BIOGRAPHICAL SKETCH

Provide the following information for the Senior/key personnel and other significant contributors.
Follow this format for each person. DO NOT EXCEED FIVE PAGES.

NAME: Dickson, Price E.

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

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)*	Completion Date MM/YYYY	FIELD OF STUDY
The University of Minnesota, Minneapolis, MN	B.A.	05/2004	Psychology
The University of Memphis, Memphis, TN	M.S.	05/2010	Experimental Psychology
The University of Memphis, Memphis, TN	Ph.D.	05/2013	Experimental Psychology
The Jackson Laboratory, Bar Harbor, ME	Postdoc	05/2013 – 05/2020	Behavioral Genetics

A. Personal Statement

The overarching goal driving my research is to identify the genetic, genomic, and environmental mechanisms underlying drug addiction, a heritable disease with devastating effects on individuals and society. To this end, I use a multidisciplinary approach which includes systems genetics, genomics, advanced mouse populations, and construct-valid behavioral techniques for addiction phenotyping, most notably intravenous drug self-administration. I completed my doctorate at the University of Memphis where I trained as a behavioral neuroscientist and focused on identifying genetic and neurophysiological mechanisms underlying psychiatric disorders using operant conditioning and in vivo electrochemistry in the mouse. As a graduate student, I lead the first systems genetics study of intravenous cocaine self-administration. During this study, we phenotyped 39 strains of the genetically complex BXD recombinant inbred panel and integrated these behavioral data with genomic data to identify gene candidates driving variation in cocaine use, including *Fam53b* which has been proposed as a human cocaine addiction candidate. After completing my Ph.D. in 2013, I relocated to The Jackson Laboratory in Bar Harbor Maine to complete postdoctoral training with Dr. Elissa Chesler. As a postdoc at JAX, I lead and published the first cocaine self-administration study in a genetically diverse mouse population (Diversity Outbred) and the first systems genetics study of sensation seeking using an operant model. In these and related studies, we identified shared genetic mechanisms driving cocaine self-administration, sensation seeking, and incentive salience attribution. Prior to leaving JAX for a faculty position at the Joan C. Edwards School of Medicine at Marshall University, I was the postdoctoral lead on the largest system genetics study of intravenous cocaine self-administration to date (a project in the Center for Systems Neurogenetics of Addiction) in which we phenotyped over 1200 genetically diverse mice including 50 strains of the Collaborative Cross recombinant inbred panel and 500 individual Diversity Outbred mice. My research at Marshall University is currently funded by an R00 from The National Institute on Drug Abuse. In this project, we are integrating the genetic diversity within the Collaborative Cross recombinant inbred panel, intravenous cocaine self-administration, and RNA sequencing in the nucleus accumbens to study the genetic mechanisms underlying stress-induced addiction vulnerability.

Recent papers relevant to this application:

a. Schoenrock SA, Kumar P, Gómez-A A, **Dickson PE**, Kim SM, Bailey L, Neira S, Riker KD, Farrington J, Gaines CH, Khan S, Wilcox TD, Roy TA, Leonardo MR, Olson AA, Gagnon LH, Philip VM, Valdar W, de Villena FP, Jentsch JD, Logan RW, McClung CA, Robinson DL, Chesler EJ, Tarantino LM. (2020) Characterization of genetically complex Collaborative Cross mouse strains that model divergent locomotor activating and reinforcing properties of cocaine. Psychopharmacology (Berl). doi: 10.1007/s00213-019-05429-3. PubMed PMID: 31897574.

b. **Dickson PE,** Roy TA, McNaughton KA, Wilcox TD, Kumar P, & Chesler EJ (2019). Systems genetics of sensation seeking. Genes Brain Behav, 18(3), e12519. doi:10.1111/gbb.12519 PMCID: PMC6965071

c. **Dickson PE,** Miller MM, Calton MA, Bubier JA, Cook MN, Goldowitz D, Chesler EJ, Mittleman G (2016) Systems genetics of intravenous cocaine self-administration in the BXD recombinant inbred mouse panel. Psychopharmacology (Berl). doi:10.1007/s00213-015-4147-z. PMCID: PMC4803082
d. **Dickson PE**, Ndukum J, Wilcox T, Clark J, Roy B, Zhang L, Li Y, Lin DT, Chesler EJ (2015). Association of novelty-related behaviors and intravenous cocaine self-administration in Diversity Outbred mice. Psychopharmacology (Berl) 232:1011-2. PMCID: PMC4774545

