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Executive Summary

The university requires an external review of all graduate degree programs every 7 years. This will be the first review of the Neuroscience Interdisciplinary Graduate program and we have developed a comprehensive self-study that provides background information on the setting up of the program, data on the students and faculty, and an overall assessment of the program.

Texas A&M recognizes the value of interdisciplinary research and has been very supportive of the Interdisciplinary Graduate Program in Neuroscience. Neuroscience is an important and growing discipline in the Life Sciences that addresses critical societal problems from neurological diseases (Alzheimer’s, epilepsy, CNS injury, autism) to alcohol and drug addiction. The presence of a neuroscience program with significant stipends for graduate students is attractive in recruiting both outstanding faculty and graduate students.

The Neuroscience Ph.D. program was established in 2010 under the auspices of the Texas A&M Institute for Neuroscience. The faculty of neuroscience has grown from 68 to 93 members during this time period. The comprehensive nature of A&M provides highly varied research areas from computer science, basic cell and molecular neuroscience, psychology and translational research in veterinary and human medicine.

Since the inception of the Neuroscience graduate program, we have recruited 50 students, with stellar academic records. We have graduated 12 students with 9 placed in postdoctoral positions in academic institutions, one has returned to the medical school to complete his MD and will graduate with an MD/PhD and one has joined a biotechnology start up company. The students in the program have excellent credentials (average GPA = 3.64; GRE-Q = 157; GRE-V = 158 and are highly productive producing 12 papers/abstracts per year. 20% of the students are ethically diverse which is similar to other neuroscience programs although we have made efforts to improve our student diversity in recent years. The fact that our program is relatively small allows the students to form a cohesive and supportive group that is highly engaged in shared governance of the program.

By virtue of the fact that TAMIN emerged as a grass roots effort by the faculty, it remains a thriving Interdisciplinary Degree Program (IDP). From the perspective of the faculty, the program provides several venues that allow for faculty to congregate and share ideas and establish collaborations. The core faculty are highly successful and productive producing an average of 4.2 publications/year/faculty member and an average of $12.3 million per year in grant funding for the group. It is noteworthy that 14% of the NIH funding to the university over the years 2014, to 2016, is from TAMIN faculty according to analysis of the NIH Rainbow report. Neuroscience faculty members, that are going up for tenure and or promotion, are required to have a letter from the Chair of TAMIN and thus their contributions to the program are valued and considered in the tenure and promotion process.

TAMIN also supports the undergraduate program with a minor in Neuroscience. There has been substantial growth in the minor from 80 students in 2009, to over 291 in 2016. Students participating in the minor must complete 15 hours of credit, including inquire-based research within a Neuroscience laboratory. The undergraduate students are key participants in increasing our research output and are encouraged to present posters at the various neuroscience events. They are usually mentored by graduate students, which allows the graduate students to have valuable teaching experience.

In 2015 we submitted fifth and final annual progress report for Texas A&M University’s Ph.D. program in Neuroscience to the Texas Higher Education Coordinating board. Amongst their comments were: “Based on your submission, it appears that this program is off to a good start. The Neuroscience doctoral program has made good progress. The success of this interdisciplinary program could serve as a model for other programs,
enabling institutions to improve interdisciplinary research.” Appendix A includes THECB final report comments.

Notable achievements since the program was initiated:

- TAMIN faculty grants account for 14% of the total NIH funding to TAMU.
- The combined faculty external grants average $12.3 million/year.
- Increase in faculty membership from 68 to 93.
- 50 stellar graduates recruited to the program.
- 9/12 graduates placed in academic postdoctoral positions.
- Voted by graduate students as the best neuroscience Ph.D. program for two consecutive years.
- Successful recruitment of the top 7 applicants into the Fall 2017 class.
- Increase in undergraduate minors from 80 to 291.
Welcome

Message from the Texas A&M Institute for Neuroscience Chair

On behalf of the students, staff, faculty and administration of Texas A&M University we are happy to welcome you to our campus for this important task of reviewing our Interdisciplinary Graduate Degree Program in Neuroscience.

We have prepared a self-study report that provides a history of the university and how the program was established. We have also included specific details of the program from the application process through to post graduation job placements.

The review process provides us with an opportunity for self-reflection and also to learn from your experience and expertise in similar degree programs.

We recognize that this review represents a considerable time commitment and that you will have an intensive few days with us but we hope that it will be a rewarding experience for you all.

Please do not hesitate to contact us before or during your meeting for any additional information that you may require.

We are looking forward to seeing you at the review May 14-18th

C. Jane Welsh Ph.D.
Chair Texas A&M Institute for Neuroscience
TAMIN Interdisciplinary Life Sciences Bldg.
Texas A&M University
3473 TAMU | College Station, Texas 77843-3474
Tel: 979.862.4974 | Fax: 979.847.8981 | jwelsh@cvm.tamu.edu
Visit Texas A&M Institute for Neuroscience (TAMIN) website
http://tamin.tamu.edu/
Charge to the Peer Review Team

The Academic Program Review (APR) process at Texas A&M University provides the occasion for academic units to plan strategically, assess the quality and efficacy of their programs, and determine the best courses of action for ongoing improvement. APR is at the heart of our institutional commitment to excellence, and we sincerely thank you for assisting us. This letter provides you with the charge to the committee and a brief overview of the department.

Please examine the department and its programs and make recommendations that will help in planning improvements. Your resources are a self-study report prepared by the department, copies of materials from the program’s last review, information you gain through personal interactions while visiting Texas A&M University, copies of strategic plans and goal-setting documents at the department, college, and/or university level, and any additional information requested by you or by the department. Within the broad charge of recommending ways the department can continue to improve are some specific questions that we would like you to address:

- Based on the data / information provided in the self-study report or gathered by the review team, what are the department’s overall strengths and weaknesses?
- How well do the department’s strategic goals align with those of its college and with those of Texas A&M University?
- How would you compare this department with its peers?
- What improvements (including student learning and faculty development) has the department made since the previous program review?
- With only current resources or a modest infusion of new ones, what specific recommendations could improve the department’s performance, marginally or significantly?

We look forward to meeting with you during your time on campus. If you have any questions or require additional information prior to your visit, please contact Dr. C. Jane Welsh jwelsh@cvm.tamu.edu or Ms. Bettyann Zito, APR Program Coordinator, at apr@tamu.edu.
Overview of the Program
The Neuroscience Ph.D. program was established in 2010 under the auspices of the Texas A&M Institute for Neuroscience. The faculty of neuroscience has grown from 68 to 93 members during this time period. The faculty have expertise in a broad range of areas in Neuroscience including basic neurobiology, psychology, engineering, human and veterinary medicine.

Since the inception of the Neuroscience graduate program, we have recruited 50 students, with stellar academic records. We have graduated 12 students with 9 placed in postdoctoral positions in academic institutions, one has returned to the medical school to complete his MD and will graduate with an MD/PhD and two are in the biotechnology industry. Students take two core classes in Neuroscience along with statistics, ethics and seminar and then have a large choice of electives.

Prior to the approval of the Ph.D. in Neuroscience, trainees received their degree in their advisor’s home department. This continues to be a popular option, with approximately 70 neuroscience-associated students pursuing their degree in this manner. These students are welcome to participate in all our Neuroscience courses and events.

Many of our TAMIN faculty members serve on NIH study sections and advisory boards and are well positioned to inform the group as to NIH grant review trends. The established faculty members within TAMIN are serving as mentors for junior faculty, postdoctoral fellows and graduate students and are assisting them with professional development and advancement, through reviewing grants and networking.

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Texas A&M University

Texas A&M University Mission Statement
Texas A&M University is dedicated to the discovery, development, communication, and application of knowledge in a wide range of academic and professional fields. Its mission of providing the highest quality undergraduate and graduate programs is inseparable from its mission of developing new understandings through research and creativity. It prepares students to assume roles in leadership, responsibility and service to society. Texas A&M assumes as its historic trust the maintenance of freedom of inquiry and an intellectual environment nurturing the human mind and spirit. It welcomes and seeks to serve persons of all racial, ethnic and geographic groups as it addresses the needs of an increasingly diverse population and a global economy. In the 21st century, Texas A&M University seeks to assume a place of preeminence among public universities while respecting its history and traditions.

History of the University
Texas A&M is the state’s first public institution of higher education. With a student body of more than 59,000 and more than 5,200 acres on the College Station campus, Texas A&M is also among the nation’s largest universities. Texas A&M owes its existence to the Morrill Act, approved by the United States Congress on July 2, 1862. This act provided for donation of public land to the states for the purpose of funding higher education whose "leading object shall be, without excluding other scientific and classical studies, and including military tactics, to teach such branches of learning as are related to agriculture and mechanic arts."

The State of Texas agreed to create a college under the terms of the Morrill Act in November 1866, but the actual formation occurred with the establishment of the Agricultural and Mechanical College of Texas by the Texas state legislature on April 17, 1871. A commission created to locate the institution accepted the offer of 2,416 acres of land from the citizens of Brazos County in 1871, and instruction began in 1876. Admission was limited to white males, and, as required by the Morrill Act, all students were required to participate in military training.

Texas A&M underwent many changes in the 1960s under the presidency of Gen. James Earl Rudder. Under his tenure the college diversified, opening its doors to African-Americans and formally admitting women. Participation in the Corps of Cadets was also made voluntary. In 1963, the Texas state legislature officially renamed the school to Texas A&M University, with the "A" and "M" being a symbolic link to the school's past but no longer officially standing for "Agricultural and Mechanical."

Since that time, Texas A&M has flourished to become one of the nation’s premier research universities. Along with the University of Texas and Rice, Texas A&M is one of only three Tier 1 universities in the state. In 1971 and 1989, respectively, Texas A&M was designated as a sea-grant and a space-grant institution, making it among the first four universities to hold the triple distinction of land-grant, sea-grant, and space-grant designations.

The George Bush Presidential Library and Museum opened in 1997 on west campus, making Texas A&M one of only a few universities to host a presidential library on their campus. President Bush maintains an active role in the university, hosting and participating in special events organized through the library. “Adapted from Texas A&M website”
Interdisciplinary Programs (IDPs) at Texas A&M

The majority of graduate programs are administered through departments. As of 2017, Texas A&M offers: 131 Bachelors, 164 Masters, 89 Doctoral and 5 professional degrees. There are also a growing number of interdisciplinary programs at A&M.

The university rules state that “an IDP involves a group of faculty from more than one discipline representing single or multiple colleges, organized for the purpose of enhancing research and scholarly activities and overseeing graduate education for a degree program not offered at any existing academic department.” University rules for IDPs are in Appendix B and C.

The current IDPS are:

- Agribusiness (Master of Agribusiness) (MAB)
- Agribusiness and Managerial Economics (PhD)
- Biotechnology (Master of Biotechnology) (MBIOT)
- Ecology and Evolutionary Biology (PhD)
- Energy (MS)
- Genetics (MS & PhD)
- Marine Biology (MS & PhD)
- Molecular and Environmental Plant Sciences (MS & PhD)
- Neuroscience (MS & PhD)
- Toxicology (MS & PhD)
- Water Management and Hydrological Science (MS & PhD)
Introduction to the Program

Brief History of Neuroscience at A&M

The Faculty of Neuroscience at Texas A&M was created in 1992 and has remained a grass-roots, faculty driven enterprise ever since. The Neuroscience Interdisciplinary Research Program was formally recognized in 2001. The program now includes 93 faculty distributed across 10 colleges and 26 departments within Texas A&M University and the Texas A&M Health Science Center (TAMHSC). A complete TAMIN faculty list can be found in Appendix D. The minor in Neuroscience was introduced in 2007 and a Memorandum of Understanding was detailed between TAMU and TAMHSC (at that time these were separate institutions), laying the groundwork for a Ph.D. proposal that was approved by the Coordinating Board in April of 2009. In 2010 the Texas A&M Institute for Neuroscience (TAMIN) was established which houses the Neuroscience graduate program and the first students were recruited into the program in 2010. Since its inception, TAMIN has focused on building interdisciplinary research collaborations, to foster funding, scholarship, and student training at both the graduate and undergraduate level.

The Texas A&M Institute for Neuroscience (TAMIN) is a multidisciplinary program originally established between Texas A&M University (TAMU) and the Texas A&M Health Science Center (TAMHSC). The faculty of neuroscience at TAMU brings researchers together to discover cures for addiction, neurological diseases, sleep disorders, and to reduce suffering from pain and promote recovery after neural injury. Neuroscientists understand that attacking these problems requires a multidisciplinary approach that couples work at the molecular level, to studies of neuron function and the immune system, and how neural-based effectors are organized into coherent systems that underlie our capacity to think, feel, and act. The hope is to translate new laboratory discoveries to effective clinical treatments, and this requires expertise drawn from nearly every college within TAMU. TAMIN provide an organizational structure that strengthens interdisciplinary and collaborative research, enhances scholarship and research funding, promotes national visibility, promotes postdoctoral and graduate training, and offers undergraduates unique training opportunities within the area of Neuroscience.

Neuroscience is the most rapidly developing field of intellectual inquiry today. This is evident both through the numbers of members in its national organization (the Society for Neuroscience, with nearly 40,000 members in 2014) and established training programs at all tier-1 universities. The central focus is the study of the nervous system, from the genetic/molecular/cellular basis to the organization of neural circuits, and the manifestation of this biological/ neurochemical machinery as behavioral, physiological and psychological processes. It aims to detail both how the normal system operates and how alterations in function contribute to clinical diseases, such as mental illness, dementia, developmental disorders, neurodegenerative diseases, chronic pain, drug addiction, and the loss of function with aging or neural injury. These health issues are a central focus of funding initiatives at the National Institutes of Health.

The National Institutes of Health devotes a large proportion of its intramural and extramural funding to the study of the nervous system. The specific institutes involved are: the National Institute of Mental Health (NIMH), the National Institute of Neurological Disorders and Stroke (NINDS), the National Institute on Drug Abuse (NIDA), National Eye Institute (NEI) and the National Institute on Aging (NIA). Other institutes also focus on the role of the nervous system in other disease processes such as the National Institute on Alcohol Abuse and Alcoholism (NIAAA) and the National Institute of Child Health and Human Development (NICHD). The USDA has a very important focus on investigation of the behavior of domestic farm animals with emphasis on the quality of life for livestock.
Initial University Multidisciplinary Research Initiatives (IUMRI) Funding

The goal of Vision 2020 is to elevate Texas A&M University into the ranks of the top ten U.S. public research universities by the year 2020. In line with this vision, in 2008 Provost Jeff Vitter called for white papers, as a part of the Academic Master Plan. The TAMIN proposal (Appendix E) was selected as one of eight white papers (out of 111 submitted from across the university) to be funded under the Initial University Multidisciplinary Research Initiatives (IUMRI) program in April of 2010. The final budget provided funding for three senior hires (62.5% of the net budget), one each in Veterinary Integrative Biosciences (VIBS), Psychology, and Biology (Appendix F). Additional, funding was provided to initiate the graduate program (29.8% of the budget), maintain the colloquia/symposia series (1.8%) and administrative support (5.9%).

