BIOGRAPHICAL SKETCH

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NAME: **Travis D. Goode**

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

POSITION TITLE: Postdoctoral Fellow

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	Dates (MM/YYYY)	FIELD OF STUDY
University of Tennessee, Knoxville, TN	BA	08/2006 – 05/2011	Psychology
Texas A&M University, College Station, TX	PhD	08/2012 – 08/2018	Neuroscience
Harvard University / Massachusetts General Hospital, Boston, MA	Postdoctoral	08/2018 – present	Neuroscience

A. Personal Statement

I am a first-generation college graduate and scientist, with strong interests in the study of the psychological and neurobiological underpinnings of affective and cognitive disorders. I earned my bachelor's in Psychology at the University of Tennessee where I worked as an undergraduate research assistant and lab technician in the social stress and neuroscience laboratory of Dr. Matthew Cooper. There, I assisted in studies of the behavioral and brain consequences of social hierarchy in a rodent model of social defeat. I completed my PhD in Neuroscience in the emotion and memory systems laboratory of Dr. Stephen Maren at Texas A&M University, where I studied the brain systems and circuits that drive (or inhibit) learned fear responses to environmental threats. My area of excellence is in the rigorous study of aversively motivated behaviors. I have a broad skillset in the behavioral neurosciences, including adept implementation of animal models for the study of stress-related pathologies and in the expert use of numerous modern neuroscientific techniques, including behavioral pharmacology, functional tracing, opto- and chemogenetics, and *in vivo* electrophysiology. Currently, I am a postdoctoral fellow in the molecular neurogenesis and hippocampal circuits laboratory of Dr. Amar Sahay at Harvard University and Massachusetts General Hospital. My postdoctoral research is focused on the molecular and circuit contributions of the hippocampus to memory, with the aim of edifying new therapeutic targets for circumventing disorders of trauma, anxiety, and aging.

B. Positions and Honors

Relevant Employment

2010 – 2011 ***Tutor*** – Student Success Center, University of Tennessee
2011 ***Laboratory Technician*** – Department of Psychology, University of Tennessee
2011 – 2012 ***Pharmacy Technician*** – Kroger Pharmacy
2012 – 2018 ***Graduate Research Assistant*** – Institute for Neuroscience, Texas A&M University
2017 ***Lab Instructor*** – Elementary Statistics in Psychology (PSYC 203), Department of Psychological and Brain Sciences, Texas A&M University
2018 ***Lab Instructor*** – Elementary Statistics in Psychology (PSYC 301), Department of Psychological and Brain Sciences, Texas A&M University
2018 – ***Postdoctoral Fellow*** – Harvard University, Massachusetts General Hospital

Service Positions

2007 – 2008 ***Webmaster*** – National Society of College Scholars, University of Tennessee
2008 ***Student Volunteer*** – Australian Tropical Research Foundation, Cape Tribulation, Qld, AU
2009 ***Student Volunteer*** – Alternative Fall Break, Leadership & Service Center, University of Tennessee
2009 – 2010 ***Executive Vice President*** – National Society of Collegiate Scholars, University of Tennessee
2010 – 2011 ***Executive Vice President*** – Psi Chi Psychology Honor Society, University of Tennessee
2012 – 2018 ***Committee Member*** – Community Outreach & Symposium Planning Committee, Institute for Neuroscience, Texas A&M University
2013 – 2015 ***Delegate & Representative*** – Graduate & Professional Student Council, Texas A&M University
2014 – 2017 ***Organizer / Participant*** – "Brain Day" Event, Henderson Elementary School, Bryan, TX, Building Researchers And Innovators In Neuroscience and Society (BRAINS), Institute for Neuroscience, Texas A&M University
2015 ***Committee Member*** – Disciplinary Review Committee, Student Conduct Office, Texas A&M University
2015 – 2017 ***Panel Member*** – University Disciplinary Appeals Panel, Office of the Vice President for Student Affairs, Texas A&M University
2016 – 2018 ***Webmaster*** – Institute for Neuroscience, Texas A&M University
2017 – 2019 ***Seminar Co-Chair*** – Amygdala Function in Emotion, Cognition and Disease Gordon Research Seminar
2018 – ***Ad Hoc Reviewer*** – *Emotion and Cognition; Frontiers in Behavioral Neuroscience; Neuropsychopharmacology*

