BIOGRAPHICAL SKETCH

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NAME: Donita Lynn Robinson

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

POSITION TITLE: Professor of Psychiatry, Associate Dean for Graduate Education

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)*	Completion Date MM/YYYY	FIELD OF STUDY
University of Texas at Austin	B.A.	5/1991	Psychology
University of Michigan at Ann Arbor	M.A.	8/1993	Biological psychology
University of Texas at Austin	Ph.D.	5/2000	Neuroscience
University of North Carolina at Chapel Hill	Post-doc	12/2002	Analytical chemistry

A. Personal Statement

I am a Professor in Psychiatry and a member of the Bowles Center for Alcohol Studies, Neuroscience Curriculum, Nutrition Obesity Research Center, and North Carolina Translational and Clinical Sciences Institute. I am currently PI or co-PI on research grants, including Project 3 and the Information Dissemination Core of the NIAAA-funded UNC P60 Alcohol Research Center. I have an administrative role as the Project Director for the NIAAA-funded NADIA Consortium. Finally, I am highly involved in scientific training as the Associate Dean of Graduate Education for the School of Medicine, the Education & Outreach Director of the Bowles Center for Alcohol Studies, contact PI of a NIAAA-funded postdoctoral T32, and contact PI of the NIGMS-funded R25 UNC PREP. My scientific expertise lies in neural dynamics of corticostriatal circuits during motivated behavior and drug exposure in animal models. My current research focuses on the brain regions that orchestrate decision making, and I investigate how addictive drugs either acutely or persistently alter function of these circuits and the resulting behavior. My goal is that our research will mechanistically inform human basic and clinical research and address public health concerns, such as the neurobiological consequences of underage drinking. Thus, I work closely with researchers studying similar brain systems and behavior in people, to ensure that my rodent studies model human phenomenon and are positioned for translation.

Below are literature reviews that highlight my expertise:

1. C.A. Dannenhoffer, M.M. Robertson, V.A. Macht, S.M. Mooney, C.A. Boettiger, **D.L. Robinson** (2021). Chronic alcohol exposure during critical periods of development differentially impacts cognitive flexibility and related circuitry. International Review of Neurobiology 160:305-340. PMC8674885
2. **D.L. Robinson,** L.R. Amodeo, L.J. Chandler, F.T. Crews, C.L. Ehlers, A. Gómez-A, K.L. Healey, C.M. Kuhn, V.A. Macht, S.A. Marshall, H.S. Swartzwelder, E.I. Varlinskaya, D.F. Werner (2021). The role of sex in the persistent effects of adolescent alcohol exposure on behavior and neurobiology in rodents. International Review of Neurobiology 160:305-340. PMC8672816
3. T.H. McKim, T.A. Shnitko, **D.L. Robinson**, C.A. Boettiger (2016). Translational research on habit and alcohol. *Current Addiction Reports,* 3(1), 37-49. PMC4767272
4. **D.L. Robinson**, A. Hermans, A.T. Seipel and R.M. Wightman (2008). Monitoring rapid chemical communication in the brain. *Chemical Reviews,* 108: 2554-2584. PMC3110685

Ongoing projects that I would like to highlight include:

P60 AA011605 “Molecular and Circuit Pathogenesis in Alcoholism”
Center PI: Thomas Kash (previously Fulton Crews)

Project: "Frontolimbic circuitry, behavioral flexibility, and adolescent alcohol history"
Project Leads: Charlotte A. Boettiger, Donita L. Robinson
Period: 12/01/2022 – 11/30/2029
Administrative supplement: "Dietary choline mitigation of adolescent alcohol-induced deficits in adult cognitive flexibility"

Component: "Translation/Information Dissemination Component"
Project Leads: Donita L. Robinson, Sara Faccidomo
Period: 12/01/2022 – 11/30/2029

R25 GM089569 "UNC PREP in the Biomedical Sciences"
MPI: Donita L. Robinson, Jose Rodríguez-Romaguera
Period: 04/01/2018 – 03/31/2024

U24AA020024 "UNC-CH NADIA Admin Core"
PI: Fulton T. Crews, Role: Project Director
Period: 09/01/2010 – 08/31/2025

P50 DA039841 "Center for Systems Neurogenetics of Addiction"
PI: Chessler (PI), Tarantino (Subcontract PI), Role: Investigator
Period: 09/01/2022 – 08/31/2027