Senior Hires

Joe Kornegay, DVM, PhD the senior hire in VIBS. For the past 30 years, has studied a spontaneous canine disease termed golden retriever muscular dystrophy (GRMD), which serves as an animal model for Duchenne muscular dystrophy (DMD) of humans. Both DMD and GRMD are X-linked, occurring due to mutations in the dystrophin gene. Boys with DMD are typically confined to wheelchairs by their early teens and die before their twentieth birthdays due to cardiopulmonary complications. Unfortunately, no treatment reverses the horrific course of DMD. Dr. Kornegay’s initial studies of the genomic and pathophysiologic features of GRMD, together with those of Dr. Barry Cooper’s group at Cornell, established this condition as a valid model of DMD. In recent years, Dr. Kornegay’s laboratory has studied various treatments (cell, molecular, and pharmacologic approaches) in affected dogs. Results of these preclinical studies should guide use of similar treatment strategies in DMD patients. They are also interested in mechanistic studies to better define certain features of the disease, including the genomic basis for phenotypic variation and reasons underlying paradoxical hypertrophy of some muscles. Dr. Kornegay collaborates extensively with TAMIN and other TAMU faculty in both preclinical and mechanistic studies. As an example, imaging available through the College of Veterinary Medicine, to include both specialized forms of ultrasound and magnetic resonance imaging (MRI), serves as an important biomarker to determine therapeutic benefit. Speaking mechanistically, they already have an ongoing collaboration with Dr. Candice Brinkmeyer-Langford from the genetics group to define the genomic basis of phenotypic variation and are excited about possibilities to collaborate on the physiologic basis of cell physiologic changes that may underlie muscle hypertrophy and also changes. Opportunities also exist to define changes at the basal lamina and neuromuscular junction in association with both initial necrosis and regeneration/reinnervation. Importantly, their colony of affected dogs literally serves as a “national resource,” as they have many subcontracts with companies and other universities. These studies have been supported principally by the National Institutes of Health (NIH) and the Muscular Dystrophy Association (MDA).

Steve Maren, Ph.D. the senior hire in the Department of Psychology and TAMIN whose laboratory is also in ILSB, builds upon existing strengths in the neurobiology of learning and memory. Research in his laboratory seeks to understand the brain circuits and cellular mechanisms underlying the encoding, storage, retrieval, and extinction of aversive memories, and how dysfunction in these circuits and processes contributes to anxiety disorders. Specifically, his lab focuses on the neurobiology of fear conditioning and extinction in rats and mice. The hippocampus, amygdala, and prefrontal cortex, a triad of interconnected brain areas with essential roles in memory and emotion, are critical for these processes. In particular, his laboratory uses both behavioral and systems neuroscience methods to understand the brain mechanisms of fear and anxiety. These approaches include reversible brain lesions, intracranial pharmacology, electrophysiology, and immunohistochemistry and optogenetics to map the activity of individual neurons in behaving animals. His work focuses on memories of emotional events, and how these memories come to be regulated by the context in which they occur. This work is highly relevant to understanding the etiology and treatment of a variety of anxiety disorders, including post-traumatic stress disorder, and has been supported continuously by the NIH. Anxiety disorders are among the most prevalent psychiatric illnesses in the world, affecting nearly one-third of the population. His arrival opens the door for many collaborative opportunities with existing faculty and the possibility of moving forward...
with a program project grant in the behavioral and cellular neuroscience might soon be realized with the large number of neuroscientists on campus studying memory processes and neuroplasticity.

**Wesley Thompson, Ph.D.,** the senior hire in Biology. Dr. Thompson investigates the neuromuscular junction (NMJ) specifically, the role of the glial cells that are present at this synapse. The lab images Schwann cells and axons in living mice to determine the relationships between axons and Schwann cells at normal NMJs as well as during reinnervation and sprouting. The group uses transgenic mice in which green fluorescent protein (GFP) is expressed in Schwann cells and mice that express cyan fluorescent protein (CFP) in axons. Thus the lab is able to stain the acetylcholine receptors with small concentrations of a snake toxin, bungarotoxin, that is conjugated to a red fluorochrome, rhodamine and observe green Schwann cells, blue axons and axon terminals, and red acetylcholine receptors. Moreover, each site bears a “fingerprint” that one can easily use to identify this same synaptic site hours, days, weeks, months, or even years later. Thus, it is possible to identify the synaptic components at individual synapses and see how they change with time. They are investigating how motor neurons regenerate and sprout in the muscle in response to nerve injury. In this way, they are learning exactly the relationships between axons and their glial cells as synapses reform. They are also examining and manipulating the molecules involved in this relationship between glia and nerve terminals.

**Neuroscience within the Colleges**

**Neuroscience within the College of Liberal Arts**

Eighteen members of TAMIN are housed within the Department of Psychology (College of Liberal Arts), with the majority affiliated with the Behavioral and Cellular Neuroscience (9) and Cognition & Cognitive Neuroscience (7) programs. Areas of research include drug addiction, affective neuroscience, cultural neuroscience, learning, memory, executive function, decision-making, language, depression, anxiety, and pain processing. Researchers are exploring how these phenomena are affected by stress, development, hormones, neural injury, sociocultural contexts, and aging, from the underlying cellular processes to neural systems and brain imaging. Faculty within Psychology are supported by grants from NIH (NIDA, NINDS, NIMH) and private foundations (e.g., McKnight, Neilson, Brain & Behavior Research Foundation).

A third (4/12) of the students that received their Ph.D. in Neuroscience worked with faculty within the Department of Psychology. Currently, Psychology faculty are supervising 14 graduate students in the TAMIN program. Psychology also contributes to graduate education through its support of six graduate courses that are cross-listed with Neuroscience (NRSC) and the co-teaching of Principles of Neuroscience II. Psychology faculty support the undergraduate program in Neuroscience by offering eight NRSC cross-listed courses and through the supervision of undergraduate independent study students and research scholars who work closely with our graduate trainees.

**Neuroscience within the College of Medicine**

The College of Medicine has 27 principal investigators that are members of the Texas A&M Institute for Neuroscience, drawn from the Department of Neuroscience and Experimental Therapeutics (19), Molecular and Cellular Medicine (5), Molecular Pathogenesis and Immunology (2) and Medical Humanities (1). Program strengths within the college include brain development, cellular/molecular basis of drug addiction, circadian biology, ocular pharmacology and experimental therapeutics, neurobiology of aging, neurologic diseases such as epilepsy, stroke, Parkinson’s disease, neuro-oncology and neurotoxicology of alcohol, nicotine and other drugs of abuse. Faculty within the department are also affiliated with university-wide interdisciplinary faculties including the TAMU Faculty of Neuroscience, Reproductive Faculty Forum, Faculty of Toxicology and our clinical science partner, the Texas Brain and Spine Institute (http://www.txbsi.com). The Neuroscience and Experimental Therapeutics department (NExT) is also home to the Women’s Health in Neuroscience Program, consisting of interdisciplinary research faculty and a clinical advisory group aimed at developing a cohesive
preclinical approach to the impact of puberty, pregnancy and menopause on brain development, mental health and brain disease.

Faculty in the College of Medicine are supported by grants from the NIH (NIA, NINDS, NIAAA), private foundations (Neilson, American Heart Association), Department of Defense and the VA. Faculty serve on grant review panels for the NIH, AHA, and NIH advisory committees. The College of Medicine was home to the first TAMIN graduates and continues to support the training of TAMIN students through course work and rotations. Currently, 5 TAMIN graduate students are at various stages of completing their thesis in College of Medicine labs. The College also participates in the Texas Alzheimer’s Research and Care Consortium (TARCC) (http://www.txalzresearch.org/). Faculty from the Medical School participate in teaching in Principles in Neuroscience I & II and teach a graduate level course in Neuropsychopharmacology.

**Neuroscience within the College of Science**

The College of Science has 13 principle investigators that hold membership in the Texas A&M Institute for Neuroscience. All of these faculty members hold appointments in the Department of Biology. Along with the Interdisciplinary Program in Neuroscience, these Biology researchers’ interests and activities span other cross-disciplinary fields, including molecular and cell biology, genetics, developmental biology and ecology and evolutionary biology. Five neuroscience researchers are also members of the Texas A&M Center for Biology Clocks Research. Neuroscience research funding within Biology has been garnered from NIH, NSF and multiple private foundations, including HHMI.

Areas of neuroscience research within Science (Biology) focus on the fundamental cellular, molecular, developmental, and systems biology of how nervous systems function to mediate animal behavior, including the evolution of those mechanisms. Biology faculty engage in cutting edge basic research in the genetic control of behaviors, the neurobiology of circadian rhythms and the molecular biology of biological clocks, the development in neural networks and signaling systems, the mechanisms regulating synaptic transmission and synapse function, the development of animal nervous systems, the neurobiology of sensory-motor integration, the molecular determinants of formation, maintenance and regeneration of neural circuits and neuromuscular junctions, the comparative genomics of animal behaviors, and the evolution and ecology of animal communication systems.

Biology neuroscience faculty members make major contributions to undergraduate and graduate education through their participation in the teaching of 9 courses cross-listed with Neuroscience (NRSC). These courses include teaching and learning of principles of neuroscience, regulatory and behavioral neuroscience, signaling in behavior and development, comparative neurobiology, biological clocks, and genes and evolution.

**Neuroscience within the College of Veterinary Medicine and Biomedical Science**

The College of Veterinary Medicine & Biomedical Sciences (CVMBS) currently includes 17 faculty members within the Texas A&M Institute for Neuroscience. The measure of cross-disciplinary research involving neuroscience faculty within CVMBS is evident by their interaction with other programs including: genetics, engineering, psychology, animal science, biology, and medicine. The College provides a unique insight for NRSC graduate students into clinical translational research.

Focus areas of research within CVMBS closely align with the most critical human neurological diseases, including brain tumors, epilepsy, multiple sclerosis (and neurotropic viral infections), diseases of the eye, muscular dystrophy, and traumatic spinal cord injury. Our faculty also engage in cutting edge basic science research in areas such as the intracellular signaling network at both post-transcriptional and post-translational levels, ion channel properties and function, synaptic plasticity, neuroimmunology, glial biology, myelination and interactions between neurons and glia.
Faculty members serve on editorial boards, grant review panels, hold offices in professional organizations, and have active or pending patents. Over and above research, CVMBS neuroscience faculty make major contributions to undergraduate, DVM, and graduate student teaching; train residents in neurology, anatomic and clinical pathology, radiology, and surgery; participate in numerous interdisciplinary rounds and journal clubs; and engage in community outreach. We have also actively contributed to programs intended to enhance diversity through contributions to TAMU’s application to the NIH Building Infrastructure Leading to Diversity (BUILD) Initiative (U54) and CVM Graduate Diversity Fellowship program. Additional educational programs directed at underprivileged K-12 schools and the Alzheimer’s and Multiple Sclerosis patients and careers, ensure that our research reaches the community in the spirit of NIH’s CTSA initiative (https://ncats.nih.gov/ctsa/community).
Mission and Goals

Mission
The mission of the Neuroscience program is to provide first class graduate education in Neuroscience. Our graduates will have cutting edge research experiences, strong publication records and be able to communicate effectively to both professionals and the public.

Neuroscience is a rapidly growing and diverse academic discipline that will significantly influence many aspects of our society over the next century through its impacts on human health, behavior, and emerging technologies in computer science and engineering. The interdisciplinary graduate program in Neuroscience at Texas A&M prepares students to meet these societal needs by providing a comprehensive training that spans these broad disciplines by bringing together faculty, staff and students from across many colleges and departments. The program provides formal training, research opportunities and public exposure for students seeking careers in basic, translational and clinical neuroscience research, teaching and industry. Students completing the Doctor of Philosophy in Neuroscience are prepared for teaching/research positions within academia and research positions in the private sector. A Masters of Science is also available for those seeking non-academic positions. The degrees were jointly conferred by TAMU and TAMHSC.

The main function of the Neuroscience Program is to administer the Doctor of Philosophy and Master of Science degree in Neuroscience according to the rules of the Office of Graduate and Professional Studies of Texas A&M University. The program also aims to develop professional development of the graduate students and to foster a collaborative environment for students, faculty and postdoctoral fellows through seminars and symposia and informal meetings.

The Goal of the Neuroscience Graduate Program
The long-term goal, given continued support from the university administration, is to become one of the top 10 Neuroscience programs in the nation, supported by an NIH funded training grant. The short-term goal of the Neuroscience Graduate Program is to establish continued support for the program and the graduate student stipends.

Specific Current Goals
1) Prepare students to succeed as independent scholars and scientists.
Coursework, formal training and research opportunities are coordinated to ensure Ph.D. students master fundamental concepts and have the academic skills to accurately evaluate and respond to current and future trends in the field of neuroscience.
2) Propel students towards a productive research career.
To be competitive in today’s market, Ph.D. students in Neuroscience must be highly involved in state of the art research, demonstrate their research productivity through peer-reviewed journal publications, and be able to effectively communicate their findings to both their peers and the public.
3) Place graduates in postdoctoral positions.
A Ph.D. in Neuroscience is typically followed by 4-6 years of postdoctoral training in a specialty area. To place students in the best position to succeed, the program must ensure that students progress through the training program in a timely manner and graduate within a competitive time frame. All graduates should be able to secure positions in top-tier postdoctoral training programs or employment in the private sector of their choice.
4) Enhance diversity in the discipline.
The long-term success of the program and its students will be promoted by the inclusion of a broad range of ideas and experiences. To achieve this we will strive to enhance diversity by encouraging the recruitment and retention of new students from population groups that have been historically under-served and/or under-represented in doctoral training.
Administrative Structure

TAMIN is unique within Texas A&M University as it is an Institute and also an Interdisciplinary Degree program. Over the years, the reporting structure has varied but currently the executive committee reports to a group of departmental heads (Biology, VIBS, NExT and Psychology) and to the Dean of the college in which the current TAMIN chair is affiliated with (currently the Dean of the College of Veterinary Medicine). All Interdisciplinary Programs within Texas A&M report to Dr. Michael Benedik the Vice Provost.

![Diagram of TAMIN Administrative Structure]

Figure 1. Administrative Structure of TAMIN within the University

In order to become a member of the Neuroscience Faculty, the applicant is required to send their CV and a letter of intent to the Membership committee chair. The membership committee vote and then the package is sent for EC approval.

Full members of the neuroscience faculty are entitled to:
1) Serve as a chair for an MS/PhD student in Neuroscience.
2) Teach a neuroscience course.
3) Stand for election for positions on the Executive committee or committees within NRSC.
4) Vote on issues arising at the business meeting.
5) Host a seminar speaker.
The executive committee of TAMIN consists of the chair and vice chair, and the chair of each of the 6 committees (Undergraduate, Graduate, Recruiting, Seminar, Membership and Finance). The graduate student representative and the President of the local chapter of the Society for Neuroscience, serve as ex-officio members. All the committee positions within TAMIN are elected by the faculty on an annual basis with positions filled for a two-year tenure. The graduate student representative is elected by the graduate students and the president of the local SfN chapter is elected by the chapter membership. The membership committee facilitates the elections and ensures a broad representation of nominations across the university. Appendix G and H includes the TAMIN policies and the duties of the TAMIN executive committee.