B. Positions and Honors

Positions and Employment

2006-2013	Graduate Research Assistant with Dr. Guy Mittleman, Department of Psychology, University of Memphis, Memphis, TN
2013-2020	Postdoctoral Associate with Dr. Elissa Chesler, The Jackson Laboratory, Bar Harbor, Maine
2020-present	Assistant Professor, Department of Biomedical Sciences, Joan C. Edwards School of Medicine, Marshall University, Huntington, WV

Honors

2007-2012	Research Travel Award, The University of Memphis, Memphis, TN
2009	Program Director's Award in Experimental Psychology, The Univ. of Memphis, Memphis, TN
2014	Travel Award, International Behavioural and Neural Genetics Society (IBANGS)
2018	NIH Pathway to Independence Award K99/R00 (NIDA)

Professional Memberships

2007-present	Society for Neuroscience
2013-present	International Behavioral and Neural Genetics Society

C. Contribution to Science (Chosen from a total of 21 peer-reviewed publications)

1. **Systems genetics of addiction.** My primary research goal is to identify and characterize the genetic, genomic, and environmental factors which drive drug use and the development of addiction. As a postdoctoral trainee at The Jackson laboratory, I identified genetic correlations among intravenous cocaine self-administration and addiction-relevant behaviors including novelty reactivity and novelty preference in the BXD and Diversity Outbred mouse populations. Using the founder strains of the Collaborative Cross recombinant inbred panel, I characterized the effects of sex and genotype on incentive salience attribution, a trait which is proposed to drive the development of addiction. I also identified a strong genetic correlation between incentive salience attribution and novelty-reactivity. These findings illustrate the existence of an underlying genetic relationship between drug use and behavioral traits which have been observed to covary with and in some cases predict addiction in humans.

    a. **Dickson PE,** Roy TA, McNaughton KA, Wilcox TD, Kumar P, & Chesler EJ (2019). Systems genetics of sensation seeking. Genes Brain Behav, 18(3), e12519. doi:10.1111/gbb.12519 PMCID: PMC6965071
    b. **Dickson PE,** Miller MM, Calton MA, Bubier JA, Cook MN, Goldowitz D, Chesler EJ, Mittleman G (2016) Systems genetics of intravenous cocaine self-administration in the BXD recombinant inbred mouse panel. Psychopharmacology (Berl). doi:10.1007/s00213-015-4147-z. PMCID: PMC4803082
    c. **Dickson PE**, Ndukum J, Wilcox T, Clark J, Roy B, Zhang L, Li Y, Lin DT, Chesler EJ (2015). Association of novelty-related behaviors and intravenous cocaine self-administration in Diversity Outbred mice. Psychopharmacology (Berl) 232:1011-2. PMCID: PMC4774545
    d. **Dickson PE**, McNaughton KA, Hou L, Anderson LC, Long KH, Chesler EJ (2015). Sex and strain influence attribution of incentive salience to reward cues in mice. Behav Brain Res 292:305-315. PMCID: PMC4558302

2. **Nicotine during adolescence influences cocaine addiction.** As a graduate student, I identified adolescent nicotine exposure as a significant factor influencing adult intravenous cocaine self-administration in mice. I also identified genotype-dependent nicotine-induced perturbations of dopamine dynamics in the nucleus accumbens shell as a mechanism underlying this effect. These findings illustrate a potential mechanism underlying the observed "Gateway Effect" of nicotine in human populations.

a. **Dickson PE**, Miller MM, Rogers TD, Blaha CD, Mittleman G (2014). Effects of adolescent nicotine exposure and withdrawal on intravenous cocaine self-administration during adulthood in male C57BL/6J mice. Addict Biol 19:37-48. PMCID: PMC4084694
b. **Dickson PE**, Rogers TD, Lester DB, Miller MM, Matta SG, Chesler EJ, Goldowitz D, Blaha CD, Mittleman G (2011). Genotype-dependent effects of adolescent nicotine exposure on dopamine functional dynamics in the nucleus accumbens shell in male and female mice: a potential mechanism underlying the gateway effect of nicotine. Psychopharmacology (Berl) 215:631-42.