B. Positions, Scientific Appointments, and Honors

Positions and Employment

1994 – 1995 Laboratory Assistant, Physiology, University of Texas Southwest Medical Center at Dallas
2002 – 2003 Research Associate, Analytical Chemistry, UNC Chapel Hill
2003 – 2009 Research Assistant Professor, Department of Psychiatry, School of Medicine, UNC Chapel Hill
2009 – 2015 Assistant Professor, Department of Psychiatry, School of Medicine, UNC Chapel Hill
2010 – now Director of Education and Outreach for the Bowles Center for Alcohol Studies, School of Medicine, UNC Chapel Hill
2015 – 2020 Associate Professor (tenure), Department of Psychiatry, School of Medicine, UNC Chapel Hill
2015 – 2021 Faculty Director of UNC PREP, School of Medicine, UNC Chapel Hill
2019 – 2020 Faculty Director of Biological & Biomedical Sciences Program, Office of Graduate Education, School of Medicine, UNC Chapel Hill
2020 – now Full Professor (tenure), Department of Psychiatry, School of Medicine, UNC Chapel Hill
2020 – 2021 Assistant Dean of Graduate Education, School of Medicine, UNC Chapel Hill
2021 – now Associate Dean of Graduate Education, School of Medicine, UNC Chapel Hill

Scientific Appointments and Professional Memberships

Memberships: Society for Neuroscience; American Chemical Society; Research Society on Alcoholism (*Membership committee, 2006 – 2008; Education Committee, 2006-2017, Chair 2014-2016; Publication Committee 2017 - now)*; Faculty for Undergraduate Neuroscience

Ad hoc reviewer for multiple journals since 2005, including Addiction Biology, Alcoholism: Clinical and Experimental Research, Behavioral Neuroscience, European Journal of Neuroscience, Journal of Neurochemistry, Journal of Neuroscience, Molecular Psychiatry, Neuropharmacology, Neuropsychopharmacology, Psychopharmacology (Advisory Editor)

Ad hoc reviewer for granting agencies since 2009, including NIH, NSF, ABMRF/The Foundation for Alcohol Research, Dutch Research Council, Israel Science Foundation, UK Medical Research Council

Member of NIH study section NAL since 2019

Selected Honors and Awards

1990 – 1993 Regents Fellowship from the Rackham School of Graduate Studies, University of Michigan.
1995 – 1999 Predoctoral fellowship from NIAAA Training Grant AA07471, University of Texas.
1998 John P. McGovern grant from Texas Research Society on Alcoholism.
1999 – 2000 Fred Murphy Jones predoctoral fellowship from the University of Texas Waggoner Alcoholism and Addiction Research Center, University of Texas.
2000 Biomedical Research Award & NIAAA Student Merit Award, Research Society on Alcoholism.
2000 – 2002 Postdoctoral fellowship from NIAAA Training Grant AA07573, University of North Carolina.
2002, 2003 NIAAA Junior Investigator Award, Research Society on Alcoholism.
2003, 2005 Kopin travel award to Gordon Conference on Catecholamines.
2015 UNC Neuroscience Curriculum student-selected Mentor of the Year Award

C. Contributions to Science

1. Alcohol pharmacokinetics in the brain. As a doctoral student, my research centered on quantitative measurements of alcohol pharmacokinetics in the brains of awake rats. This research provided the first quantitative measurement of alcohol pharmacokinetics in the brain via microdialysis sampling and applied this tool to investigate the effects of sex and estrous cycle on alcohol pharmacokinetics that potentially influence drinking behavior. In a series of publications, I demonstrated that sex but not estrous cycle altered the distribution of alcohol to the brain. *(Published under Crippens and Robinson)*
   a. **D. Crippens**, M.L. White, M.A. George, J.N. Jaworski, L.J. Brunner, F.E. Lancaster, R.A. Gonzales (1999). Gender differences in blood levels, but not brain levels, of ethanol in rats. *Alcoholism: Clinical and Experimental Research,* 23: 414-420. PMID: 10195812
   b. **D.L. Robinson**, J.A. Lara, L.J. Brunner, R.A. Gonzales (2000). Quantification of ethanol concentrations in the extracellular fluid of the rat brain: in vivo calibration of microdialysis probes. *Journal of Neurochemistry*, 75: 1685-1693. PMID: 10987851
   c. **D.L. Robinson**, L.J. Brunner, R.A. Gonzales (2002). Effect of gender and estrous cycle on the pharmacokinetics of ethanol in the rat brain. *Alcoholism: Clinical and Experimental Research*, 26: 165-172. PMID: 11964555
   d. R.A. Gonzales, A. Tang, **D.L. Robinson** (2002). Quantitative microdialysis for in vivo studies of ethanol pharmacodynamics. In Y. Liu and D.M. Lovinger (Eds.) *Methods in Alcohol-Related Neuroscience Research*, pp. 287-317, CRC Press: Boca Raton.