Figure 2. Structure of TAMIN Executive Committee
Texas A&M Institute for Neuroscience Current Committees
FY 2016-2017

Chair: Jane Welsh (9/15-8/17)
Vice Chair: Farida Sohrabji, NEXT (9/16-8/18)

Program Coordinator: Sylvia M. Bernal Jones (11/10)

GRADUATE PROGRAM COMMITTEE
Co-Chair (TAMU) – Mike Smotherman, BIOL (9/15-8/17)
Co-Chair (HSC) – Jun Wang, NEXT (9/16-8/18)
Member 1- Rachel Smith, PSYC (9/15-8/17)
Member 2- Gladys Ko, VIBS (9/16-8/18)
Member 3- Wes Thompson, BIOL (9/15-8/17)
Member 4- Steve Maren, PSYC (9/16-8/18)

MEMBERSHIP COMMITTEE
Chair- Rajesh Miranda, NEXT (9/16-8/18)
Member 1- Shoshy Eitan, PSYC (9/15-8/17)
Member 2- Louis Abbott, VIBS (9/16-8/18)
Member 3- Jianrong Li, VIBS (9/15-8/17)
Member 4- Laura Smith, NEXT (9/16-8/18)

GRADUATE RECRUITING COMMITTEE
Chair- Gregg Wells, MCM (9/16-8/18)
Member 1- Scott Dindot, VTPB (9/15-8/17)
Member 2- Michelle Hook, NEXT (9/16-8/18)
Member 3- Ursula Winzer-Serhan, NEXT (9/15-8/17)
Member 4- Yani Dawson Mathur, PSYC (9/16-8/18)
Member 5- Jun Wang, NEXT (9/16-8/18)

SEMINAR COMMITTEE
Chair- Ursula Winzer-Serhan, NEXT (9/15-8/17)
Vice-Chair - Paul Wellman, PSYC (9/16-8/18)
Member 1- Ginger E. Carney, BIOL (9/15-8/17)
Member 2- Mary Meagher, PSYC (9/15-8/17)

UNDERGRADUATE PROGRAM COMMITTEE
Chair- Jim Grau, PSYC (9/15-8/17)
Member 1- Shoshy Eitan, PSYC (9/15-8/17)
Member 2- Gladys Ko, VIBS (9/16-8/18)
Member 3- Mendell Rimer, NEXT (9/15-8/17)
Member 4- Joseph Orr, PSYC (9/16-8/18)

FINANCE COMMITTEE
Chair- Mark Zoran, BIOL (9/16-8/18)
Member 1- Jim Grau, PSYC (9/15-8/17)
Member 2- Erin Scott, (VSCS) (9/16-8/18)
Member 3- Mark Harlow, BIOL (9/15-8/17)

Graduate Student Representative: Ian Smith, BIOL (09/16-08/17)

Society for Neuroscience -SfN Chapter President: Mark Harlow, BIOL

SfN Grad Student Rep: Robert Louis Hastings, BIOL
(term of office is in parentheses)
Department and Program Resources
The program has a full-time program coordinator, Sylvia Bernal Jones, who was recruited in 2010 when the program began and she reports to the Chair of TAMIN. The program coordinator is included in all the meetings and decision-making processes. She is responsible for all the day-to-day running the program, accounting and advising both the graduate and undergraduate students.

Facilities
The program has a small office (approximately 146 square feet) in the ILSB building equipped with computer, printer and scanner. There is a neuroscience presence on the 3rd floor of the ILSB building with several faculty members and their laboratories located there and we make use of the ILSB lecture theater and meeting rooms.

University Resources - Relevant to Neuroscience

Center for Biological Clocks Research
The CBCR provides an organizational structure to enhance and coordinate research and education activities among circadian rhythms researchers at Texas A&M University, the Texas A&M University System Health Science Center.

Center for Chemical Characterization and Analysis
This is a component of the Department of Chemistry and a research support facility partially funded by the Office of the Vice President for Research and Associate Provost for Graduate Studies. The Center provides state-of-the-art capabilities for organic and inorganic analysis and structural characterization. Four specialized laboratories each address a specific area of interest. The areas and laboratories include:

The Elemental Analysis Laboratory provides research support in the area of elemental and trace analysis, as well as service analyses to TAMU users, other university and government agencies, and private industry. It is unique in that it features fast neutron activation analysis (FNAA) capabilities in addition to thermal instrumental neutron activation analysis (INAA) using the University's Nuclear Science Center 1 MW TRIGA research reactor. Furthermore, the laboratory has recently added inductively-coupled plasma – mass spectrometry to its facilities. The ICP-MS has been fitted with conventional sample introduction hardware for solution work as well as a 213 nm laser ablation system for studying solids and surfaces. The laboratory is extensively used to benefit a wide variety of research programs, reporting some 50,000 measurements completed each operational year.

The X-ray Crystallography and Molecular Structure Laboratory, a full service X-ray Diffraction laboratory offering state of the art instrumentation for the analysis of solid materials. Services include single-crystal, powder diffraction and small angle x-ray scattering analysis for Chemistry, Material Sciences and Pharmaceuticals. The laboratory is staffed by fully trained Ph.D. scientists who employ the most current diffraction and x-ray techniques.

The Nuclear Magnetic Resonance Laboratory provides NMR services, as well as X-Ray Crystallography, Mass Spectrometry, and Neutron Activation Analysis. The NMR Facility includes 9 superconducting spectrometer systems and 3 full time staff to support them with maintenance, user training, and spectroscopic service. Although this facility is physically housed within the Chemistry Department, it provides services to the entire campus community.

The Laboratory for Biological Mass Spectrometry (LBMS) provides expertise in mass spectrometry to all TAMU research activities. This includes the analysis of organic compounds ranging from small molecule to large biological molecules including proteins, DNA, RNA, and natural products. To accomplish the research objectives, the LBMS maintains a complete inventory of routine and cutting-edge mass
spectrometers. The LBMS research scientists are actively involved in the development of new analysis methods and techniques and the development of next-generation instrumentation for analysis and sample handling, including robotics and microfluidics. The activities within the LBMS are divided into three categories: (i) core research, (ii) applications, and (iii) training and dissemination.

**Center for Microencapsulation and Drug Delivery**
The CMDD is a small, highly skilled multidisciplinary group of researchers focused on the application of controlled release to modulate immune responses.

**Center for Translational and Environmental Health Research (CTEHR)**
The newly funded NIEHS Center for Translational and Environmental Health Research at Texas A&M University currently provides funding to faculty who are members for discounted use of several university supported core facilities, including the Quantitative Biology Core, the Advanced Imaging Core, the Integrated Health Sciences Core, and the Targeted Genomics Core. These Cores provide the following services: RNA/DNA Quality, microRNA Detection, DNA Size Selection with Pippen Prep, Real Time PCR, Sequencing Library Prep, Cellular Bioenergetics, Statistical Analyses, Integromic and Higher Level Computational Analyses, Transmission Electron Microscopy, Optical Microscopy/Digital Imaging/Analysis, Live Cell Imaging, High Throughput Automated Fluorescence Microscopy and Image Analysis, Multiplexing and Quantitative Readout, Image Processing, Analysis, Quantification, Super Resolution Microscopy, Virtual Repository and Protocol Development, Metagenomics and Metatranscriptomics, Gnotobiotic Mice, Metabolic Phenotyping, Basic Pronuclear Injection, Pronuclear Injection of CRISPR/CAS9 and TALEN, Blastocyst Injection/Chimera Production, Sperm Cryopreservation, Embryo Cryopreservation, Rederivation via IVF, Embryo Transfer, Colony Expansion/Maintenance, Gene Targeting via Homologous Recombination (Constitutive or Conditional), ES cell Electroporation and Screening, Knockout Mice from Repository, and Gene Trapped ES cells from Repository.

**Comparative Medicine Program**
This is a centrally administered support service for animal research and teaching programs at Texas A&M University, College Station. The facilities and services are available for all Texas A&M University, College Station campus affiliated faculty, staff, and students who have been approved to conduct animal research by the Institutional Animal Care and Use Committee (TAMU IACUC). CMP is accredited by the Association for the Assessment and Accreditation of Laboratory Animal Care, International (AAALAC, International) through its affiliation with other AAALAC-accredited TAMU programs. Facilities offer housing and care for most standard laboratory animals, and specialized housing can be provided for biohazard projects (ABSL I-III) and hazardous chemical projects with varying degrees of animal isolation available. Housing for large animal species is limited; however, various other campus animal care facilities can provide housing for large animals. Furthermore, a variety of services are provided to institutional personnel, including the procurement of animals and animal husbandry supplies, provision of veterinary care, use of CMP surgical facilities, a polyclonal antibody production service, technical support services, and animal use training services.

**Core for Integrated Microbiota Research**
The Core for Integrated Microbiota Research (CIMR) is an inter-collegial resource for investigators performing or planning to integrate microbiota research into their current research programs. The CIMR provides a new-to-campus infrastructure for germ-free and gnotobiotic rodent studies, germ-free line derivation, microbiota specimen collection, and metagenomic and metabolomic analyses. The goal of the CIMR is to facilitate new and innovative research that empowers investigators to remain highly competitive for extramural federal and foundation grants. To this end, the CIMR integrates facilities and expertise of 3 core components — gnotobiotics, metabolomics and metagenomics, to provide users a seamless, comprehensive workflow for the complete analysis of the microbiota.
DNA Technologies Core Laboratory
The DNA Technologies Core Laboratory in the College of Veterinary Medicine, provides automated nucleotide sequencing, genotyping, and bacterial and fungal DNA sequence-based identification technologies. It also offers individual identification, parentage testing, and genetic trait testing for livestock producers, wildlife managers, and clinical microbiology labs worldwide. The lab houses shared capital equipment used by researchers and students throughout the college.

Flow Cytometry Core Laboratories

Flow Cytometry Core in the College of Medicine
Principle Investigators utilizing this core facility have two analytical instruments for their flow cytometry analyses. Both deliver quantitative data that identifies and characterizes individual cells by labeling cell surface proteins providing immunophenotypic and lineage data as well as intracellular (cytoplasmic) protein expression in diverse cell types. The core performs several functional assays such as apoptosis, mitochondrial membrane potential, intercellular pH, calcium flux (signal transduction), and cell cycle kinetics. The first instrument is a two laser (20mW Blue/488nm & 25mW Red/635nm), seven parameter (5-color) Beckman-Coulter FC500 bench top analyzer with a dedicated operator or with training may be used individually. The second is a three laser (200mW Blue/488nm, 100mW UV/351nm & 25mW Red/633nm) eleven parameter (9-color) Beckman-Coulter MoFlo XDP fluorescence activated cell sorter. This instrument is capable of steriley separating and depositing up to four purified subsets of cells into tubes based on any of the eleven parameters at a rate of about 25,000 cells/second.

Flow Cytometry Core Laboratory in the College of Veterinary Medicine
The Flow Cytometry Core Laboratory, equipped with a Beckman Coulter MoFlo® Astrios™ High-Speed Cell Sorter and a Becton Dickinson FACSCalibur™ Analyzer, serves scientists in the department, college, and university.

Histology Laboratory
The histology laboratory in the College of Veterinary Medicine performs routine histologological techniques (paraffin processing and sectioning, frozen sections, H&E staining) and the staff work with researchers to develop special stains for specific research projects.

Image Analysis Laboratory
Established in 1987 to serve microscopy and imaging needs of the investigators in the College of Veterinary Medicine & Biomedical Sciences (CVM), the laboratory has expanded to serve the Texas A&M University System and currently serves as an Advanced Imaging Facility Core for the interdisciplinary NIEHS-supported Center for Translational Environmental Health Research (CTEHR). The center, a collaboration between Baylor College of Medicine, Texas A&M University and the University of Houston, is one of only 21 Centers of Excellence in the country and will focus on better understanding the effects of the environment on human health. The core supports the goal of the Center to “improve human environmental health by integrating advances in basic, biomedical and engineering research across translational boundaries from the laboratory to the clinic and to the community and back.” The laboratory also provides core support to the Center for Organ and Cell Biotechnology, a collaboration between the Texas Heart Institute (THI) and CVM with objectives to develop, test, and commercialize disruptive cell & organ biotechnologies and molecular tools and to build the medicines of tomorrow.

Fluorescence imaging of a range of samples - widefield, deconvolution, laser scanning confocal, multiphoton, TIRF, super resolution. Live-cell imaging capabilities FRAP, FLIP, Photoactivation/conversion FRET, FLIM. Image processing, analysis, quantification and statistical analysis using SAS, Minitab, Prism. Additional algorithms may
be developed using Labview and/or Matlab Transmission electron microscopy. Individual training, short courses and formal courses that can be taken for credit are also offered.

**Microscopy and Imaging Center**
The Microscopy and Imaging Center (MIC) is supported by the Vice President for Research and serves a wide range of faculty and students at Texas A&M University, in addition to researchers from outside of the University. The mission is to provide current and emerging technologies for teaching and research involving microscopy and imaging in Life and Physical Sciences on the Texas A&M campus and beyond, training and support services for electron microscopy, sample preparation, in situ elemental/molecular analyses, as well as digital image analysis and processing. An affiliated Materials Characterization Facility provides access to additional instrumentation and expertise. The MIC promotes cutting edge research in basic and applied sciences through Research and Development activities and quality training programs through individual training and short courses.

**Protein Chemistry Laboratory**
The Protein Chemistry Laboratory is a core resource facility created and funded under the auspices of the Office of the Vice President for Research. The laboratory has been established to support research in protein chemistry and molecular biology in the Texas A&M University System and to provide state-of-the-art instrumentation and technical expertise for the application of modern molecular biological technologies. The PCL is overseen by a committee that meets regularly to discuss issues relevant to the facility's operation. The laboratory operates as a fee-for-service facility and accepts samples on a first-come-first-served basis from faculty, scientists and students of Texas A&M, other educational institutions and industrial scientists. Main campus users are given preference whenever possible as the facility exists primarily to support Texas A&M research.

**Texas A&M Institute for Genome Sciences and Society (TIGSS)**
TIGSS is founded on principles first laid out in the white paper proposal for the Whole Systems Genomics Initiative. Participation in TIGSS is open to all faculty, staff and students. Contributions in genetics and genomics by these world-renowned scientists have advanced agricultural productivity, human and animal health, and have influenced economics, policy, ethics, geography and business. TIGSS functions as a virtual institute to unite genome scientists with researchers who study the social, economic, and ethical consequences and impacts of genomics technology, as well as bioinformatics scientists who conduct research on how to analyze and manage large datasets such as those generated by high-throughput genomics experiments. The group has a molecular genomics workspace in the College of Veterinary Medicine and a behavioral phenotyping core located in the main comparative medicine building ([https://genomics.tamu.edu/molecular-genomics-workspace](https://genomics.tamu.edu/molecular-genomics-workspace)).

**Texas A&M Institute for Genomic Medicine (TIGM)**
TIGM is a research institute of Texas A&M AgriLife Research founded in 2005 to accelerate the pace of medical discoveries and foster the development of the biotechnology industry in Texas. TIGM creates comprehensive knockout mice and has the world’s largest collection of embryonic stem (ES) cells cloned from gene trap libraries.

**Texas A&M Institute for Preclinical Studies (TIPS)**
TIPS provides a range of services that promote translational research studies at TAMU. The facility allows researchers exploring neurobiological mechanisms access to expertise in biomedical engineering, advanced imaging (including fMRI), pathology, radiography, interventional cardiology, neurology, animal behavior, chemistry, and engineering. Collaborative work with the Texas A&M College of Veterinary Medicine and Biomedical Sciences, is examining naturally occurring disease models in animals with the aim of developing new therapeutics.
TIPS also serves as the center for human brain imaging, with state-of-the-art resources including 128 slice PET/CT, and 3T MRI (Siemens 3-Tesla MAGNETOM Verio) that is 100% dedicated to research imaging, and fully equipped for any neuroimaging study. Support equipment for the 3T magnet includes a full physiological monitoring system (blood pressure, heart rate), 2 different power injectors, and the equipment needed to assess pain, track eye movements, record startle, record behavioral responses, and present visual stimuli. These facilities are currently used by 6 researchers affiliated with TAMIL (all housed in the Department of Psychology).

Texas A&M University Libraries
Texas A&M University Libraries serves both the research and study needs of students and faculty across campus. Online research collections and services include “Get it for me” and “Ask the Libraries”, thousands of books and journals, subject guides and more. Study space and additional research help can be found in any of the libraries located across campus. Space is reserved for graduate students in the medical library which is located between the old Reynolds Medical College building and the Veterinary College. The University Libraries System consists of five facilities and the online library. These facilities contain approximately 4 million volumes, with 400,000 e-books, and the library ranks second in the nation for electronic serials expenditures.