Honors and Awards

2006 – 2011 ***Tennessee HOPE Scholarship & ASPIRE Award*** – 6 cycles, $5500/cycle; Tennessee Student Assistance Corp.
2009 – 2010 ***Orange Scholars Program*** – 2 cycles, $2500/cycle; Homer Fund, The Home Depot, Inc.
2010 ***Summer Research Internship*** – Office of Undergraduate Research, University of Tennessee; $2000
2010 ***Social Science Research Excellence Award*** – $150; Undergraduate Research and Creative Achievement Symposium, University of Tennessee
2012 – 2015 ***Herman F. & Minnie Belle Heep Graduate Fellowship*** – $30000/year stipend; Institute for Neuroscience, Texas A&M University
2012 – 2017 ***Travel Award*** – 6 cycles, $1200/cycle; Institute for Neuroscience, Texas A&M University
2014 ***Honorable Mention*** – Graduate Research Fellowship Program, National Science Foundation
2015 ***Best Abstract Award*** – $100; Conference on Learning & Memory, University of Texas
2015 ***Aggies Commit Fellowship*** – $2000; Graduate & Professional Student Council, Texas A&M University
2016 ***Travel Award*** – $500; Association of Former Students, Texas A&M University
2016 ***Cover Image Design*** – *Neurobiology of Learning of Memory* (Volume 130)
2017 ***Close the Gap Fellowship*** – $1500; National Science Foundation / Graduate & Professional Studies at Texas A&M University
2017 ***Travel Award*** – $500; One Health Initiative, Texas A&M University
2017 ***Trainee Professional Development Award*** – $1000; Society for Neuroscience
2018 ***U.S. Senator Phil Gramm Doctoral Fellowship*** – $5000; Texas A&M University

C. Contributions to Science

NCBI Bibliography web address:
https://www.ncbi.nlm.nih.gov/myncbi/browse/collection/48347943/?sort=date&direction=

Contextual control of fear relapse. Fear-inhibiting extinction memories are slow to form and are susceptible to disruption. Relapse of fear is common in humans and other animals, but mitigating factors are limited. As such, we explored learning and memory mechanisms that contribute to the loss of successful reduction of fear. This work demonstrated not only that stressful and dangerous contexts can induce long-lasting relapse,

but also that safe contexts may help buffer against full-blown relapse. We also found that robust and augmented forms of extinction do not mitigate the possibility of relapse, further suggesting that fear extinction is a fear-inhibiting (not -erasing) process.

a. **Goode TD**, Maren S (2014) Animal models of fear relapse. ***ILAR Journal*** 55: 246-258.
b. **Goode TD**, Kim JJ, Maren S (2015) Relapse of extinguished fear after exposure to a dangerous context is mitigated by testing in a safe context. ***Learning & Memory*** 22: 170-178.
c. **Goode TD**, Holloway-Erickson CM, Maren S (2017) Extinction after fear memory reactivation fails to eliminate renewal in rats. ***Neurobiology of Learning and Memory*** 142: 41-47.

Neural circuits of relapse. Relapse of fear is thought to involve hippocampal-prefrontal interactions, but the function of this neural circuitry has only been indirectly tested. Thus, using a combination of electrophysiological recordings, intracranial pharmacology, and opto-/chemogenetics, we demonstrated that relapse of fear involves feed-forward inhibitory mechanisms of hippocampal projections to the infralimbic (IL) region of the prefrontal cortex, resulting in a net inhibition of IL and relapse. This work now identifies a common circuit mechanism by which fear relapses, serving as a potential therapeutic target.

a. Marek R*****, Jin J*****, **Goode TD***, Giustino TF, Wang Q, Acca GM, Holehonnur R, Ploski JE, Fitzgerald PJ, Lynagh TP, Lynch JW, Maren S, Sah P (2017) Hippocampus-driven feed-forward inhibition of the prefrontal cortex mediates relapse of extinguished fear. ***Nature Neuroscience*** 21: 384-392. ***** = equal contribution.
b. **Goode TD**, Jin J, Maren S (2018) Neural circuits for fear relapse. Pp 182-202, In ***Neurobiology of Abnormal Emotion & Motivated Behaviors*** (S Sangha, D Foti, Eds) San Diego: Elsevier.
c. **Goode TD**, Maren S (2018) Common neurocircuitry mediating drug and fear relapse in preclinical models. ***Psychopharmacology*** 236: 415-437.