2. Role of spontaneous dopamine transients in naturalistic behavior, including maternal behavior. As a postdoc and assistant professor, my research determined that dopamine transients occur throughout the dorsal and ventral striatum at a baseline rate, that they increase upon the presentation of salient stimuli (especially social stimuli), that there are developmental differences in the expression of dopamine transients, and that they can be pharmacologically manipulated. These studies were among the first to describe subsecond dopamine fluctuations during behavior and have been critical for understanding phasic dopamine transmission. I have also written several highly-cited reviews on phasic dopamine release *in vivo*. As an associate professor, I have investigated dopamine transient release during maternal behavior in typical and cocaine-exposed rat mothers. I found that evoked dopamine is higher during early postpartum rats than in virgin females, and that maternal cocaine exposure blunts this effect of parturition. In addition, dopamine transients that naturally occur during maternal behavior are less frequent in cocaine-exposed then control-exposed rat mothers. *(Supported by NIAAA, NIDA)*
   a. **D.L. Robinson**, M.L. Heien, R.M. Wightman (2002). Frequency of dopamine concentration transients increases in dorsal and ventral striatum of male rats during introduction of conspecifics. *Journal of Neuroscience*, 22: 10477-10486. PMID: 12451147
   b. **D.L. Robinson**, D.L. Zitzman, K.J. Smith, L.P. Spear (2011). Fast dopamine release events in the nucleus accumbens of early adolescent rats. *Neuroscience*, 176: 296-307. PMC3061289
   c. T.A. Shnitko*, K.D. Mace*, K.M. Sullivan, W.K. Martin, J.M. Johns, **D.L. Robinson** (2017). Use of fast-scan cyclic voltammetry to assess phasic dopamine release in rat models of early postpartum maternal behavior and neglect. *Behavioural Pharmacology* 28: 648-660. PMC5680131.
   d. A. Gómez-A, T.A. Shnitko, K.L. Caref, S.M. Nicola, **D.L. Robinson** (2022)**.** Stimulus predicting high-calorie reward increases dopamine release and drives food consumption in absence of homeostatic need. *Nutritional Neuroscience*, doi: 10.1080/1028415X.2020.1782613. PMC7758188

3. Pharmacological effects of alcohol on phasic dopamine signals. A large part of my independent research has centered on the acute and chronic effects of ethanol on dopamine transients in corticostriatal circuitry. I have directly compared the effects of alcohol on phasic versus tonic extracellular dopamine release, revealing that while alcohol produces reliable increases in tonic dopamine concentrations, its effects on phasic dopamine release are spatially selective. I have also quantified alcohol effects on dopamine release and clearance, both in the striatum and prefrontal cortex. While effects were largely consistent in naïve rats across regions, dopamine dynamics in the nucleus accumbens were insensitive to alcohol challenge in rats with a history of adolescent alcohol exposure. Notably, insensitivity to alcohol effects is a risk factor for alcohol use disorder. *(Supported by NIAAA, NIDA)*
   a. **D.L. Robinson**, T. Volz, J.O. Schenk, R.M. Wightman (2005). Acute ethanol decreases dopamine transporter velocity in rat striatum: in vivo and in vitro electrochemical measurements. *Alcoholism: Clinical and Experimental Research*, 29: 746-755. PMID: 15897718

b. **D.L. Robinson**, E.C. Howard, S. McConnell, R.A. Gonzales, R.M. Wightman (2009). Disparity between tonic and phasic ethanol-induced dopamine increases in the nucleus accumbens of rats. *Alcoholism: Clinical and Experimental Research*, 33:1187-96. PMC2947861
c. T.A. Shnitko, L. Kennerly, L.P. Spear, **D.L. Robinson** (2014). Ethanol reduces evoked dopamine release and slows clearance in the rat medial prefrontal cortex. *Alcoholism: Clinical and Experimental Research,* 38(12): 2969-2977. PMC4293045
d. T.A. Shnitko, **D.L. Robinson** (2015). Regional variation in phasic dopamine encoding of alcohol self-administration in rats. *ACS Chemical Neuroscience,* 6(1):147–154. PMC4304482