Trace Element Research Lab
The Trace Element Research Laboratory (TERL) is dedicated to providing students with a high quality education in environmental chemistry and sponsors with high quality trace element data. (TERL) offers an uncommon combination of academic research expertise and extensive experience in the environmental monitoring and regulation arenas. TERL has an international reputation, earned through numerous blind intercalibration exercises, for consistently producing high quality, low detection limit trace element data for environmental samples (including water, wastewater, sediment and biota). All work is conducted under a comprehensive quality assurance/ quality control (QA/QC) program. TERL is experienced in the use of clean (1 part per billion level) and ultra-clean (< 0.1ppb level) sampling and preconcentration procedures required to make accurate measurements of trace metals at ambient levels in both freshwater and seawater. TERL has participated in most major US environmental monitoring programs over the past decade (e.g. NOAA Mussel Watch, EPA EMAP, USFWS Contaminants Program, etc.) and has an established reputation for accurately measuring trace metal levels in samples from even the most pristine environments.

TERL facilities for low level analyses include a clean room, three modern graphite furnace atomic absorption spectrophotometers, a PSA hydride generation atomic fluorescence analyzer, a Hewlett-Packard model 4500 ICP/MS, a Perkin-Elmer Optima 3000 dual-view ICP, and complete neutron activation analysis facilities.

Finances
The neuroscience program receives the following financial resources:

Table 2

<table>
<thead>
<tr>
<th>Fiscal Year</th>
<th>HSC</th>
<th>OGS IDP Funds</th>
<th>AMP Operational Funds</th>
<th>VPR Payroll Funds</th>
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<td>2011-2012</td>
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<td>$108,122.00</td>
<td>$34,563.00</td>
<td>$238,153.00</td>
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</table>

*Received one-time additional funding of $20,000 in OGS IDP funds and $5,891 in one-time Graduate Excellence Funding.

In common with other IDPs on campus, the Chair receives $15,000 per annum in salary support. TAMIL also receives additional funding for the vice chair and each of the committee chairs ($2000 per annum).
The office of graduate and professional studies provides all IDPs with annual funding based on the formula:
$300 (#students) + $250 (#unique faculty advisory committee chairs/co-chairs) – as a measure of the interdisciplinary nature of the program
+ $12 (#weighted student credit hours) – graduate enhancement
+ $300 (#PhD students enrolled)/$240 (#MS students enrolled) + $500 (#PhD students graduated) + $400 (#MS students graduated) = strategic support

Additional funding has been provided by OGAPS in recent years as a result of re-allocation by the university.

Student Funding
The regular NRSC students receive funding for the first year, while they rotate and find a laboratory, and then each summer for the remaining 4 years. From the 2nd year of their program onwards, students are funded by their PIs for the Fall and Spring semesters. From 2011 the university provided HEEP fellowships, which funded students for 3 years and allowed us to recruit the top ranked applicants. However, the HEEP fellowships were re-allocated from IDPs in 2014, which adversely affected our ability to attract the top students. In recent years we have nominated students for the highly competitive university wide Merit and Diversity fellowships. All the applicants we have put forward have received approval for these fellowships although not all chose to come to A&M. We have recruited 1 merit and 2 diversity student fellowships using these mechanisms, which have been in extremely helpful in re-gaining our capacity to attract our top ranked student applicants.

### Table 3
TAMIN PhD Student Funding

<table>
<thead>
<tr>
<th>Year</th>
<th>Stipend Fall/Spring</th>
<th>Tuition Fall/Spring</th>
<th>Fees Fall/Spring</th>
<th>Stipend Summer</th>
<th>Tuition Summer</th>
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<td>1</td>
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<td>PI Support</td>
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<td>PI Support</td>
<td>$6,000</td>
<td>$1,399</td>
<td>$935</td>
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<td>3</td>
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<td>$6,000</td>
<td>$1,399</td>
<td>$935</td>
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<tr>
<td>4</td>
<td>PI Support</td>
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<tr>
<td>5</td>
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<td>$1,399</td>
<td>$935</td>
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### Table 4
TAMIN Diversity Fellowship Funding

<table>
<thead>
<tr>
<th>Year</th>
<th>TAMIN Stipend</th>
<th>OGAPS Stipend</th>
<th>PI Stipend</th>
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### Table 5
TAMIN Merit Fellowship Funding

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<td>PI Support</td>
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</table>

External Program Accreditations
Neuroscience is accredited as part of the University through the Southern Association of Colleges and Schools Commission on Colleges (SACS COC). External program accreditation documents can be found in Appendix I.

Date of Last APR Review
This is the first review of the program although it was reviewed by external reviewers when the program was first established on October 13th 2008.

Analysis

Alignment with Institutional Goals
Texas A&M developed a strategic plan: Vision 2020 that outlines a road map for the university. College strategic plans have been developed with Vision 2020 as a directive. The Neuroscience program closely aligns with the imperatives of Vision 2020. The twelve imperatives of Texas A&M University’s Vision can be found in Appendix J. Specifically, the Neuroscience Program addresses 3 of these imperatives: #1 elevating the faculty: the presence of a Neuroscience community assists in the recruitment and retention of top faculty; #2 strengthening the graduate program: the ability to offer stipends allows us to attract high caliber students; #6 diversifying: we have a diverse faculty and graduate students who are committed to improving diversity.

Texas A&M strives to improve its standing in the world rankings of universities and also the American Associations of Universities. The presence of a graduate program in Neuroscience is common in highly ranked institutions.

Improvements in the Program
Since the program was initiated we have made significant improvements as a result of annual retreats, continued consultations with the students, feedback from course evaluations and learning outcomes.

We have annual retreats to discuss the program and for faculty data blitz sessions. Our last retreat focused on strategies to bolster grant funding and external funds for graduate training. As part of the first retreat we examined our areas of expertise and identified 5 areas:

- Biological Rhythms, Nutrition & Metabolism
- Environmental Impact, Toxicology, Drugs & Addiction
- Learning & Memory, Cognitive & Addictive Neuroscience
- Neuroplasticity, Degeneration, Trauma & Recovery
- Women’s Health, Sex differences & Neuroendocrine
Recent hires have allowed the development of a critical mass of faculty in research in the following areas:

**Spinal Cord Injury**

**Neuromuscular Junction**

**fMRI research**

**Course Improvements and Developments**

The students suggested the formation of a “TAMIN 101”- a one-week orientation for new incoming students to introduce them to the program and research facilities. Appendix K is a sample TAMIN 101 schedule. This has proved to be very helpful for settling students into the program, introducing them to faculty and other students and showing them a range of techniques available on campus for their research. As a result of student evaluations we re-assigned the course leader and restructured our core classes Principles in Neuroscience I & II (Appendix L and M). In collaboration with the neurosurgeons in the Texas Brain and Spine Institute, we have developed the potential for neuroscience students to enroll in a clinical rotation. Students reported difficulties in scientific writing so we have developed a writing workshop for Spring 2017. In order to improve our student tracking, we instigated annual committee meetings for students. Appendix N is the Annual Committee Meeting Form students must submit upon meeting with their committee. This has proved helpful in detecting issues that may arise. In collaboration with Heather Moberly, a librarian associated with the College of Veterinary Medicine, we have a library resource page devoted to neuroscience (http://guides.library.tamu.edu/Neuroscience).

**Retention**

Since the start of the NRSC Ph.D. we have recruited a total of 50 students, with eight leaving the program and four switching to MS thesis option or other programs. The reasons for students leaving are varied and include health issues, changes in career goals/interests and family circumstances.

Graduate school is becoming increasingly more stressful and to mitigate the adverse effects of stress we try to ensure that we create a friendly atmosphere where students feel comfortable in approaching us for assistance. The program coordinator and graduate faculty advisors play a key role in facilitating this environment. There are multiple venues to bring people together that help develop a sense of camaraderie amongst students and faculty. In addition to the weekly seminars and symposia, we have an annual holiday party at a faculty member’s house. The graduate students founded a university-supported association: Students for Advancing Neuroscience Discovery and Innovation (SANDI), which meets regularly and plans outreach activities etc. Students are also involved in recruitment of new graduate students into the program and planning the symposia and poster sessions.

**Recruiting and Diversity**

In terms of recruitment, we do not have a specific allocation of resources but we do ensure that recruitment material is sent to OGAPS for graduate recruiting throughout USA. We encourage faculty to showcase our program when they visit other institutions. We recognize that the website is a crucial tool for recruitment and established an ad hoc web committee in order to continually update and improve the website (http://www.tamin.tamu.edu). We now have two recruiting sessions for students to visit A&M as many students were not able to come to our first session. We have moved the date of the recruiting weekend to February in line with other programs. We have also increased the number of members on the recruiting committee to 5 to ensure more diversity in terms of departmental and college representation and also because the work-load on this committee is intense.

The number of students from diverse backgrounds in the program is on a par with other neuroscience programs, at about 20%, but Texas is a more diverse state and so we have made significant efforts to improve the diversity of the pool of applicants. TAMIN applied for, and received, a competitive university Innovative
Graduate Recruiting Grant in order to work with neuroscience and STEM-focused training programs at the University of Texas at El Paso (UTEP) to identify undergraduate students potentially interested in PhD training in neuroscience. The main focus of this grant was to work with UTEP with the objective of expanding the number of Texas A&M PhD students with ethnically and economically diverse background. The first part of this initiative consisted of sending a group of representatives from TAMIN to visit students at UTEP. During the two day visit (March 28th and 29th), one TAMIN faculty member and two graduate students met with the directors of programs at UTEP dedicated to enhance research experience in STEM field including:

- The Research Initiative for Scientific Enhancement (RISE)
- Minority Access to Research Careers (MARC)
- Louis Stokes Alliance for Minority Participation (LSAMP)

During the El Paso visit, the TAMIN group also met with 30 students from the programs mentioned above to distribute information about TAMU, TAMIN PhD program, and the opportunity to participate in a sponsored visit to the Texas A&M campus.

As a result of this trip, UTEP students submitted brief applications to visit Texas A&M and six students were selected to come in August 2016. During this visit UTEP students went on a campus tour, had lunch with current and new TAMIN graduate students, attended a TAMIN data blitz, had dinner with graduate student mentors and faculty members of the TAMIN recruiting committee, and visited a total of eight research laboratories that are part of TAMIN.

In 2016 Drs Zoran and Welsh were CoPIs with Dr. Dann Howard on an NIH training grant: PAR-16-341, *BRIDGES TO THE DOCTORATE* (*R25*), with Texas A&M International University (TAMIU), a Hispanic-Serving Institution in Laredo, Texas, in partnership with Texas A&M University at College Station (TAMU), “The TAMIU-TAMU Bridges to the Doctorate Program (Bridges Program)”. The goal of the Bridges Program is to support educational activities with a primary focus on courses for skill development and research experiences that enhance the diversity of the biomedical, behavioral and clinical research workforce. The Bridges Program is intended to provide these activities to Master’s Level students to increase transition to and completion of PhDs in biomedical sciences. Several other TAMIN faculty were mentors on this grant application.

**Fellowship Support**
In order to encourage students to apply for fellowships we have held annual workshops for NSF (in conjunction with the College of Veterinary Medicine) and NIH graduate fellowships. Faculty within TAMIN serve as review panelists have also volunteered to serve as reviewers for these grants. To date one student Travis Goode (Maren Lab) was awarded NRSA F31 grant from NIH-NIMH in 2016.

**Funding Revenue for Student Prizes**
The local Chapter for the Society of Neuroscience established a vendor show to coincide with the annual poster session and this has proved very successful and generates funds for student poster prizes.
Academic Programs and Curricula

Programs Offered
Graduate students are only recruited to the Ph.D. program in Neuroscience although the program also has MS or non-thesis MS in Neuroscience. Course requirements are the same for MS and Ph.D. degrees.

Program Curricula (duration and comparison to peers)
Similar Ph.D. programs are offered at UT Austin and Dallas, have two Core Neuroscience classes and then a selection of electives. The Neuroscience Program at UT Austin reports the time to degree for the last 3 years is 6.6, 6.5 and 6.4 years and the degree program in Cognition and Neuroscience at UT Dallas is 4.81, 4.83 and 5.07 years (data from their 18 characteristics published on line). Our current student funding is not competitive with our peer Institutions. For example, as of FY 2016 the University of Texas at Austin provides a guaranteed 30k/year 12-month stipend (https://neuroscienceinstitute.utexas.edu/program-study)

Neuroscience Course Requirements
For Ph.D. students, a minimum of 96 credit hours beyond the baccalaureate degree or 64 credit hours beyond the Master’s degree is required. The MS degree requires a minimum of 32 credit hours and 36 credit hours for a non-thesis Master’s degree. The student’s degree plan includes formal coursework that breaks down into 6 neuroscience-related lecture-based courses (2 core courses plus 4 electives), a statistics course, a research ethics course, and academic credits received for attending weekly seminars, journal clubs and participating in first year rotations. The organization and composition of the pedagogical curriculum was based upon a comprehensive review of comparable graduate programs at peer institutions. Most students are expected to complete all formal coursework by the end of their second year, and five years is the target duration of the program. Details of the course requirements are outlined below:

1) All Ph.D. students are required to complete the two core courses, Principles of Neuroscience 1 (fall) and 2 (spring) (NRSC 601 and 602), receiving a letter grade of at least a B. Each course is 3 credit hours.
2) All Ph.D. students are required to take a minimum of 4 approved elective courses. Each elective course is 3 or 4 credit hours. The graduate program committee considers exemptions of one or more electives if the student’s thesis committee considers the exemption justified based on previous coursework (for example if a student enters the program with an MS in a related field).
3) All first-year Ph.D. students are required to take NRSC 685 Neuroscience Rotations during the fall semester. Two half-semester rotations in the fall are required, but additional rotations in the spring semester are allowable as needed. Students receive 1 credit hour per rotation.
4) All Ph.D. students are required to complete an approved course in Statistics and Experimental Design. Most options are 3 credit hour courses.
5) All Ph.D. students are required to complete an approved course in the Responsible Conduct of Research. 1 credit hour.
6) All Ph.D. students are required to be continuously enrolled in NRSC 681 Neuroscience Seminar. 1 credit hour per semester.
7) All student fill out the rest of their degree plan with research credit hours (NRSC 691). Students in their 3rd through 5th year typically take 6-8 research credit hours per semester, plus 1 credit hour each for a journal club and the weekly neuroscience seminar series. 9 credit hours per semester is considered full-time for the fall and spring semesters. Students also register for 6 credit hours of NRSC 691 during the summer session.
<table>
<thead>
<tr>
<th>Course</th>
<th>Course Name</th>
<th>Credits</th>
<th>Course Description</th>
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<tbody>
<tr>
<td>NRSC 601/BIOL 627</td>
<td>Principles of Neuroscience I</td>
<td>3 Lecture Hours</td>
<td>Detailed introduction to the basic fundamentals of cellular and molecular neuroscience; topics include membrane potentials, action potential generation, and the mechanisms underlying synaptic transmission, as well as their molecular basis. Prerequisites: Graduate classification or approval of instructor.</td>
</tr>
<tr>
<td>NRSC 602/BIOL 628</td>
<td>Principles of Neuroscience II</td>
<td>3 Lecture Hours</td>
<td>Fully integrated overview of nervous system organization and systems-level neurobiology; broad topics include sensory systems and sensory systems function, motor systems and neuromuscular function, central pattern generation and locomotion, homeostatic regulation, motivation, emotions, learning and memory, and circadian rhythms. Prerequisites: Graduate classification or approval of instructor.</td>
</tr>
<tr>
<td>NRSC 603/VIBS 603</td>
<td>Neuroanatomy</td>
<td>4 Lecture Hours 6 Lab Hours</td>
<td>Gross, developmental and microscopic anatomy of nervous system of selected laboratory and domestic animals. Prerequisite: Approval of instructor.</td>
</tr>
<tr>
<td>NRSC 604/VIBS 604</td>
<td>Biomedical Neuroendocrinology and Endocrine Disorders</td>
<td>3 Lecture Hours</td>
<td>Gross and functional anatomy and endocrine functions of neuroendocrine systems, hypothalamus and pituitary. Neuroendocrine control of puberty, sexual behavior, menstruation, ovulation, pregnancy, labor, lactation, testis, thyroid, growth, stress, diabetes, obesity, sleep, memory, learning and aging and their disorders. Overview biosynthesis, transport and signaling of neuropeptides, prostaglandins, peptide and steroid hormones. Prerequisite: Approval of instructor.</td>
</tr>
<tr>
<td>NRSC 605/VIBS 606</td>
<td>Neuroanatomical Systems</td>
<td>3 Lecture Hours</td>
<td>Emphasis on major neural systems that govern identifiable physiological functions, behavior and neurodegenerative disease; whole-brain anatomy is approached from a &quot;systems&quot; perspective, wherein components of defined functional systems are described in terms of their location, inputs and outputs, and physiological/behavioral significance in health and disease. Prerequisite: Approval of instructor.</td>
</tr>
<tr>
<td>NRSC 606/PSYC 606</td>
<td>Learning</td>
<td>3 Lecture Hours</td>
<td>Procedural and theoretical issues in study of basic learning mechanisms in animals and humans, including Pavlovian and instrumental conditioning. Application of this work to other domains and relevant biological mechanisms also discussed. Prerequisites: PSYC 340/NRSC 340 or approval of instructor.</td>
</tr>
<tr>
<td>NRSC 609/PSYC 609</td>
<td>Physiological Psychology</td>
<td>3 Lecture Hours</td>
<td>Current research and methodological procedures on physiological bases of sensation-perception, memory and learning, arousal-sleep attention, emotions and motivation. Prerequisite: PSYC 335/NRSC 335.</td>
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<tr>
<td>NRSC 611</td>
<td>Molecular Biology of Differentiation and Development</td>
<td>3 Lecture Hours</td>
<td>Major paradigms of eukaryotic gene regulation in terms of the role of gene expression during ontogeny and the effect of dysfunction in these processes on the neoplastic state. Prerequisite: None.</td>
</tr>
<tr>
<td>NRSC 615/PSYC 615</td>
<td>Perceptual Processes</td>
<td>3 Lecture Hours</td>
<td>Perceptual Processes. Complex sensory and perceptual phenomena with emphasis on the relationship between perception and motivation, cognition, creativity and instinctive/ethological; learning/ experiential factors in higher-level perceptual processes. Prerequisite: None.</td>
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<tr>
<td>Course Code</td>
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<td>Advanced Developmental Neurotoxicology</td>
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<td>Functional Neuroanatomy</td>
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<td>Neuropsychopharmacology</td>
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<td>Comparative Neurobiology</td>
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<td>Biological Clocks</td>
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<td>Signaling in Behavior and Development</td>
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<td>Neurobiology</td>
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<td>Principles of Neuropsychology</td>
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<td>Neural Development</td>
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<td>Seminar in Behavioral Neuroscience</td>
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<tr>
<td>NRSC 650/</td>
<td>Clinical Psychopharmacology</td>
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<td>PSYC 650</td>
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<td>NRSC 671/</td>
<td>Experimental Design for Behavioral Scientists</td>
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<td>Directed Studies</td>
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<td>Special Topics in...</td>
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<td>1-4 Lecture</td>
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<td>Hours.</td>
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<td>NRSC 691</td>
<td>Research</td>
<td>1-23</td>
<td>1-23 Other</td>
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<td></td>
<td>Hours.</td>
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<tr>
<td>NRSC 698/</td>
<td>Behavior, Genes, and Evolution</td>
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<tr>
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<td>Lecture</td>
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<tr>
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<td></td>
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Texas A&M Institute for Neuroscience
Admissions Criteria

The recruiting committee, consisting of the chair and 5 members of TAMIN faculty from several different departments, comprehensively reviews every application. The applicants initially are evaluated based on their curriculum vitae including research experiences and accomplishments, GRE scores, academic record including GPA, letters of recommendation, and their personal statement. Highly accomplished applicants videoconference with the recruiting committee. TAMIN invites about 10 applicants to a recruiting visit at TAMIN in early February. Final decisions about offers of admission incorporate comments from TAMIN faculty members and students about applicants with whom they interact during this visit.

Table 8

<table>
<thead>
<tr>
<th>PhD Application Information</th>
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<tr>
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<tr>
<td>Number of Applicants</td>
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<tr>
<td>Number of Transfers</td>
</tr>
<tr>
<td>2011 3 2012 2 2013 0 2014 1 2015 2 2016 0</td>
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<tr>
<td>Number Applying Directly to TAMIN</td>
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</table>

The majority of our students apply directly to Neuroscience program. However, there are a number of students that apply to the Behavioral and Cellular Neuroscience program in the department of Psychology and then their application is transferred to the Neuroscience recruiting committee, if it is more in line with our program. We also accept applications from transfer students that have been focused on neuroscience research but are in other Ph.D. programs within the university. These applicants submit a package to the recruiting committee and are interviewed prior to acceptance.

The annual average GPA and GRE scores are shown the table below and the cumulative averages since 2011 are: GRE scores V. 158 (580); Q 157 (730); GPA 3.64

Table 9

<table>
<thead>
<tr>
<th>GPA/GRE Scores For Newly Admitted Graduate Students</th>
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<tbody>
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<td>Year</td>
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<tr>
<td>-----------</td>
</tr>
<tr>
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</tr>
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<td>2012</td>
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<td>2014</td>
</tr>
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<td>2015</td>
</tr>
<tr>
<td>2016</td>
</tr>
<tr>
<td>2017</td>
</tr>
</tbody>
</table>

*New scores since 2012 have been converted to equivalent old scores in parentheses
Number of Degrees Awarded per Year

Table 10 shows the number of Ph.D. and M.S degrees in Neuroscience by year

<table>
<thead>
<tr>
<th></th>
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<td>Ph.D.</td>
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<td>2</td>
<td>2</td>
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<tr>
<td>Totals</td>
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<td>4</td>
<td>2</td>
<td>2</td>
<td>4</td>
</tr>
</tbody>
</table>

Average Time to Degree

Average time to complete the Ph.D. degree is 4.75 years and MS 2.5 years.

Academic Enhancements/High Impact Opportunities for Students

Texas A&M University has professional development opportunities for graduate students through the Office of Graduate and Professional Studies and also the Center for Teaching Excellence. For incoming students, the Office of Graduate and Professional studies has an Orientation day. Leadership opportunities are numerous and our graduate students are active in the University Graduate Student Council and also their own College Graduate Student Associations.

The Center for Teaching Excellence provides TA training and workshops throughout the year. Students can also participate in the Academy for Future Faculty. Graduate students are welcome to attend University and College sponsored grant writing workshops along with the TAMIN workshops.

The Career Center assists with job placement and provides assistance with CV writing and interview skills.

Following the rotations, NRSC students can also take advantage of development opportunities within the College of the Chair of their committee. TAMIN runs a “boot camp” TAMIN 101 the week before classes commence and we have a weekly seminar series with mandatory attendance for all Neuroscience students. The speaker is usually from another institution although internal speakers are also included, particularly new TAMIN faculty. Each semester one speaker slot is allocated to TAMIN students who are required to present prior to graduation. Appendix O contains the seminar series schedule for FY16 and FY17. An annual spring symposium is organized by the Neuroscience graduate students and a Fall poster session with vendor show organized by the local Chapter for the Society for Neuroscience.

There are four weekly journal clubs that students can attend: Neuroscience (in the medical school); Neuroscience (in ILSB); Synapses (in ILSB) and the Circadian Group (in Biology). The students may also attend several meetings in College of Veterinary Medicine: MRI rounds, Neuropathology rounds and monthly neuroscience lunch meetings. Finally, there is the Cognoscenti Working Group, which is an interdisciplinary forum for intellectual exchange on issues concerning mental functioning in humans and other species.

Every year, the Texas Brain and Spine Institute holds an annual symposium with a poster session, which provides another venue for TAMIN students to present. Awards are made to the students for best poster presentations at all these events.

The TAMIN faculty have increased their availability for 485/491 undergraduate directed studies/research experiences, which is highly recommended to all students participating in the undergraduate minor in
neuroscience. These undergraduates are frequently mentored by NRSC graduate students, which provides valuable mentoring experience for the Ph.D. students.

**Travel Grants**
Each TAMIN student is eligible to receive an annual travel award ($600-$1,200) for attending a conference. Appendix P is the Travel Award Application. We also fund some non-TAMIN graduate students and postdoctoral fellows for travel awards. The university also provides competitive travel awards and some colleges also have travel funds.

**Outreach Activities**
The TAMIN students are very active in outreach programs and founded an organization: Students for Advancing Neuroscience Discovery and Innovation (SANDI), which is dedicated to a higher understanding of the human mind and body. Its commitment to discovery, collaboration and developing technologies in the field of neuroscience provides an exciting environment for development, growth, and improvement of undergraduate and graduate research at Texas A&M University. In order to fulfill this purpose SANDI provides an enriched environment, promoting interdisciplinary communication and scientific collaboration for all students interested in neuroscience research at Texas A&M University. SANDI establishes a nourishing environment with the resources necessary to educate, inspire, unite and empower student scientists to unlock scientific mysteries through renewed vigor, creativity and innovation. In addition to scientific advancement amongst the students of Texas A&M, SANDI is committed to the future of scientific funding and discovery. In order to improve this future for neuroscience, SANDI has a responsibility to reach out into the community. It is critical for the public to understand past and current work in neuroscience in order to appreciate the need for support and funding to continue these advances. Similarly, we must prepare for the future by cultivating the minds of our youth. Exposure to the beauty and complexities of the human brain and nervous system will aid in sparking the imaginations of our children. Collectively, SANDI is an organization focused on the perpetual pursuit of education, innovation and unity for advocates of neuroscience within Texas A&M University and beyond.

Each year the students visit a local elementary school for Brain Day and participate in the Annual End Alzheimer’s walk and raised $3,500 last year. Students also assist with the annual meeting with the local Brazos Valley Multiple Sclerosis Patients Support Group. Neuroscience students have taken on leadership roles in organizing the Women in Science and Engineering (WISE) annual conference. In previous years faculty and graduate students worked with the undergraduate students to prepare for the annual Brain Bowl and then participated successfully.

**Assessment of Student Learning Outcomes**
The University requires an annual analysis of graduate programs via the WEAVE online and 18 Characteristics assessment tools. These surveys assess, amongst other measures, student-learning outcomes. The Program Coordinator, Graduate Advisor and Chair of TAMIN are responsible for inputting data into these systems. We have improved our WEAVE criteria after the first iteration and use both these tools to assess and improve the graduate program. The students are very interactive with the TAMIN administration and continually provide feedback on issues and suggest improvements to the program.

The goals of the Neuroscience Program are:
1) Prepare students to succeed as independent scholars and scientists
2) Propels students to productive research careers
3) Place graduates in postdoctoral positions
4) Enhance diversity
Our objectives for the students are:
1) Mastery of fundamental concepts
2) Experimental design and execution
3) Communications
4) Program progression
5) Publications
6) Post-graduate employment
7) Increase the diversity of the students in the program

Assessment of these objectives (1-3) are made during the Principles of Neuroscience I & II courses, the annual committee meeting and at the oral presentations made by the students. The student’s annual reports address objectives 4-6.

Overall Analysis
As a result of data collection for the 18 Characteristics of the Neuroscience Program (Appendix Q) and the WEAVE assessment we have established a mandatory annual committee meeting and evaluation report to ensure the students are on track for a timely graduation. Our students are expected to develop an in-depth knowledge within their specialty area and generate empirical papers describing their scientific results and their publications/presentations are documented annually. During the student's annual committee meeting their breadth and depth of knowledge is evaluated, noting the student's progression and reported via the Advisory Committee Meeting Evaluation form. In addition, the student's ability to interpret and apply scientific literature is evaluated and reported during annual progress meeting. They are also evaluated on their ability to communicate experimental results and conclusions. Students are now required to present to TAMIN prior to graduation and the audience evaluates their presentations. We have updated the student handbook to reflect these changes (Appendix R).

The advisory office documents the student's progress to the set milestones including taking prelims, submitting a thesis proposal, defending, as well as the completion of core curriculum NRSC PhD courses (Seminar, Principles of NRSC, Electives, Ethics, Statistics and Experimental Design). Progression evaluation is based upon the Neuroscience Ph.D. roadmap (Appendix S) and the expected Ph.D. program completion in 5 years.

Our goals and objectives have been satisfactorily achieved with one caveat. The majority of our students are compliant with our requirement for an annual meeting but a few do not follow this rule. We are considering a registration block to ensure compliance. Students have requested more assistance with writing and we have established a writing workshop for Spring 2017.
Faculty Profile

The Texas A&M Institute for Neuroscience consists of 93 faculty members. Although Texas A&M University and the Texas A&M Health Science Center have historically been independent entities of the Texas A&M System, the structure of TAMIN accommodates students from both institutions. Appendix D provides a list of all TAMIN members and their department and college. NIH style biosketches for individual TAMIN members are provided in Appendix T. The distribution of TAMIN faculty by department and college are summarized in Figures 3 and 4.

Figure 3. Faculty Distributed by Department. There are 26 departments and multiple associated laboratories. The departments with greatest representation are identified on the chart.
Core Faculty

Core Faculty are defined as full time, tenured and tenure-track, with 50% or more doctoral instruction or other individuals integral to the PhD program who direct research.

Number of Core Faculty

Based on the above criteria, all 93 current members are considered core faculty at the Texas A&M Institute for Neuroscience.

Core Faculty/Student Ratio

The student core faculty ratio is calculated by the three-year average of full-time student equivalent (FTSE)/three-year average of full-time faculty equivalent (FTFE) of core faculty. The current core faculty/student ratio is 0.3.

Core Faculty Publications

The three-year average in the number of discipline related refereed papers/publication, books/book chapters is 5 per faculty member. Appendix U lists the member’s recent publications.
Core Faculty External Funding

Table 11 shows data collected from Maestro and only includes external funding for the Texas A&M Institute for Neuroscience Faculty.

<table>
<thead>
<tr>
<th>Fiscal Year</th>
<th>Total For Year</th>
<th>% of Faculty w/External Funds</th>
<th>No. Total Faculty</th>
<th>No. Total Faculty w/External Funds</th>
</tr>
</thead>
<tbody>
<tr>
<td>FY 12</td>
<td>$12,127,447.98</td>
<td>34.44%</td>
<td>84</td>
<td>41</td>
</tr>
<tr>
<td>FY 13</td>
<td>$10,030,175.72</td>
<td>29.23%</td>
<td>79</td>
<td>37</td>
</tr>
<tr>
<td>FY 14</td>
<td>$7,891,434.40</td>
<td>29.40%</td>
<td>84</td>
<td>35</td>
</tr>
<tr>
<td>FY 15</td>
<td>$17,539,438.70</td>
<td>32.40%</td>
<td>81</td>
<td>40</td>
</tr>
<tr>
<td>FY 16</td>
<td>$14,185,712.73</td>
<td>26.66%</td>
<td>86</td>
<td>31</td>
</tr>
<tr>
<td><strong>Average</strong></td>
<td><strong>$12,354,841.91</strong></td>
<td><strong>30.43%</strong></td>
<td><strong>82.8</strong></td>
<td><strong>36.8</strong></td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>$61,774,209.53</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Core Faculty Teaching Load

The Core Faculty teaching load is determined by each department and varies considerably across the University. Many of our courses are cross-listed with departments and faculty teach neuroscience related courses throughout the university. The course instructors for the Principles in Neuroscience I & II classes invite several members of the neuroscience faculty to lecture and contribute to the associated journal club.

