinoid subtype-1 receptor (CB1R) antagonist and in mice with conditional deletion of CB1Rs in the gut lining (IntCB1^{-/-})³. It is unclear, however, if CB1Rs in the intestinal epithelium mediate gut-brain neurotransmission that controls dopaminergic signaling associated with palatable food preference. In this study, we are beginning to explore roles for CB1R in the gut in these processes by examining dopaminergic signaling in the nucleus accumbens using an acute dietary preference test in tandem with the genetically encoded fluorescent dopamine sensor (GrabDA). These studies will be important in the development of therapeutics that target the endocannabinoid system in the periphery for the treatment of overeating or obesity that are void of psychiatric side effects inherent to drugs that directly access the brain (i.e., rimonabant).

5. The bidirectional effect of 2-AG on hyperdopaminergic states: Implications for therapeutic 2-AG modulation in psychosis

Catharine A. Mielnik, Claudia Lutelmowski, Clare T. Johnson, Julia Zebarth, Junchao Tong, Anna Pees, Andrea Narvaez, Kristyn Fournier, Marija Milenkovic, Wendy Horsfall, Steven H. Liang, Isabelle Boileau, Walter Swardfager, Heather B. Bradshaw, N. Vasdev, Ali Salahpour, Ruth A. Ross

University of Toronto

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The endocannabinoid system is dysregulated in schizophrenia (SCZ). Enzymes in the biosynthesis of 2-arachidonylglycerol (2-AG) are altered in SCZ; DAGL (2-AG synthesis) and MAGL (2-AG metabolism) are decreased in patients with first episode psychosis/SCZ, and 2-AG is elevated in individuals at high risk of psychosis. In certain clinical contexts, elevation of 2-AG is coveted, therefore clinical trials of MAGL inhibitors (MAGLi) are underway. However, evidence suggests increasing 2-AG may be detrimental in SCZ. Before wide therapeutic use, it's imperative to understand the effect of MAGLi in vulnerable SCZ populations, and whether decreasing 2-AG is therapeutic. Therefore, we assessed pre-clinical effects of MAGLi (increase 2-AG) and DAGLi (decrease 2-AG) in two models of hyperdopaminergia; based on well-established association between SCZ and increased subcortical dopamine.

Dopamine transporter knockout (DATKO) present with subcortical hyperdopaminergia; exploratory hyperactivity, impaired sensorimotor gating, blunted response to psychostimulants, and disrupted lipid profiles. MAGLi exacerbated hyperlocomotion, sensorimotor deficits, and further disrupted lipid networks in DATKO. MAGLi increased reward association in DATKO, but not WT, suggesting a worrisome addiction liability. MAGLi effects weren't limited to DATKO; it exacerbated psychostimulant responses in C57BL/6J. Data suggests that increasing 2-AG via MAGLi exacerbates states of hyperdopaminergia, mediated by CB1. Interestingly, decreasing 2-AG (via DAGLi) presented opposite effects on all measured hyperdopaminergic behavioural outputs in both DATKO and C57BL/6J, highlighting a potential therapeutic avenue for novel, dopamine-indirect, treatments that target lowering 2-AG in vulnerable SCZ populations.

6. Cannabis as a catalyst for harm reduction? An exploratory analysis among people who use drugs in the Inland Empire

Jennifer Syvertsen (1), Ale Cabral (2), Isabelle Swanson (3), Equinox Hartman (1), Sarah Zimmerman (1)

University of California, Riverside

University of California, Los Angeles

California State University, Long Beach

Emerging research has documented the potential of cannabis to help people cut down or abstain from other drug and alcohol use. As a psychoactive plant with understudied medicinal properties, this exploratory analysis considers cannabis as a catalyst for harm reduction, or the positive changes that people can make in navigating less harmful addiction trajectories.

This analysis draws from surveys conducted in 2023 with people who use drugs (e.g., opioids, methamphetamine) to learn about overdose in the Inland Empire. We employed descriptive statistics and multivariable modeling to examine cannabis use in the context of harm reduction.

Nearly all participants reported ever using cannabis (95%, n=187); participants were an average age of 43, 32% were women, and 60% identified as non-white. Nearly half (48%) reported using cannabis to “manage your other alcohol and drug use,” suggesting its use as a form of harm reduction. Univariate analyses indicated significant associations with younger age, positive screening for problematic drug use, perceived stress, opioid use, ever injecting drugs, experiencing an opioid overdose, and overamping on stimulants. In the final model, younger age, opioid overdose, and overamping remained independently associated with cannabis use as harm reduction.

In our sample of people who use drugs, cannabis use as harm reduction may be indicative of more intensive drug use trajectories. Our work calls for further biocultural research on the potential biochemical mechanisms and subjective effects of cannabis in the context of polydrug use, including its broader conceptualization as a catalyst for “any positive change” as advocated by harm reductionists.