4. Neurophysiology during alcohol drinking and motivated behavior. As an assistant professor, I studied real-time neuronal firing patterns during operant self-administration of alcohol and non-drug rewards. This research found that alcohol reward is often processed distinctly from non-drug reward, that multiple basal ganglia circuits are active during both alcohol seeking and in response to alcohol-predictive cues, that goal-directed and habitual alcohol seeking preferentially induce specific firing patterns in dorsal striatal neurons, and that rats are differentially sensitive to naltrexone depending on whether the reward is alcoholic and on what behavioral strategy the rats employ. These studies show that alcohol cues, seeking and reinforcement are encoded throughout the striatum and that the behavioral strategy employed by the animal - goal-directed versus habitual – may influence relapse-like behavior as well as response to therapeutic drugs, potentially due to the constellation of neural ensemble activity underlying that behavior. As an associate professor, this research has expanded to determine how prefrontal and striatal neurons encode reward-predictive cues in other contexts, such as Pavlovian conditioning, and how chronic nicotine and adolescent alcohol exposure alter neuronal firing patterns. *(Supported by NIAAA, Foundation of Hope)*
   a. **D.L. Robinson**, R.M. Carelli (2008). Distinct subsets of nucleus accumbens neurons encode operant responding for ethanol versus water. *European Journal of Neuroscience*, 28:1887-1894. PMC2597565
   b. R.F. Fanelli, J.T. Klein, R.M. Reese, **D.L. Robinson** (2013). Dorsomedial and dorsolateral striatum exhibit distinct phasic neuronal activity during alcohol self-administration in rats. *European Journal of Neuroscience*, 38:2637-2648. PMC3748264
   c. T.A. Shnitko, **D.L. Robinson** (2015). Regional variation in phasic dopamine encoding of alcohol self-administration in rats. *ACS Chemical Neuroscience*, 6(1): 147–154. PMC4304482
   d. S.J. Stringfield, M.I. Palmatier, C.A. Boettiger, **D.L. Robinson**. (2017) Orbitofrontal involvement in sign and goal tracking conditioned responses: effects of nicotine. *Neuropharmacology*, 116: 208-223. PMC5385154.

5. Effects of chronic drug exposure on behavioral flexibility. For the past several years, I have investigated how alcohol and nicotine – legal but addictive drugs – alter behavioral flexibility both acutely and across the lifespan. Much of this research is collaborative with Dr. Charlotte Boettiger (UNC Department of Psychology and Neuroscience), who studies the same neurocircuitry and behavioral constructs in human subjects. We found that both current nicotine exposure/use and alcohol binge exposure/drinking in adolescence increase sensitivity to reward-associated cues. In collaboration with Dr. Ian Shih (UNC Department of Neurology), I assess how drug exposure alters functional connectivity across brain regions in rats by using resting-state fMRI. We determined that adolescent alcohol exposure persistently blunted functional connectivity in fronto-striatal circuits, several weeks after the alcohol exposure ended. Moreover, we found that adolescent alcohol-induced reductions in functional connectivity within a subnetwork of affected brain regions statistically mediated errors committed during reversal learning. By testing the hypothesis that chronic drug exposure, particularly during adolescence, reduces behavioral flexibility in both humans and rodents, we gain translational value and can better understand the neurobiological consequences of drug use on adaptive decision making. *(Supported by NIAAA)*
   a. A.C. Madayag, S.J. Stringfield, K.J. Reissner, C.A. Boettiger, **D.L. Robinson.** (2017) Sex and adolescent ethanol exposure influence Pavlovian conditioned approach. *Alcoholism: Clinical & Experimental Research,* 41(4):846-856. PMC5419304.
   b. M.A. Broadwater, Y. Yu, S.-H. Lee, H. Zhu, F.T. Crews, **D.L. Robinson**, Y.-Y.I. Shih. (2018) Adolescent alcohol exposure alters frontolimbic resting state connectivity in adult rat brain. *Addiction Biology* 23(2):810-823. PMC5760482.
   c. S.J. Stringfield, A.C. Madayag, C.A. Boettiger, **D.L. Robinson** (2019). Sex differences in nicotine-enhanced Pavlovian conditioned approach in rats. *Biology of Sex Differences,* 10(1):37. PMC6637589.

d. A. Gómez-A, C.A. Dannenhoffer, A. Elton, S.H. Lee, W. Ban, Y.I. Shih, C.A. Boettiger, **D.L. Robinson** (2021). Altered cortico-subcortical network after adolescent alcohol exposure mediates behavioral deficits in flexible decision-making. *Frontiers in Pharmacology*, 12: 778884. PMC8666507.

Complete bibliography: ORCID ID 0000-0001-7540-3363; complete list of published work in MyBibliography https://www.ncbi.nlm.nih.gov/sites/myncbi/donita.robinson.1/bibliography/44286800/public/