Faculty Diversity

The most recent data that we have for faculty diversity is from the prior year (FY16), when we had 86 faculty (10 of whom chose to keep this information confidential). Of the 76 faculty, the ethnic composition is: 49% white male, 1% black male, 1% Hispanic male, 12% other male, 24% white female, 3% Hispanic Female and 10% other female. The faculty is 37% female and 65% male. Table 12 shows the breakdown of our faculty diversity.

<table>
<thead>
<tr>
<th>FY 2016</th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>White</td>
<td>37</td>
<td>2</td>
</tr>
<tr>
<td>Black</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Hispanic</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Other</td>
<td>9</td>
<td>8</td>
</tr>
<tr>
<td>Total</td>
<td>48</td>
<td>28</td>
</tr>
</tbody>
</table>

Faculty Qualifications

Since neuroscience is an interdisciplinary program across the University, faculty hires are made based on departmental standards which differ across the university.

Analysis

The faculty of neuroscience has grown by 27% from 68 to 93 in 7 years. The faculty members contribute to the program in a number of ways: mentoring students, and teaching neuroscience either in the core courses or through courses within their departments. As an IDP we do not evaluate in terms of teaching, research and service. We do send out an annual survey to the faculty to collect data and to determine their involvement in the program. Our faculty receive many prestigious awards. For instance, in March 2017 3/24 recipients of the
Presidential Impact Awards were Neuroscience Faculty: Drs. Arum Han (College of Engineering), Stephen Maren (College of Liberal Arts), and Farida Sohrabji (College of Medicine). The Presidential Impact Fellows program includes the use of the honorific title for life, and an annual stipend of $25,000 each for the next three years to accelerate each recipient’s pedagogy, research and service impacts. Identified by his or her Dean and confirmed by academic leadership, these faculty are considered candidates for continued or new national and international acclaim and will utilize this honor to participate in national dialogue, advance their scholarship and create new partnerships.
Student Profile

Doctoral Students
Enrollment and Full-Time Status
As of Fall 2015 there are 32 total students in the Texas A&M Institute for Neuroscience graduate program consisting of 31 doctoral students and 1 masters students. They are distributed between five different departments (Biology, Neuroscience and Experimental Therapeutics, Nutrition and Food Science, Psychology and Veterinary Integrative Biosciences) in the Colleges of Science, Health Science Center/Medicine, Agriculture & Life Sciences, and Veterinary and Biomedical Sciences. Figure 5. 97% of these students are full-time (one part-time student).

Student Diversity and Demographics
The gender and ethnicity of each new incoming class for the past five years is shown in Table 13. A diversity of 20% is similar to other neuroscience programs.

Table 13

<table>
<thead>
<tr>
<th>New Student Demographics</th>
<th>2012</th>
<th>2013</th>
<th>2014</th>
<th>2015</th>
<th>2016</th>
</tr>
</thead>
<tbody>
<tr>
<td>Males</td>
<td>5</td>
<td>2</td>
<td>6</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Females</td>
<td>4</td>
<td>5</td>
<td>2</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Domestic – Caucasian</td>
<td>4</td>
<td>5</td>
<td>2</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Domestic – Non-Caucasian</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>International</td>
<td>4</td>
<td>1</td>
<td>2</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>MS</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>PhD</td>
<td>9</td>
<td>7</td>
<td>8</td>
<td>5</td>
<td>5</td>
</tr>
</tbody>
</table>

Retention and Graduation Rates
The retention and graduation rates for NRSC students since 2011 are shown below in Tables 14 and 15. It is important to note that the first PhD class began Fall 2010. Due to this, the graduated values are lower and the current student values are higher. Current students were used to calculate the retention, but not the graduation percentages.

Table 14

<table>
<thead>
<tr>
<th>Student Retention and Graduation Rates</th>
<th>Total Students</th>
<th>Current Students*</th>
<th>Left Program</th>
<th>Graduated</th>
<th>% Retained</th>
<th>% Graduated</th>
</tr>
</thead>
<tbody>
<tr>
<td>MS</td>
<td>3</td>
<td>1</td>
<td>0</td>
<td>2</td>
<td>100%</td>
<td>100%</td>
</tr>
<tr>
<td>PhD</td>
<td>45</td>
<td>31</td>
<td>5</td>
<td>10</td>
<td>88.9%</td>
<td>50%</td>
</tr>
<tr>
<td>Overall</td>
<td>48</td>
<td>32</td>
<td>5</td>
<td>12</td>
<td>89.6%</td>
<td>70.6%</td>
</tr>
</tbody>
</table>
Table 15
Graduate Degrees Awarded

<table>
<thead>
<tr>
<th>Year</th>
<th>M.S.</th>
<th>Ph.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>2016</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>2015</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>2014</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>2013</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>2012</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Average Time to Degree
The average time to degree from 2011 to 2017 was 4.75 for the PhD, and 2.5 for the MS.

Average Institutional Financial Support Provided
The financial support for TAMIN students varies between departments but the stipend averages $20,000/year plus the cost of tuition for full-time status. This is usually provided in the form of assistantships, scholarships, fellowships and sometimes, individual grants to the student. All students in good standing and with full-time status receive a non-resident tuition waiver. Table 16 shows the total and average financial support for the past 4 years. These totals only include TAMIN and TAMIN awarded fellowships, not financial support provided by student PIs.

Table 16
Graduate Financial Support Per Year

<table>
<thead>
<tr>
<th>Year</th>
<th>Total Financial Support</th>
<th>Avg. Tuition &amp; Fees</th>
<th>Avg. Stipend</th>
<th>Avg. Travel Awards</th>
<th>Avg. Heap Fellow Insurance</th>
<th>Avg. Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>2016</td>
<td>$510,652</td>
<td>$4,522</td>
<td>$13,847</td>
<td>$924</td>
<td>$2,496</td>
<td>$21,788</td>
</tr>
<tr>
<td>2015</td>
<td>$572,870</td>
<td>$5,149</td>
<td>$16,024</td>
<td>$1,073</td>
<td>$2,496</td>
<td>$24,741</td>
</tr>
<tr>
<td>2014</td>
<td>$672,335</td>
<td>$6,045</td>
<td>$19,170</td>
<td>$1,232</td>
<td>$2,496</td>
<td>$28,942</td>
</tr>
<tr>
<td>2013</td>
<td>$568,029</td>
<td>$6,206</td>
<td>$19,598</td>
<td>$1,174</td>
<td>$2,496</td>
<td>$29,474</td>
</tr>
</tbody>
</table>

Percent of Full-Time Students with Institutional Financial Support
Of the current graduate students, 100% have at least $1,000 of annual support, and 100% of the full-time students in years 1-5 receive financial support for cost of tuition.

Student Publications and Presentations
Doctoral students throughout the past 5 years averaged 7.83 presentations and 2.11 publications while in the program. The FY 16 class of students had a total of 292 presentations and 79 publications.

Graduate Employment/Placement Profile
From 2013-2016, 10 PhD and 2 MS degrees were awarded. Of these 12 students, 100% were employed in the field within one year of graduation. Figure 6. Complete TAMIN graduate student employment profile.

Figure 6. Graduate Employment Profile.
Concluding Observations

The TAMIN EC has worked diligently to maintain the democratic integrity and grass roots nature of TAMIN by working closely with the elected TAMIN committees. The faculty remains highly collegial and committed to concept of TAMIN and the Neuroscience Ph.D.

The main challenge to TAMIN faculty is the reduction in federal funding which is impacting the number of R01 grants - the fundamental building blocks for academic programs in neuroscience. We have established a research committee, which aims to provide a supportive environment to improve our grant funding success for faculty, postdoctoral fellows and graduate students.

Our goals over the next several years are to continue to improve the graduate program and to obtain NIH and NSF training funds and NIH multi-investigator grants. This will allow us to develop a track record in our areas of strength and then to become competitive for center grant consideration. The senior hires made in TAMIN have significantly strengthened existing focus areas in basic neuroscience, translational neurology, learning and memory. These senior faculty members also add to the experience of the TAMIN faculty and to the combined TAMIN portfolio for garnering these large multidisciplinary grants. Moreover, Texas A&M is increasing the number of faculty in the area of spinal cord injury research with 4 new hires expected in 2017, bringing the total number to 8. This is by virtue of the efforts of Dr. Grau and generous funding from Mission Connect funding.

Highlights

- A highly collegial, committed faculty willing to give of their time in teaching and advising students in neuroscience.
- Outstanding program coordinator.
- Wide ranging expertise of faculty across departments.
- Presence of Core labs: fMRI, Image Analysis, Genomics, NMR etc.
- Excellent caliber of students applying to the program.
- Highly productive and successful students in the program.
- Successful postdoctoral placement of students upon graduation.
- NIH fellowship awarded to Travis Goode.
- Ample opportunities for outreach and leadership.
- Students empowered through shared governance of the program.
- Unique collaborations between Colleges of Veterinary Medicine and Medicine collaborations and opportunities for students in One Health initiatives.
- Excellent funding from the university to support the program.
- Successful and expanding minor in Neuroscience.
- University Degree in Neuroscience under development.

Table 17

<table>
<thead>
<tr>
<th>Markers for Success and Achievement</th>
<th>2010</th>
<th>Projected at Start of Program</th>
<th>2017</th>
</tr>
</thead>
<tbody>
<tr>
<td>Publications/faculty</td>
<td>2.2</td>
<td>3</td>
<td>4.2</td>
</tr>
<tr>
<td>Graduate students</td>
<td>5</td>
<td>25</td>
<td>31</td>
</tr>
<tr>
<td>Undergraduate students</td>
<td>80</td>
<td>160</td>
<td>291</td>
</tr>
<tr>
<td>Faculty</td>
<td>68</td>
<td>82</td>
<td>93</td>
</tr>
</tbody>
</table>
Future improvements to the program will be increasing the graduate course offerings, improving our recruitment and retention (particularly of diverse students), the development of an NIH T32 training grant application and increased F31 applications. The new research committee of TMIN will continue to work with faculty and trainees to bolster our RO1 grant applications and multi-PI grants. The four new hires in spinal cord injury will increase the number of faculty in this focus area to 7 and increase the opportunities for program project grants.

The popularity of neuroscience with the increasing numbers of undergraduates taking the minor in neuroscience and the new University Studies Degree in Neuroscience, presents opportunities for teaching assistantships for the graduate students and potential for the development of new courses and recruitment of new faculty into the university.

We look forward to the administration’s continued support of our program and working with the research support teams with the VPR’s office to identify neuroscience-related RFPs coming down the pipeline, to produce competitive group proposals in our areas of strength, and once obtaining these resources, to move TMIN and Texas A&M research to the next level of national and international prominence.
APPENDICES

Appendix A: THECB Comments
Appendix B: University Rules Framework for IDPs
Appendix C: University Rules Standard Admin. Procedures for IDPs
Appendix D: TAMIN Faculty
Appendix E: TAMIN Proposal
Appendix F: TAMIN Budget
Appendix G: TAMIN Policies
Appendix H: Duties of EC
Appendix I: Institutional Profile for DOE Accredited Docs.
Appendix J: Texas A&M University Vision 2020
Appendix K: TAMIN 101 Schedule
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Appendix M: Principles in Neuroscience II Syllabus
Appendix N: Graduate Annual Meeting Evaluation Form
Appendix O: TAMIN Seminar Series
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Appendix Q: TAMIN 18 Characteristics
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Appendix S: Roadmap to the Ph.D.
Appendix T: Faculty Biosketches
Appendix U: Faculty Publications
APPENDIX A

THECB Comments
THECB Comments from Final Report

Thank you for submitting your fifth and final annual progress report for Texas A&M University’s (TAMU) PhD program in Neuroscience. Based on your submission, it appears that this program is off to a good start.

Enrollments continue to increase, and are meeting or exceeding enrollment projections for Year Five. The program has enrolled 27 students, 100 percent of whom are full-time. The program is also committed to its students, providing 100 percent of them with financial assistance averaging $28,837 per student. This is a slight decline from the average of $29,473 offered in Year Four, and while this amount is competitive and should allow the program to continue to attract and retain high-quality students, I encourage the program to continue its level of commitment to its students.

Students continued to generate publications and presentations with 27 publications and 69 presentations. The number of publications and presentations is a clear indicator that students are actively engaged in research, collaboration with faculty, and professional development. I congratulate the program for its success in this area. Four students advanced to candidacy and three earned a degree in Year Five, which makes seven graduates for the program in its first five years, exceeding original expectations for those completing their degrees.

In Year Five, four faculty were added to the program, either directly or through interdisciplinary affiliation. Faculty productivity remained steady at five publications per year per faculty member. Faculty also remained active in seeking external funding, with 45 PIs securing 129 grants. The level of external funding should enable the program to continue to attract and retain high-quality students.

The Neuroscience doctoral program has made good progress. The success of this interdisciplinary program could serve as a model for other programs, enabling institutions to improve interdisciplinary research.
APPENDIX B

University Rules Framework for IDPs
UNIVERSITY RULE

03.02.99.M1 Administrative Framework for Interdisciplinary Programs
Approved December 14, 2006
Revised October 12, 2012
Revised February 11, 2016
Next Scheduled Review: February 11, 2021

Rule Statement

This Rule ensures that appropriate offices at the University are engaged in verifying that the establishment and review of administrative units and programs comply with the required information and steps needed for notification and/or approval by the System, the Texas Higher Education Coordinating Board, and all relevant accreditation bodies.

Reason for Rule

This Rule clarifies responsibilities and expected support for creation and engagement in interdisciplinary activities and ensures that appropriate evaluations and notifications occur in the creation and continuation of such programs.

Procedure and Responsibilities

1. GENERAL

1.1 An Interdisciplinary Program (IDP) involves a group of faculty from more than one discipline representing more than one college, organized and administered by the procedures outlined in this Rule.

1.2 An IDP may be formed for the purpose of enhancing research and scholarly activities beyond what is possible through the traditional administrative structure.

1.3 An IDP may also be formed for the purpose of enhancing curricular activities and overseeing graduate and/or undergraduate education for a degree program, a concentration, a minor, or a transcripted certificate that does not exist in an existing academic unit. Such programs should follow the normal approval process for curricular programs.

1.4 An Interdisciplinary Overview Council (IOC), which consists of all deans of colleges having faculty participating in any IDP, together with the Vice Provost,
will review proposals for the creation of new interdisciplinary programs and evaluate cyclical reviews for reaffirmation or discontinuation of established programs. The Vice Provost shall serve as chair of the IOC.

2. ESTABLISHING INTERDISCIPLINARY PROGRAMS

2.1 For a group of faculty to establish an IDP, they must submit a proposal in compliance with University SAP 03.02.99.M1.01.

2.2 The deans of colleges, the Office of the Provost and Executive Vice President, the Office of the Executive Vice President for the HSC, or the Office of the Vice President for Research may provide support for the development of IDPs consistent with their respective areas of responsibility.

2.3 All proposals for creating Interdisciplinary Programs will be submitted to the Vice Provost through a committee composed of the department heads with faculty participating in the proposed program, and the IOC.

2.4 The Provost and Executive Vice President will make the final decision regarding the approval of IDPs. The establishment of, and funding for, the operating budget is the collective responsibility of the participating units or other offices in the university as proposed and approved by the IOC.

3. PROGRAMMATIC REVIEW

3.1 It is responsibility of the IOC to establish a review schedule that will involve the department heads with faculty participating in the program and the Executive Committee of the program.

3.1.1 All IDPs that do not award degrees will be dissolved after 5 years unless a review explicitly states the IOC, in concurrence with the Vice President for Research and the Provost and Executive Vice President recommend their continuation.