Role of the bed nucleus of the stria terminalis (BNST) in aversive learning. The BNST makes important contributions to the learning and expression of conditioned fear, but the precise boundary conditions of its influence are poorly defined. Accordingly, we have proposed that its complex impacts on fear can be explained by situations in which the animal is uncertain of precise timing of an aversive event, which may have important implications for relapse and anxiety at large. We have tested this hypothesis through a number of experiments comparing imminent vs. ambiguous threats; this work has revealed subregion-specific activation of the BNST and its efferents during fear expression to ambiguous, as well as dissociable roles for the BNST in predictable vs. unpredictable training. This ongoing work should shed light on the understudied role of the BNST in associative learning.

a. **Goode TD**, Kim JJ, Maren S (2015) Reversible inactivation of the bed nucleus of the stria terminalis blocks reinstatement but not renewal of extinguished fear. ***eNeuro*** 2: ENEURO.0037-15.2015.
b. **Goode TD**, Maren S (2017) Role of the bed nucleus of the stria terminalis in aversive learning and memory. ***Learning & Memory*** 24: 480-491.
c. Luyck K, **Goode TD**, Masson HL, Luyten L (2018) Distinct activity patterns of the human bed nucleus of the stria terminalis and amygdala during fear learning. ***Neuropsychology Review*** 1-5.
d. **Goode TD**, Ressler R, Acca GM, Maren S (preprint) Bed nucleus of the stria terminalis mediates fear to ambiguous threat signals. ***BioRxiv*** 376228.

Amygdalar regulation of stress-sensitive behavior. Chronic stress can potentiate social avoidance, promote habitual learning, and impair extinction of fear—outcomes that are thought to rely on neuromodulatory signaling systems within the amygdala. However, these neural processes are still not well understood. Accordingly, I have made a number of contributions to our knowledge of the role of the amygdala in stress-dependent modulation of affective behaviors.

a. Dulka BN, Ford EC, Lee MA, Donnell NK, **Goode TD**, Prosser R, Cooper MA (2016) Proteolytic cleavage of proBDNF into mature BDNF in the basolateral amygdala is necessary for defeat-induced social avoidance. ***Learning & Memory*** 23: 156-160.
b. **Goode TD***, Leong K-C*, Goodman J, Maren S, Packard M (2016) Enhancement of striatum-dependent memory by conditioned fear is mediated by beta-adrenergic receptors in the basolateral amygdala. ***Neurobiology of Stress*** 3: 74-82. ***** = equal contribution.
c. Giustino TF, Seemann JR, Acca GM, **Goode TD**, Fitzgerald PJ, Maren S (2017) β-adrenoceptor blockade in the basolateral amygdala, but not the medial prefrontal cortex, rescues the immediate extinction deficit. ***Neuropsychopharmacology*** 42: 2537-2544.

D. Additional Information: Research Support

Completed Research Support

Neural Circuits for Reinstatement of Fear
F31 Predoctoral Ruth L. Kirschstein National Research Service Award (NRSA) (F31MH107113)
08/01/16 – 07/31/18
Role: PI ($66456 [Direct costs]); Sponsor: Stephen Maren; Co-Sponsor: Jun Wang
Impact Score: 19; Percentile: 4%
Project description: Relapse of fear is common to many forms of therapy for a variety of fear-related anxiety and trauma disorders (e.g., phobias, panic disorder, and post-traumatic stress disorder), yet the brain circuits that mediate and modulate the return of fear after aversive events are not well understood. Accordingly, this project seeks to isolate the functional role of fear-regulating brain circuits in the relapse of fear after dangerous (shock-associated) context exposure in rats, especially about interactions of the bed nucleus of the stria terminalis (BNST) with the amygdala and hypothalamus. BNST efferents may serve as important clinical targets in the future of pharmacological and therapeutic interventions during times of aversive experiences.