BIOGRAPHICAL SKETCH

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NAME: Childs, Jessica

eRA COMMONS USER NAME (credential, e.g., agency login): jessicachilds

POSITION TITLE: Assistant Professor

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable. Add/delete rows as necessary.)*

INSTITUTION AND LOCATION	DEGREE (if applicable)	END DATE MM/YYYY	FIELD OF STUDY
UT Dallas, Richardson, TX	BS	05/2012	Neuroscience
UT Dallas, Richardson, TX	MS	05/2016	Cognition and Neuroscience
UT Dallas, Richardson, TX	PHD	12/2018	Cognition and Neuroscience
UC Irvine, Irvine, California	Postdoctoral Fellow	09/2025	Neuroepigenetics of Substance Use Disorder

A. Personal Statement

My long-term goal is to lead an academic research program focused on disorders with a maladaptive neuroplasticity component, such as substance use disorders and stress and anxiety disorders, using the converging methods of behavioral modeling, electrophysiology, molecular biology, and bioinformatics. I am specifically interested in discovering mechanisms that encode pathological affective cue associations, and subsequently in harnessing these mechanisms to reverse or ameliorate maladaptive behavioral responses, such as relapse in substance use disorders. My graduate research under Dr. Sven Kroener focused on the process of extinction learning, specifically on methods of facilitating extinction of both fear memories and drug-seeking behaviors. Extinction is a process that requires new learning about a conditioned stimulus that previously predicted aversive outcomes (in relation to fear memories) or pleasurable outcomes (in relation to drug-seeking behaviors). Augmenting extinction is a key approach to addressing maladaptive responses related to fear (posttraumatic stress disorder) and to drugs of abuse (addiction), and thus developing new methods to facilitate extinction is both important and highly translational. In my graduate research I found vagus nerve stimulation (VNS) could facilitate extinction of fear memories and drug-seeking behaviors (please see Contributions to Science section below). In one study, I found that VNS enhanced extinction of conditioned fear and modulated plasticity between the ventromedial prefrontal cortex and the amygdala—two important regions involved in fear memory (Pena and Childs, et al., 2014). Similarly, I found that VNS facilitated extinction of drug-seeking behavior and reduced reinstatement, potentially by altering plasticity in the extinction circuits (Childs et al., 2016) and that the therapeutic effect of VNS on reinstatement was independent of context as long as animals had the ability to engage in the operant response during extinction training (Childs et al., 2019). In the last two studies, I used two widely employed models of drug-seeking in rats: conditioned place preference and operant cocaine self-administration. In graduate school, I lacked the tools to answer important questions about how VNS was affecting neuroplasticity. Therefore, in my postdoctoral research, I chose to continue studying models of addiction under Dr. Marcelo Wood, an expert in epigenetics and memory. In Dr. Wood's lab I studied reinstatement of drug-seeking behavior. We discovered that the medial habenula is critically involved in reinstatement of cocaine self-administration, and that this effect is dependent on the expression of a transcription factor called NR4A2 (Childs et al., 2024). Working on this project, I became familiar with state-of-the-art molecular and genetic tools, as well as sophisticated behavioral approaches. I was part of several collaborations, including a project with Dr. Gary Lynch examining synaptic function in medial habenula cholinergic neurons in cocaine exposed animals, and a project with Dr. Vivek Swarup using bioinformatics to study the transcriptomic landscape of the MHb after reinstatement. I am now an Assistant Professor in the Department of Biomedical Sciences at Marshall University's Joan C. Edwards

School of Medicine, where I am establishing an independent research program centered on the medial habenula and its role in relapse vulnerability. My lab integrates behavioral neuroscience, epigenetics, and computational biology to understand how experience-dependent molecular and circuit-level plasticity contributes to substance use and affective disorders. Building on my prior work in extinction learning, vagus nerve stimulation, and medial habenula regulation of drug-seeking, my current studies focus on identifying persistent epigenetic signatures that encode drug experience and promote relapse-related behaviors.

B. Positions, Scientific Appointments and Honors

Positions and Scientific Appointments

2025 -	Assistant Professor, Marshall University Biomedical Sciences, Huntington, WV
2024 - 2025	Project Scientist , UC Irvine Neurobiology and Behavior, Marcelo Wood, Irvine, CA
2022 - 2024	NIDA F32 NRSA Postdoctoral Fellow, UC Irvine Neurobiology and Behavior, Marcelo Wood, Irvine , CA
2019 - 2022	Hewitt Foundation Postdoctoral Fellow, UC Irvine Neurobiology and Behavior, Marcelo Wood, Irvine , CA
2013 - 2018	Graduate Student, UT Dallas Behavioral and Brain Sciences, Sven Kroener, Richardson, TX