3. **Genetic factors driving executive dysfunction.** Impairments in higher cognitive functions such as behavioral flexibility and attentional control are thought to underlie, at least in part, the maladaptive behaviors which characterize a wide range of neuropsychiatric disorders including drug addiction and developmental disorders. During my graduate work, I used mouse models to examine the cerebellar contribution to these cognitive functions using operant conditioning assays including reversal learning and attentional set-shifting. During this work, I identified cerebellar mutants which exhibited profound behavioral flexibility deficits, learning deficits, or both including *Grid2*, *Fmr1*, and *dmd*. Furthermore, using a touchscreen-based assay, I characterized attentional set-shifting in the BXD founder strains and identified in DBA/2J relative to C57BL/6J mice a significantly elevated propensity for a highly salient stimulus to capture attention and drive behavior. Collectively, these findings reveal the effect of the cerebellum and genetic background on higher cognitive functions.

    a. **Dickson PE**, Rogers TD, Del Mar N, Martin LA, Heck D, Blaha CD, Goldowitz D, Mittleman G (2010) Behavioral flexibility in a mouse model of developmental cerebellar Purkinje cell loss. Neurobiol Learn Mem 94:220-8. PMCID: PMC2922405
    b. **Dickson PE**, Corkill B, McKimm E, Miller MM, Calton MA, Goldowitz D, Blaha CD, Mittleman G (2013) Effects of stimulus salience on touchscreen serial reversal learning in a mouse model of fragile X syndrome. Behav Brain Res 252:126-35. PMCID: PMC3854797
    c. **Dickson PE**, Calton MA, Mittleman G (2014) Performance of C57BL/6J and DBA/2J mice on a touchscreen-based attentional set-shifting task. Behav Brain Res 261:158-70. PMCID: PMC4060595
    d. **Dickson, PE,** Cairns, J, Goldowitz, D, and Mittleman, G (2016) Cerebellar contribution to higher and lower order rule learning and cognitive flexibility in mice. Neuroscience. doi:10.1016/j.neuroscience.2016.03.040. PMCID: PMC5031514

4. **Systems neuroscience of executive dysfunction.** Following the identification of cerebellar mutants which exhibited cognitive impairments, collaborators and I used *in vivo* electrochemical and immunohistochemical techniques to characterize underlying mechanisms driving these deficits. We found that neuropathological changes in the cerebellum of *Fmr1* and *Grid2* mutants resulted in a loss of functionality of circuitry connecting the cerebellum to medial prefrontal cortex (mPFC), and this was manifested as aberrant dopaminergic activity in the mPFC. Moreover, we identified glutamate as a modulator of mPFC dopaminergic activity and identified in *Fmr1* and *Grid2* mutants relative to controls a functional reorganization of the ventral tegmental area and thalamic pathways mediating cerebellar modulation of mPFC dopamine release. These findings offer a mechanism through which natural variation or pathology in cerebellar morphology can affect higher cognitive functions.

    a. Rogers TD, **Dickson PE**, Heck DH, Goldowitz D, Mittleman G, Blaha CD (2011). Connecting the dots of the cerebro-cerebellar role in cognitive function: neuronal pathways for cerebellar modulation of dopamine release in the prefrontal cortex. Synapse 65:1204-12. PMCID: PMC3854794
    b. Rogers TD, **Dickson PE**, McKimm E, Heck DH, Goldowitz D, Blaha CD, Mittleman G (2013). Reorganization of circuits underlying cerebellar modulation of prefrontal cortical dopamine in mouse models of autism spectrum disorder. Cerebellum 12:547-56. PMCID: PMC3854915
    c. Cairns J, Swanson D, Yeung J, Sinova A, Chan R, Potluri P, **Dickson PE**, Mittleman G, Goldowitz D (2017). Abnormalities in the structure and function of cerebellar neurons and neuroglia in the Lc/+ chimeric mouse model of variable developmental Purkinje cell loss. Cerebellum. doi: 10.1007/s12311-015-0756-7

Complete List of Published Work in MyBibliography:
http://www.ncbi.nlm.nih.gov/sites/myncbi/1NuJ8wdGkiM/bibliography/40334407/public/?sort=date&direction=descending

D. Research Support

Ongoing Research Support

4 R00 DA043573
Role: Principal Investigator
Duration of Funding: 07/01/2020 - 06/30/2023
Source: NIDA
Project Title: Discovery and Characterization of Genetic Mechanisms Driving Stress-Induced Addiction Vulnerability
Project Description: The goal of this project is to use advanced mouse resources to identify and characterize the genes and mechanisms in brain reward pathways driving the enhancing effect of environmental stress in addiction liability.

Completed Research Support

5 K99 DA043573
Role: Principal Investigator
Duration of Funding: 03/15/2018 - 02/28/2020
Source: NIDA
Project Title: Discovery and Characterization of Genetic Mechanisms Driving Stress-Induced Addiction Vulnerability
Project Description: The goal of this project was to use advanced mouse resources to identify and characterize the genes and mechanisms in brain reward pathways driving the enhancing effect of environmental stress in addiction liability.