3.1.2 All IDPs that do award degrees will be reviewed in the 7 year cycle required by the University Academic Program Reviews. These reviews or any notification from the Texas Higher Education Coordinating Board of a low producing program could result in the termination of the degree, with an appropriate ‘teaching out’ of the program for participants in the IDP.

3.2 As part of the normal annual review of faculty, each participating department head will review with each of their involved faculty members their future level of participation in the IDP as well as the results of their previous participation. These discussions may be incorporated into promotion, tenure, and merit raise decisions.
4. COORDINATION OF INTERDISCIPLINARY PROGRAMS WITH OTHER MEMBERS OF THE A&M SYSTEM

IDP efforts may include faculty from other members of The Texas A&M University System as outlined in the program’s bylaws. A memorandum of understanding must be initiated between the system member(s) and Texas A&M University, as represented by the Vice Provost with the concurrence of the Vice President for Research or Dean of Faculties, to establish the level of participation of the system member(s) in the IDP.

Related Statutes, Policies, or Requirements

University SAP 03.02.99.M1.01 Creation and Review for Interdisciplinary Programs

Contact Office

Vice Provost
979-845-4016
APPENDIX C

University Rules Standard Admin. Procedures for IDPs
STANDARD ADMINISTRATIVE PROCEDURE

03.02.99.M1.01 Creation and Review for Interdisciplinary Programs

Approved October 12, 2012
Revised February 11, 2016
Next Scheduled Review: February 11, 2021

SAP Statement

This Procedure describes the required steps in creating and reviewing interdisciplinary programs (IDPs) either research or curricular, and the functioning of the interdisciplinary overview council (IOC).

Reason for SAP

The Procedure clarifies the information required and reviews and approvals required in the creation or continuation of interdisciplinary programs.

Procedure and Responsibilities

1. GENERAL

All Interdisciplinary Programs must ensure that the faculty and student roles are clearly articulated and align with the administrative units where their positions are located. In concert with these understandings the expectations of support from these administrative units should be clearly defined and authorized in a well documented manner.

2. ESTABLISHING INTERDISCIPLINARY PROGRAMS

2.1 For a group of faculty to establish an IDP they must;

2.1.1 Develop a set of bylaws for the IDP including a mission statement, membership criteria, and procedures for selecting an Executive Committee (EC) and its leadership (e.g., a chair or co-chairs). The Vice Provost must approve these bylaws.
2.1.2 Identify a department or set of departments (called sponsoring departments) and the corresponding colleges (called sponsoring colleges) to act as advocates for the proposed program during the creation/evaluation process.

2.1.3 In conjunction with the sponsoring units, identify a department in which the IDP will reside administratively. This department is called the administrative department, and the corresponding college is called the administrative college.

2.1.4 Submit a proposal for the establishment of the IDP containing the items listed in Section 2.2 of this SAP. This proposal originates with the faculty group seeking to establish the IDP and is routed for approval through the following:

a. The sponsoring department heads.

b. The sponsoring deans.

c. The Interdisciplinary Overview Council (IOC).

d. The Vice Provost, who will consult with appropriate University units such as the Executive Vice President of the HSC, the Vice President for Research, the Associate Provost for Graduate and Professional Programs, the Associate Provost for Undergraduate Programs, and the Associate Provost for Academic Affairs (for accreditation and assessment issues).

e. Final approval is through the Provost and Executive Vice President before submission to any external authority required before implementation.

2.2 The proposal for establishing an IDP must contain the following items:

2.2.1 The rationale for the creation of the IDP.

2.2.2 The items described in Section 2.1.

2.2.3 A description of the Advisory and Evaluation Committee (A&EC) of Participating Department Heads, which consists of the department heads of academic departments having faculty members participating in the IDP. Annually, one of the Department Heads will be selected by this Committee to serve as the Lead Department Head.

2.2.4 A budget (and budget justification) for the operating costs of the IDP.
2.3 The Provost and Executive Vice President will make final decisions regarding the approval of IDPs. Establishment of, and funding for, the operating budget is the collective responsibility of the A&EC, other participating units or other offices in the university as proposed and approved by the IOC.

3. PROGRAMMATIC AND FACULTY REVIEWS

3.1 Annual reports will be submitted by the EC to the A&EC for the program, who will ensure that the report is distributed appropriately. A yearly meeting of the A&EC and EC will be held to review the performance of the IDP. Additional meetings may be called at the discretion of the A&EC or IOC.

3.2 As part of the normal annual review of faculty, each participating department head will review with each of their involved faculty members their future level of participation in the IDP as well as the results of their previous participation. These discussions may be incorporated into promotion, tenure, and merit raise decisions.

Related Statutes, Policies, or Requirements

University rule 03.02.99.M1, Administrative Framework for Interdisciplinary Programs

Contact Office

Vice Provost
979.845.4016
APPENDIX D

TAMIN Faculty
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<tr>
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School of Public Health (CLPH)

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APPENDIX E

TAMIN Proposal
April 1, 2010

MEMORANDUM

TO: Dr. Karan L. Watson
    Interim Provost and Senior Vice President for Academics

FROM: Dr. Jeffrey R. Seemann
       Vice President for Research

SUBJECT: Texas A&M Institute for Neuroscience (TAMIN) – Step 2 Agenda Briefing Item for Consideration at the Meeting of the Board of Regents in May, 2010

Attached for your review and for forwarding to Texas A&M University President Bowen Loftin is a proposal for creation of the Texas A&M Institute for Neuroscience (TAMIN), a multidisciplinary program between Texas A&M and the Texas A&M Health Science Center. Dr. Nancy W. Dickey, president of the Health Science Center, is sending separately a letter of support for this proposal.

Please contact my office if you need further information or have any questions regarding the proposal. Thank you for your consideration of this request.

Attachment

cc: Dr. Nancy W. Dickey
    Dr. Charles A. Johnson
    Dr. James W. Grau
Agenda Item No.

AGENDA ITEM BRIEFING

Submitted by: R. Bowen Loftin, President
Texas A&M University

Subject: Establishment of the Texas A&M Institute for Neuroscience (TAMIN)

Proposed Board Action:

Approve the establishment of the Texas A&M Institute for Neuroscience, a multidisciplinary program between Texas A&M University and the Texas A&M Health Science Center.

Background Information:

The Board of Regents discussed the concept for the Texas A&M Institute for Neuroscience at its January 2010 meeting.

The proposed Texas A&M Institute for Neuroscience will be a multidisciplinary program between Texas A&M University and the Texas A&M Health Science Center. The faculty of neuroscience at the university and the health science center brings researchers together to discover cures for addiction, Alzheimer’s, sleep disorders, and to reduce suffering from pain and promote recovery after neural injury. The hope is to translate new laboratory discoveries to effective clinical treatments, and this requires expertise drawn from nearly every college within Texas A&M and the Health Science Center. The Texas A&M Institute for Neuroscience will provide an organizational structure for investigators to strengthen interdisciplinary, collaborative research and enhance research funding opportunities among members.

A&M System Funding or Other Financial Implications:

The Texas A&M Institute for Neuroscience will be supported through internal commitments made by units in Texas A&M University and The Texas A&M Health Science Center. This includes building upon an established solid base of support through its existing interdisciplinary faculty and approved graduate program, as well as funding provided through the selection of TAMIN as one of eight Initial University Multidisciplinary Initiatives (IUMRIs) in the Academic Master Plan White Paper Process. As an IUMRI, the institute will receive funding from Texas A&M for important senior faculty hires and operational expenses. Additionally, for the first two years of the program, the Vice Presidents of Research at Texas A&M and the Health Science Center, along with the Deans of the Colleges of Medicine, Veterinary Medicine, Science, and Liberal Arts, will provide an additional $12,000/year.
Agenda Item No.

TEXAS A&M UNIVERSITY
Office of the President
April 1, 2010

Members, Board of Regents
The Texas A&M University System

Subject: Establishment of the Texas A&M Institute for Neuroscience (TAMIN)

I recommend adoption of the following minute order:

“The Texas A&M Institute for Neuroscience is hereby established as a multidisciplinary program between Texas A&M University and The Texas A&M University System Health Science Center as a joint venture between these two System institutions.”

Respectfully submitted,

R. Bowen Loftin, President
Texas A&M University

Approval Recommended:

Michael D. McKinney
Chancellor

Nancy W. Dickey, President
Texas A&M Health Science Center

Approved for Legal Sufficiency:

Andrew L. Strong

Frank B. Ashley III
Vice Chancellor for Academic Affairs
ATTACHMENT TO ITEM

Executive Summary
Texas A&M Institute for Neuroscience
Texas A&M University and
Texas A&M System Health Science Center

Rationale

Neuroscience is a field devoted to the scientific study of the nervous system, from its molecular/cellular underpinnings to the organization of neural circuits, and the manifestation of this biological/neurochemical machinery as behavioral, physiological and psychological processes. It aims to detail both how the normal system operates and how alterations in function contribute to clinical diseases, such as mental illness, dementia, developmental disorders, neurodegenerative diseases, chronic pain, drug addiction, and the loss of function with aging or neural injury. These health issues are a central focus of funding initiatives at the National Institutes of Health, which directs over 18% of its budget to the area of neuroscience.

The Faculty of Neuroscience at Texas A&M University (TAMU) and the Texas A&M Health Sciences Center (TAMHSC) brings researchers together to discover cures for addiction, Alzheimer’s, sleep disorders, and to reduce suffering from pain and promote recovery after neural injury. Neuroscientists understand that attacking these problems requires a multidisciplinary approach that couples research at the molecular level, to studies of neuron function and the immune system, and to how the whole brain is organized into coherent systems that underlie our capacity to think, feel, and act. The hope is to translate new laboratory discoveries to effective clinical treatments, and this requires expertise drawn from nearly every college within TAMU and TAMHSC.

TAMU and the TAMHSC have recognized the strength of the neuroscience program and have targeted it for hiring, with 18 new assistant professors hired since 2003. The Texas A&M Institute for Neuroscience (TAMIN) concept was submitted as a white paper to the Research Roadmap Committee and selected as one of 8 (out of 111 proposals) recommended to the Steering Committee. The development of a new Institute for Neuroscience will coalesce over 70 established, well-funded, investigators and a growing group of young investigators to foster innovation. The Institute, in conjunction with targeted hires and the allocation of research space in the new Interdisciplinary Life Sciences Building, will enhance the visibility of Neuroscience research conducted at Texas A&M to national and international levels. This research enterprise is bolstered by an active training program that includes over 70 graduate students, and 22 postdoctoral trainees, working in the area of Neuroscience at Texas A&M. Training opportunities were recently augmented through the approval (May 2009) of the Ph.D. in Neuroscience (NRSC). A state-of-the-art infrastructure to support successful well-funded research programs and formal training in Neuroscience will make Texas A&M highly attractive for additional prominent faculty, highly skilled junior faculty and outstanding graduate and undergraduate students seeking degrees in Neuroscience. The ability of Texas A&M to combine resources from diverse Colleges within a single institute will make the Texas A&M Institute for Neuroscience unique among currently established programs in the State and in the Nation.

Impact on Training and Education

TAMIN will serve as the administrative home for both the graduate program in Neuroscience and the undergraduate minor. Over 70 students are currently working with members of the Faculty of Neuroscience. A Ph.D. in Neuroscience (NRSC) was approved in 2009 and the program is reviewing students seeking admission to the program in the Fall of 2010. Current TAMU/TAMHSC students
have also been given the opportunity to transfer into the program. We anticipate admitting 6-8 graduate students per year and that the program will grow to a size of 25-30 Ph.D. students within 5 years. Another 50-60 students that are working in the area of Neuroscience, but receiving more specialized degrees, will also benefit from Institute activities. These include a weekly seminar series, annual colloquia, travel awards, and enhanced course offerings.

Neuroscience has also encouraged undergraduate education in two ways. First, we have established a minor in NRSC that has undergone rapid growth since its inception a few years ago (with close to 100 students now participating). Second, many students benefit from the opportunity to do independent research, and senior research projects, in the area of Neuroscience. In the future, we hope to introduce a major in Neuroscience.

**Financial Support**

Neuroscience was selected for funding under the Initial University Multidisciplinary Research Initiatives (IUMRI) program. These funds will provide a base budget of approximately $270,000 by FY'14. The bulk of these funds (close to 75%) will be used to support graduate training, allowing students to perform laboratory rotations during their first two semesters in the program. The remaining funds needed for graduate training (estimated at $492,000) are provided through participating faculty in the form of teaching and research assistantships. The remaining IUMRI funds are used to support TMIN programs, including the seminar series, graduate recruiting, student travel awards, and program administration. Additional funding for these activities is provided by participating TAMU colleges (approximately $1,000/faculty member) and TAMHSC ($32,000 in the current fiscal year).

**Governance**

TAMIN will be overseen by an Administrative Council that includes the Council of Participating Deans, the Deans of Graduate Studies (TAMU and TAMHSC), and the Vice Presidents of Research (TAMU and TAMHSC). The internal governance of the program is dictated by a set of approved Policies. An elected Chair provides administrative leadership and will serve as the Director of the Institute. The Chair oversees TAMIN with input from the Executive Committee, composed of the chairs of each standing committee (Curriculum, Recruiting, Membership, and Seminar) and a graduate student representative. Each standing committee is composed of a Committee Chair and 4 additional members. All positions are elected for two-year terms, staggered across years. To encourage coordination with Department-level activities, the Chair and Executive Committee will receive input from an Advisory Committee composed of Participating Department Heads.

**Review**

Current trends suggest that TAMIN will provide an essential integrative structure that will serve TAMU and TAMHSC for decades to come. The program will undergo annual review by participating Department Heads, Deans, and the VPRs at TAMU and TAMHSC. An external review of the graduate program was conducted in 2008. Every 5 years, TAMIN and the graduate program will undergo an external review as dictated by TAMU/TAMHSC policy.
Proposal
Texas A&M Institute for Neuroscience
Texas A&M University
Texas A&M System Health Science Center

Proposed Institute
Texas A&M Institute for Neuroscience

Overview
The proposed Texas A&M Institute for Neuroscience (TAMIN) will be a multidisciplinary program between Texas A&M University (TAMU) and The Texas A&M University Health Science Center (TAMHSC). The faculty of neuroscience at TAMU/TAMHSC brings researchers together to discover cures for addiction, Alzheimer's, sleep disorders, and to reduce suffering from pain and promote recovery after neural injury. Neuroscientists understand that attacking these problems requires a multidisciplinary approach that couples work at the molecular level, to studies of neuron function and the immune system, and how neural-based effectors are organized into coherent systems that underlie our capacity to think, feel, and act. The hope is to translate new laboratory discoveries to effective clinical treatments, and this requires expertise drawn from nearly every college within TAMU and TAMHSC. The TAMIN will provide an organizational structure for investigators to strengthen interdisciplinary, collaborative research and enhance research funding among members.

TAMIN Highlights
- Builds on an Interdisciplinary Research Program initiated in 1992 and officially approved in 2001, with over 70 faculty, 22 postdoctoral trainees and 70 graduate students involved in Neuroscience research
- Involves 9 colleges, 20 departments, from both TAMU and TAMHSC
- Has an established administrative structure and policies
- Has an approved memorandum of understand for cross-TAMU/TAMHSC operation
- Proposal is guided by an external program review (fall, 2008)
- TAMIN is integrated with the newly approved Ph.D. program in NRSC (spring 2009)
- An undergraduate minor in NRSC and the development of a major
- Funded under the Initial University Multidisciplinary Research Initiative (IUMRI)
- Identified for growth, with the recruitment of 3 senior researchers
- Heep fellowships for graduate training
- Builds on a strong commitment from the TAMHSC and TAMU colleges
1. RATIONALE

1.1. Global Merit and Impact

Neuroscience is a field devoted to the scientific study of the nervous system, from its molecular/cellular underpinnings to the organization of neural circuits, and the manifestation of this biological/neurochemical machinery as behavioral and psychological processes. It aims to detail both how the normal system operates and how alterations in function contribute to clinical diseases, such as mental illness, dementia, developmental disorders, neurodegenerative diseases, chronic pain, drug addiction, and the loss of function with aging or neural injury. These health issues are a central focus of funding initiatives at the National Institutes of Health, which directs over 18% of its budget to the area of neuroscience.