C. Contribution to Science

1. Graduate Research: Extinction learning paired with vagus nerve stimulation enhances behavioral outcomes and drives plasticity in extinction networks. My graduate research focused on reducing expression of the maladaptive cue and/or environmental associations made during drug use, expressed as relapse to drug-seeking behavior in models of substance use disorder. The approach I used involved vagus nerve stimulation (VNS), currently FDA approved for the treatment of epilepsy and depression. I examined the effect of VNS during extinction learning to enhance extinction, and ultimately reduce relapse-like behavior. When given during extinction training, VNS was found to reduce rates of relapse and to alter connectivity between the infralimbic prefrontal cortex and basolateral amygdala (Childs et al., 2016). This involved using the ‘gold standard’ approach of self-administration in rats, as well as electrophysiology, both of which I developed in Dr. Kroener’s lab as a graduate student. Overall, these preclinical findings open the door to human studies and provide important information to the addiction research community about the involvement of the basolateral amygdala in the reward circuit, which is currently supported by a small but growing body of research. Prior to my research examining VNS effects on extinction of drug-seeking behavior, I performed a study examining the ability of VNS to facilitate extinction of fear memory (Pena and Childs, et al., 2014). This established the foundation for my subsequent addiction studies. The studies below summarize our findings.
    a. Driskill CM, Childs JE, Phensy AJ, Rodriguez SR, O'Brien JT, Lindquist KL, Naderi A, Bordieanu B, McGinty JF, Kroener S. Vagus Nerve Stimulation (VNS) Modulates Synaptic Plasticity in the Infralimbic Cortex via Trk-B Receptor Activation to Reduce Drug-Seeking in Male Rats. J Neurosci. 2024 Jun 5;44(23) PubMed Central PMCID: PMC11154660.
    b. Childs JE, Kim S, Driskill CM, Hsiu E, Kroener S. Vagus nerve stimulation during extinction learning reduces conditioned place preference and context-induced reinstatement of cocaine seeking. Brain Stimul. 2019 Nov-Dec;12(6):1448-1455. PubMed Central PMCID: PMC10766375.
    c. Childs JE, DeLeon J, Nickel E, Kroener S. Vagus nerve stimulation reduces cocaine seeking and alters plasticity in the extinction network. Learn Mem. 2017 Jan;24(1):35-42. PubMed Central PMCID: PMC5159656.
    d. Childs JE, Alvarez-Dieppa AC, McIntyre CK, Kroener S. Vagus Nerve Stimulation as a Tool to Induce Plasticity in Pathways Relevant for Extinction Learning. J Vis Exp. 2015 Aug 21; PubMed Central PMCID: PMC4591905.

2. Postdoctoral Research: Examining molecular mechanisms of reinstatement in the medial habenula using cocaine self-administration in male and female mice. My postdoctoral research focused on the role the medial habenula (MHb) plays in regulating reinstatement of cocaine self-administration. My first experiment found that chemogenetic activation of the cholinergic neurons in the MHb facilitated cocaine primed reinstatement of self-administration. These data were critical for the resubmission of an ultimately successful NIDA R01 grant in the Wood lab. For my next experiments I needed bidirectional control of reinstatement, so after directly conversing with Dr. David Self, who is an expert in mouse self-administration, I implemented an incubation of craving model that enabled cued reinstatement of self-administration in mice. Using this model, I am studied the effects of MHb activation and inactivation on reinstatement behavior, and I also used single nuclei RNA sequencing to look at changes in the MHb transcriptome after reinstatement (paper in submission). I was also able to demonstrate that in key epigenetic targets within the MHb regulate cued reinstatement without affecting response during self-administration or extinction (Childs et al., 2024).
    a. Alizo Vera V, Childs JE, Kim J, Matheos DP, Wood MA. Expression of HDAC3-Y298H Point Mutant in Medial Habenula Cholinergic Neurons Has No Effect on Cocaine-Induced Behaviors. eNeuro. 2025 May;12(5) PubMed Central PMCID: PMC12077810.
    b. Childs JE, Morabito S, Das S, Santelli C, Pham V, Kusche K, Vera VA, Reese F, Campbell RR, Matheos DP, Swarup V, Wood MA. Relapse to cocaine seeking is regulated by medial habenula NR4A2/NURR1 in mice. Cell Rep. 2024 Mar 26;43(3):113956. PubMed Central PMCID: PMC11100346.
3. During my postdoc I also contributed to several collaborative studies, including a project in the Wood lab that used RNA sequencing to study the VTA after drug exposure in the four most common rodent models of addiction, including acute and chronic passive administration, conditioned place preference, and self-administration. Each paradigm was found to induce different sets of genes, and within self-administration expression levels correlated with cocaine intake.
    a. Campbell RR, Chen S, Beardwood JH, López AJ, Pham LV, Keiser AM, Childs JE, Matheos DP, Swarup V, Baldi P, Wood MA. Cocaine induces paradigm-specific changes to the transcriptome within the ventral tegmental area. Neuropsychopharmacology. 2021 Sep;46(10):1768-1779. PubMed Central PMCID: PMC8357835.
4. I was also able to contribute to a tremendous undertaking at UCI in which knock-in mice expressing human amyloid beta were generated to model late onset Alzheimer's Disease (AD). An array of measures was used to validate the use of these animals to model late onset AD, and they were found to have age-dependent changes in behavior, synaptic plasticity, inflammatory response, and in the transcriptome (changes in gene expression relevant for metabolism and neuroplasticity).
    a. Baglietto-Vargas D, Forner S, Cai L, Martini AC, Trujillo-Estrada L, Swarup V, Nguyen MMT, Do Huynh K, Javonillo DI, Tran KM, Phan J, Jiang S, Kramár EA, Nuñez-Diaz C, Balderrama-Gutierrez G, Garcia F, Childs J, Rodriguez-Ortiz CJ, Garcia-Leon JA, Kitazawa M, Shahnawaz M, Matheos DP, Ma X, Da Cunha C, Walls KC, Ager RR, Soto C, Gutierrez A, Moreno-Gonzalez I, Mortazavi A, Tenner AJ, MacGregor GR, Wood M, Green KN, LaFerla FM. Generation of a humanized Aβ expressing mouse demonstrating aspects of Alzheimer's disease-like pathology. Nat Commun. 2021 Apr 23;12(1):2421. PubMed Central PMCID: PMC8065162.