BIOGRAPHICAL SKETCH

NAME: Tiffany Rogers

POSITION TITLE: Assistant Professor of Psychology, Middle Tennessee State University

EDUCATION/TRAINING:

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	Completion Date MM/YYYY	FIELD OF STUDY
Lipscomb University	B.S.	05/2006	Psychology
University of Memphis	M.A.	08/2009	Psychology
University of Memphis	Ph.D.	08/2012	Behavioral Neuroscience
Vanderbilt University	Postdoctoral Fellow	2012-2014	Molecular and Cellular Neuroscience
Vanderbilt University	Postdoctoral Fellow	2015	Molecular and Cellular Neuroscience

A. Personal Statement

As an early career investigator, I am continuing to hone my career goals as a scientist. I am now able to leverage my experiences across multiple techniques and mouse models to pursue an understanding of the neurochemical underpinnings of social behaviors in mice. I am currently interested in understanding the role of dopamine and oxytocin and their interactions to produce complex social behaviors.

B. Positions and Honors

Positions and Employment

2009 – 2012 Lecturer, Psychology Department, The University of Memphis, Memphis, TN
2012 – 2014 Postdoctoral Fellow, Psychiatry Department, Vanderbilt University, Nashville, TN
2015 Postdoctoral Fellow, Pharmacology Department, Vanderbilt University, Nashville, TN
2015 Scientific Director of Conte Behavioral Core, Vanderbilt University, Nashville, TN
2014 – 2018 Lecturer, Psychology Department, Lipscomb University, Nashville, TN
2015 – 2019 Full-time Lecturer, Psychology Department, Middle Tennessee State University, Murfreesboro, TN
2016 – 2018 Course Developer, Professional Studies Department, Lipscomb University, Nashville, TN
2017 – 2018 Lecturer, Biology Department, Lipscomb University, Nashville, TN
2019 – Assistant Professor, Psychology Department, Middle Tennessee State University, Murfreesboro, TN

Other Experience and Memberships

2013 – Member of Society for Neuroscience
2018 – Co-Sponsor for the Collegiate Neuroscience Society of Middle Tennessee State University

C. Contributions to Science

1. In my early work, I investigated a novel pathway originating in the cerebellum and terminating in the prefrontal cortex and its potential alterations in mouse models of autism using fixed potential amperometry. This work has provided evidence that cerebellar abnormalities observed in autism may be associated with some behavioral symptoms of autism via downstream cerebellar-prefrontal cortex circuitry alterations. The work also may prove useful in providing therapeutic targets for future treatments for patients with autism spectrum disorders.

a. **Rogers, T.D.**, Dickson, P.E., Heck, D.H., Goldowitz, D., Mittleman, G., & Blaha, C.D. (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(11)*,1204-12. PMID: 21638338
b. **Rogers, T.D.**, Dickson, P.E., McKimm, E., Heck, D.H., Goldowitz, D., & Blaha, C.D., & Mittleman, G. (2013). Reorganization of circuits underlying cerebellar modulation of prefrontal cortical dopamine in mouse models of autism spectrum disorder. *Cerebellum, 12(4)*, 547-56. PMID: 23436049
c. **Rogers, T.D.**, Lester, D.B., Dickson, P.E., Miller, M.M., Heck, D.H., Goldowitz, D., Mittleman, G., Blaha, C.D. (2010). Connecting the dots of the autism disconnection hypothesis: Neural pathways for cerebellar modulation of dopamine release in the prefrontal cortex. *Society for Neuroscience Abstracts*, 562.21.

d. **Rogers, T.D.**, Spight, V., Heck, D.H., Goldowitz, D., Mittleman, G., Blaha, C.D. (2011). Cerebellar Purkinje cell loss results in a shift in modulatory control of cortical dopamine release by two distinct cerebellar-prefrontal cortex pathways: Relevance to the Autism disconnection hypothesis. *Society for Neuroscience Abstracts*, 56.08.