7. Impact of Cannabis Exposure on Gut Barrier Function in Health and Disease

Martin Olmos, Nicholas DiPatrizio

University of California, Riverside

The endocannabinoid (eCB) system is a lipid-derived signaling pathway that controls food intake, energy homeostasis, and reward (Argueta, et al., 2019). This system is expressed throughout the body, including in the gastrointestinal tract, where it becomes dysregulated in diet-induced obesity (DIO) and may participate in gut barrier function; however, it is unclear if the eCB system exerts a protective or detrimental role in gut function. Indeed, studies from our lab suggest that the eCB system in intestinal epithelial cells controls gut barrier function, associated inflammation, and its activity exerts a protective influence during diet-induced obesity in mice (Wiley & DiPatrizio, 2022). Moreover, studies suggest that activating the eCB system with chemicals found in cannabis prevents colitis and increases colonic barrier integrity (Becker 2021). Conversely, other studies have shown that pharmacological activation of cannabinoid receptors in lean mice led to an increase in plasma levels of LPS and its high presence in circulation may suggest compromised gut barrier function (Alhouayek, et al., 2011). Given these discrepancies as to the protective or detrimental roles for the eCB system in gut function, we now are examining the impact of THC (i.e., the primary intoxicating chemical in cannabis), as well as whole cannabis extracts, on gut barrier function and inflammation in two mouse models of disrupted gut function: (i) a diet-induced obese mouse model with a mild phenotype of disrupted barrier function, and (ii) a mouse model of colitis (DSS). Preliminary results highlight a largely protective effect for chemicals in the obese model that requires cannabinoid CB1 receptors in the intestinal epithelium in a sex-dependent manner. The knowledge gained from these investigations will provide critical insights into the therapeutic potential of cannabis as a treatment for diseases that affect gastrointestinal barrier function.

8. The Effects of Maternal Exposure to Δ 9-THC During the Period of Rapid Placental Growth in Swiss Webster Mice

Sandra M. Banzon, Zoe Walker, Juanita Jellyman

California State Polytechnic University, Pomona

Over the past decade, marijuana use has increased, including use by pregnant women. Exposure to the psychoactive phytocannabinoid in *Cannabis sativa*, delta-9-tetrahydrocannabinol (Δ 9-THC), throughout pregnancy decreases birth weight in humans and animals. However, whether maternal exposure to Δ 9-THC during the period of rapid placental growth (gestational days 11-16) decreases fetal weight is unknown. All experiments were approved by the Institutional Animal Care and Use Committee at Cal Poly Pomona. Pregnant Swiss Webster mice were untreated (control group, n=24), received an intraperitoneal (IP) injection of vehicle (ethanol, cremophor, and saline; 1:1:18; vehicle group; n=21), or 4 mg/kg Δ 9-THC in vehicle (Δ 9-THC group; n=20). After euthanasia on day 16 or day 19 of gestation (term ~20 days), fetuses and their placentas were weighed, and the fetal: placental ratio was calculated as an index of placental efficiency. When data were analyzed for placentas on day 16, there was no difference in placental weight among the groups. There was a difference in placental weights on day 19 in the control, vehicle-treated, and THC-treated (0.08 ± 0.003 g; 0.08 ± 0.004 g; 0.09 ± 0.001 g; $P < 0.05$) mice, respectively. There was a difference in fetal weights on day 16 in control, vehicle-treated, and THC-treated (0.43 ± 0.02 g; 0.47 ± 0.03 g; 0.46 ± 0.04 g; $P < 0.05$) mice, respectively. Fetal weights on day 19 were different in control, vehicle-treated, and THC-treated (1.20 ± 0.04 g; 1.25 ± 0.03 g; 1.18 ± 0.03 g; $P < 0.05$) mice, respectively. Male placentas on day 19 (0.09 ± 0.003 g; 0.09 ± 0.004 g; 0.09 ± 0.003 g; $P < 0.05$) were different in comparison to male placentas on day 16 (0.09 ± 0.004 g; 0.09 ± 0.004 g; 0.09 ± 0.004 g). Female placentas on day 19 (0.09 ± 0.003 g; 0.09 ± 0.004 g; 0.09 ± 0.003 g; $P < 0.05$) were different in comparison to female placentas on day 16 (0.08 ± 0.005 g; 0.08 ± 0.003 g; 0.08 ± 0.006 g). Male fetal weights were different on day 16 (0.43 ± 0.01 g; 0.48 ± 0.03 g; 0.44 ± 0.03 g) and day 19 (1.23 ± 0.04 g; 1.28 ± 0.05 g; 1.21 ± 0.03 g). Female fetal weights were different on day 16 (0.43 ± 0.02 g; 0.45 ± 0.03 g; 0.45 ± 0.04 g) and day 19 (1.19 ± 0.03 g; 1.23 ± 0.03 g; 1.15 ± 0.04 g). The data suggests that maternal exposure to Δ 9-THC during the period of most rapid growth of the placenta does alter placental and fetal growth.

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Andrew Villa	avill307@ucr.edu	Zlebnik Lab	Graduate Student
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