2. I have worked with multiple mouse models of autism to attempt to better understand the neurobiological underpinnings of social behavior and social behavior deficits such as those seen in clinical populations diagnosed with autism. My research in this area has focused on three mouse models of autism specifically: the fragile X mental retardation 1 (*Fmr1*) null mouse, the Gly56Ala (*G56A*) transgenic mouse, and the K-Cl cotransporter 2 (*KCC2b*) mutant mouse. In the *Fmr1* mouse, I characterized the patterns of neuronal activation following the presentation of a social stimulus. I performed RNA sequencing in the prefrontal cortex and amygdala and compared *Fmr1* null mice to genetic controls and to mice that had been exposed to a novel non-social stimulus as a control for social exposure. This allowed me to identify genetic expression changes that were specific to social interaction in the prefrontal cortex and amygdala and to determine aberrant patterns of gene expression changes in the *Fmr1* null mice in the same social exposure. I also used RNA sequencing to explore the prefrontal cortex and amygdala gene expression changes in the *G56A* transgenic mouse, a mouse model of hyperserotonemia observed in autism due to the rare variant in the serotonin transporter gene *SLC6A4.* Additionally, I performed behavioral tasks to characterize two proposed autism mouse models, the KCC2b and the *Lurcher* mouse. In the *KCC2b* mouse I measured the social and anxiety behaviors and found that the *KCC2b* mutant mouse has disturbed social dominance behaviors. In the *Lurcher* mouse, I measured behavioral flexibility via a serial reversal learning task and found reduced behavioral flexibility in the *Lurcher* mouse as compared to controls. Autism is particularly noted for being associated with a broad spectrum of symptoms, genetic mutations, and differences in macro and micro anatomy of the nervous system. The behavioral characterization of mouse models of autism allows for the investigation of a diverse range of neurobiological alterations and behavioral symptoms identified in this neurodevelopmental disorder.

a. **Rogers, T.D.**, Anacker, A.M.J., Kerr, T.M., Forsberg, C.G., Wang, J., Zhang, B., & Veenstra-VanderWeele, J. (2017). Effects of a social stimulus on gene expression in a mouse model of fragile X syndrome. *Mol Autism, Jun 23;8:30*. PMID: 28649315
b. Anacker, A.M.J., Moran, J.T., Santarelli, S., Forsberg, C.G., **Rogers, T.D.**, Stanwood, G.D., Hall, B.J., Delpire, E., Veenstra-VanderWeele, J., & Saxe, M.D. (2019). Enhanced Social Dominance and Altered Neuronal Excitability in the Prefrontal Cortex of Male KCC2b Mutant Mice. *Autism Res, 12(5)*,732-743. PMID: 30977597
c. **Rogers, T.D.**, Forsberg, C.G., Veenstra-VanderWeele, J. (2014). Differences in neuronal activation and gene expression in the fragile X mouse. *International Meeting for Autism Research Abstracts*, 18132.
d. Dickson, P.E., **Rogers, T.D.**, Del Mar, N., Martin, L.A., Heck, D., Blaha, C.D., Goldowitz, D., & Mittleman, G. (2010). Behavioral flexibility in a mouse model of developmental cerebellar Purkinje cell loss. *Neurobiol Learn Mem, 94(2)*, 220-228. PMID 20566377

3. In more recent work, I have begun to explore the role of oxytocin in social behaviors in mice. I explored the effects of social isolation in juvenile C57BL/6J mice on social behaviors such as sociability and preference for social novelty as tested by the Three-Chamber Sociability Task. Subchronic pretreatment of i.p. oxytocin elicited sex-specific patterns of social discrimination between a familiar and novel social stimulus. Similarly, social isolation caused sex-specific patterns in social discrimination providing a three-way pretreatment by housing condition by sex interaction. I also measured stimulation-evoked mesolimbic dopamine release in mice having undergone the same social isolation and oxytocin pretreatment. In these results, we found that while oxytocin changed the levels of dopamine release, social isolations altered dopamine release levels in a sex-specific manner. These studies were completed in collaboration with Dr. Deranda Lester, the Co-PI of The University of Memphis. These results are currently in preparation to be submitted for publication.

a. Berry, K., Estes, M.K., Paige, N.B., Meadows, M., Lester, D.B., & **Rogers, T.D.** (2019). Effects of subchronic oxytocin treatment on social behavior following social isolation in juvenile mice. *Society for Neuroscience Abstracts*, 647.11.
b. Estes, M.K., Paige, N.B., Mills, M.N., **Rogers, T.D.**, & Lester, D.B. (2019). Systemic oxytocin treatment reverses the effect of social isolation on mesolimbic dopamine release. *Society for Neuroscience Abstracts*, 647.12.

Complete List of Published Work in MyBibliography:
https://www.ncbi.nlm.nih.gov/myncbi/tiffany.rogers.1/bibliography/public/.