The recognition that treating neurological disorders required an interdisciplinary approach led to the formation of the Society for Neuroscience in 1970. In just under 4 decades, this organization has become one of the largest scientific organizations in the world, with over 38,000 members and is still growing (see Figure 1). The society, in turn, has fueled the development of neuroscience graduate programs across the nation, which now number over 160, with many schools also offering an undergraduate major in neuroscience.

Neuroscience has exhibited phenomenal growth because it addresses important societal issues and offers tractable solutions. Examples of progress, in domains studied by neuroscientists at Texas A&M University (TAMU) and the Texas A&M Health Sciences Center (TAMHSC):

- **New treatments for Alzheimer’s disease**: Studies have shown that the brain afflicted with Alzheimer’s disease contains lower levels of the neurotransmitter acetylcholine. Based on this idea, researchers developed new drug treatments (cholinesterase inhibitors) that attempt to maintain normal levels of acetylcholine and can aid memory, thinking, and functional abilities in some people with Alzheimer’s disease. Improvements allow some patients to resume normal routines in life. In addition to helping people directly affected by memory-impairing ailments, these treatments help cut financial costs to society and the government.

- **Multiple sclerosis** impacts about 1 in every thousand individuals and costs the United States more than $9.5 billion annually in medical care and lost productivity.
Neuroscience research has begun to elucidate the processes that underlie this terrible disease (how the immune system destroys the protective myelin covering around nerves) and has led to techniques that allow physicians to diagnose multiple sclerosis earlier and track its progress so that treatments can be effectively adjusted. New drugs (e.g., interferon, glatiramer acetate, mitoxantrone) can make relapses less frequent and delay further damage from disease.

Annually, about 11,000 Americans experience a spinal cord injury. Medical professionals once viewed severe spinal injuries as incurable, counseling patients that there was no hope of regaining sensory/motor function. Over the last 20 years, neuroscientists have shown that there is reason for hope—that rehabilitation can activate circuits in the lower spine that organize locomotor behavior and allow patients, who were confined to a wheelchair, to walk using a rollator. New techniques (e.g., hypothermia) have been developed to reduce tissue loss after injury and researchers are working to develop procedures that promote recovery by encouraging new neural growth across an injury.

Pain can destroy a person’s quality of life and ability to work, costing U.S. employers an estimated $80 billion a year in sick days and lost productivity. Opiate drugs effectively inhibit pain, but are highly addictive. Neuroscience research has shown that the problem of addiction can be lessened by microinjecting opiates into the spinal cord, blocking the pain signal before it is relayed to the brain. Patients suffering from cancer, who received spinal opiate administration, experienced better pain relief, significantly fewer side effects, and lived longer.

Opiate addiction impacts between 750,000 and 1 million people in the U.S. and costs an estimated $180 billion a year, in health care, reduced job productivity, and crime. Neuroscience research has identified the receptors engaged by opiates, where they are localized, and how they affect reward systems (e.g., the neurotransmitter dopamine). New drugs (e.g., buprenorphine) that engage these opiate receptors can help control the craving that fuels addiction. Other drugs (e.g., naltrexone) block opiate receptors and are used to prevent over-dose. Interestingly, alcoholism also appears to be linked to the activation of endogenous opiate systems and blocking opiate receptors with naltrexone has been shown to reduce the craving for alcohol in some alcoholic individuals.

Sleep disorders (insomnia) impact 1 in 3 people and cost an estimated $14 billion for the direct costs of health-care services and $28 billion for indirect costs such as loss of work. Early research showed that drugs (benzodiazepines) that enhance the operation of the inhibitory neurotransmitter GABA can promote sleep, but may have adverse effects (e.g., memory impairments). Neuroscientists, working with biochemists and structural biologists, have discovered new drugs (e.g., Ambien, Sonata) that target GABA and promote sleep with few adverse side effects.

Autism affects about one in every 200 children in the U.S. and the societal cost for caring for people with autism is approximately $90 billion per year. Neuroscientists have begun to link this disease to a genetic vulnerability, identifying several genetic “hotspots” associated with the disorder. New imaging techniques have revealed differences in the
brains of children with autism. Evidence suggests that the disease may be linked to problems in immune function and, in some cases, exposure to environmental toxins. Though no cure has been discovered, behavioral treatments (e.g., one-on-one teaching approaches) can improve function.


The Faculty of Neuroscience at TAMU/TAMHSC brings researchers together to discover cures for addiction, Alzheimer's, sleep disorders, and to reduce suffering from pain and promote recovery after neural injury. Neuroscientists understand that attacking these problems requires a multidisciplinary approach that couples work at the molecular level (leading to new drug discoveries), to studies of neuron function and the immune system, and to how neural-based effectors are organized into coherent systems that underlie our capacity to think, feel, and act. The hope is to translate new laboratory discoveries to effective clinical treatments, and this requires expertise drawn from nearly every college within TAMU and TAMHSC.

The Faculty of Neuroscience (FNS) at Texas A&M was created in 1992 and was formally recognized as an Interdisciplinary Research Program (IRP) in 2001. The FNS includes 74 faculty (at TAMU/TAMHSC; plus adjuncts at TAMU-Corpus Christi [1] and Kingsville [3]), 22 research scientists/postdoctoral trainees, and 73 graduate students. Membership in the faculty requires review by the FNS Membership Committee and the approval of the FNS Executive Committee. Nearly half of the faculty members have been involved in the program for over a decade, demonstrating the long-term commitment and operational solidity of this body. Within the FNS, areas of concentration have developed that focus on research strengths at Texas A&M, including: Aging, Biological Rhythms and Sleep Disorders, Cognitive and Affective Neuroscience, Drugs and Addiction, Injury and Repair, Learning, Development and Genetic Disorders, Neurogenetics, Neuroendocrine Function and Sexual Behavior, Neuroimmunology and Degenerative Processes, Social Neuroscience, Structural and Cellular Neuroscience, and Translational Neuroscience.

These interdisciplinary ties have yielded increased grant support (current funding: over $50,000,000 [direct-indirect] and productivity (approximately 2.5 peer-reviewed articles/year/FNS member) (see [FNS Annual Report](#)).

The FNS became an Interdisciplinary Degree Program (IDP) in the fall of 2009 with the approval of our Ph.D. program by the Coordinating Board (April, 2009). As part of the Ph.D. proposal, the FNS addressed the administrative details (in a Memorandum of Understanding) required to implement a cross-system program. The FNS has also promoted an undergraduate minor in Neuroscience (NRSC) and plans to introduce a major.

The FNS underwent an external review in the fall of 2008. Their report, stated:

- "If the new Doctoral Program receives the necessary support, there is no question that it can become a world class program and one of the leading neuroscience graduate programs in the country."
• “The success of the faculty in generating grant funds is likely due, in part, to the research areas of interest and the complementary approaches. As a group, the faculty research interests address important human health problems including aging, diseases of the nervous system, pain, recovery after injury, and addiction.”
• “We were enormously impressed with the atmosphere of collegiality among the Faculty of Neuroscience.”
• “We were also impressed by the leadership of the Faculty of Neuroscience, both past and present. It is outstanding at all levels.”
• “One strength of this effort is the apparent support of the administration. This is evident in the recent hiring successes undoubtedly supported by excellent start-up packages, construction of a new Life Sciences Building devoted partially to Neuroscience laboratories, and the strong and well-supported core facilities.”

1.2. Building Intellectual Capacity at Texas A&M

The Texas A&M Institute for Neuroscience (TAMIN) will provide an organizational structure for investigators to strengthen multidisciplinary collaborative research and enhance research funding opportunities among members. The development of a new Institute for Neuroscience will coalesce established, well-funded, investigators with a growing group of young investigators to foster innovation. The Institute, in conjunction with targeted hires and the allocation of research space in the new Interdisciplinary Life Sciences Building, will enhance the visibility of Neuroscience Research conducted at Texas A&M to national and international levels. Moreover, the establishment of a Graduate Training Program in Neuroscience will place Texas A&M among the few institutions granting degrees in Neuroscience in Texas. While students obtaining degrees in various disciplines can acquire experience in neuroscience through research, a formal degree in neuroscience will improve students’ competitiveness for employment in areas that increasingly require such a degree. Therefore, state-of-the-art infrastructure to support successful well-funded research programs and formal training in neuroscience will make Texas A&M highly attractive for additional prominent faculty, highly-skilled junior faculty and outstanding graduate and undergraduate students seeking degrees in neuroscience. The ability of Texas A&M to combine resources from diverse Colleges within a single institute will make the Texas A&M Institute for Neuroscience unique among currently established programs in the State and in the Nation.

1.3. Multidisciplinary Aspects

The field of neuroscience is fundamentally multidisciplinary and spans both animal and human research. For example, genetic defects and abnormal gene expression can alter development to produce neurological dysfunctions or increase the risk for depression or addiction. Anatomical details provide the basis for assembly of neural systems into functional circuits that are involved in these dysfunctions. Due to the complex layered nature of neural integration, computational and statistical modeling and multivariate data analysis have enabled better understanding of these circuits. Biochemical and biophysical properties of membrane channels, one of the functional units of neural transmission involved in neural dysfunction, are investigated at molecular and cellular levels to reveal targets for drugs with potential clinical use. Physical and behavioral
aspects of therapeutic applications and effectiveness of new drugs can then be evaluated. Engineering solutions for early diagnosis can assist in disease prevention.

The contribution of multiple colleges is evident from existing research programs. Molecular and cellular aspects of the nervous system are areas of investigation centered in the Colleges of Agriculture and Life Sciences, Liberal Arts, Science, and Veterinary Medicine and Biomedical Sciences, through the use of diverse biomedical models. Behavioral, affective and cognitive neuroscience are largely represented in the College of Liberal Arts and Architecture and the bioengineering of solutions is developed primarily in collaborations within the College of Engineering. Research conducted in the College of Education focuses on neuropsychology and motor learning. Partnership with the TAMHSC College of Medicine strengthens fundamental neuroscience research and the opportunity to translate basic research into clinical application. The Institute for Neuroscience will provide a bridge to promote effective collaboration among investigators. It will bring together researchers with a common interest (neural function) that specialize in one or more topic areas aimed at understanding basic neurological function with the goal of uncovering new ways to treat diseases of the nervous system.

1.4. Synergies with University and College Plans

TAMU Colleges identifying neuroscience as an area of strength include Liberal Arts (Health, Human Wellness, and Health Care), Veterinary Medicine and Biomedical Sciences (Clinical Neurology and Neurodegenerative Diseases), and Science (Biological Clocks, Neurobiology). Faculty hiring within the recent reinvestment program brought 18 new assistant professors with research programs focused in neuroscience-related areas. Support from the TAMHSC College of Medicine brought an additional 6 neuroscience faculty. More recently, TAMIN was identified as one of 8 proposals (out of 111) to be funded under the Initial University Multidisciplinary Research Initiatives (IUMRI), which will provide the resources needed for 3 senior hires and a stable base of funding for graduate and administrative support (see 1.7).

Relevance to 2020 Imperatives: Promoting Neuroscience as an institute, and enhancing our capacity to develop, will elevate our faculty and their teaching, research, and scholarship (Imperative 1 of Vision 2020). Neuroscience is seen as a priority research program that addresses issues of national and global significance. It is, inherently and by necessity, a multidisciplinary endeavor and a primary aim of this proposal is to attract "superstars" that will raise the program to the highest level. Neuroscience seeks to strengthen our graduate program (Imperative 2) with the creation of a new degree in NRSC, interdisciplinary coursework in Neuroscience, and enhanced graduate support (through both graduate stipends in Neuroscience and Heep Fellowships). Neuroscience enhances the undergraduate academic experience (Imperative 3) through the development of a minor in NRSC and through research opportunities within the laboratory. These students have been going on to the best graduate programs and medical schools in the country. Neuroscience has raised the bar on professional education (Imperative 5). Members of the FNS routinely have the strongest records of grant support and publications within their home department at the time of tenure. At the same time, Neuroscience has worked to diversify and globalize the A&M community (Imperative 6). Neuroscience faculty at TAMU and TAMHSC participate in a T32 training grant that targets under-represented groups and promotes training at both the graduate and postdoctoral level. Further, Neuroscience has done an
outstanding job of recruiting a diverse/international faculty. Members of the FNS have demanded enlightened governance and leadership (Imperative 10) and have developed and approved Policies that assure effective faculty governance. All key positions within the group are elected and policies have been enacted to protect participating members (as recommended by the External Review team [October, 2008]). We have sought, and gained, improved space (Imperative 13). Neuroscience has been allocated new space in both the Interdisciplinary Life Sciences Building and on the new TAMHSC campus.

Neuroscience aims to promote academic quality through the development of a top-tier research/training program in neuroscience that focus on areas of strength. The proposal builds on 2 decades of investment in neuroscience that has promoted the development of the FNS through the 90s and fueled its expansion during the faculty reinvestment program (hiring 18 new faculty since 2003). Further value is achieved through the consolidation of administrative components that will work to promote the area, research strengths, grant funding, and postdoctoral, graduate, and undergraduate training. Research excellence requires ties to the strongest resources, both nationally and internationally (globalization). We seek to attract the best faculty and students, both for training and for collaboration. TAMIN builds on an existing infrastructure, taking it to the next level, and using a prime resource (space within the Interdisciplinary Life Sciences Building [ILSB]) to foster interdisciplinary ties, both with members of the FNS and other areas. We promote enlightened governance through an elected chair, executive committee, and through shared governance in committee duties.

1.5. Potential Texas A&M Competitive Advantage

The Texas A&M Institute for Neuroscience will focus on building a program designed to foster new interdisciplinary ties in multiple areas of neuroscience. This feature will be important for attracting both students and faculty to the program. Further, this Institute will gain from the diverse range of applications being studied within the Colleges of Medicine, Veterinary Medicine and Biomedical Science, Science, and Agriculture and Life Sciences, that have established the use of traditional (laboratory rodents) and non-traditional (e.g., invertebrates, fish and livestock species) animal models for fundamental research with clinical implications. This breadth of potential model systems is not found in any other institution. A&M also has advantages in key areas (structural biology, biochemistry) for drug development and an engineering college that is at the forefront in the development of new technologies in drug delivery, imaging, and the application of nanotechnology.

1.6. Existing Critical Mass

The FNS is composed of 74 faculty members. Members are drawn from 9 colleges, and 20 departments, which are distributed across both TAMU and TAMHSC (see Figure 2). All members of the FNS are participants in this submission and will be affiliated with the Texas A&M Institute for Neuroscience. The unique integration between the Colleges of Medicine and Veterinary Medicine sets our program apart, not just within Texas, but also nationally. At Texas A&M, students can learn about underlying cellular mechanisms, how new procedures are evaluated within an experimental setting, and how experimental findings can be translated into clinical practice.
Faculty highlights include recent publications in top-tier journals (e.g., PNAS [Tassinary, Behmer, Li], Science [Behmer]), citation rates over the last 10 years among the top 1% worldwide (Harmon-Jones, Packard, Setlow), and multiple collaborations with groups outside A&M (e.g., Mission Connect, MD Anderson, VA Center of Excellence).

1.7. An Initial University Multidisciplinary Research Initiative (IUMRI)

In 2009, TAMIN was selected as one of eight interdisciplinary programs (out of 111) to be funded under the IUMRI. This program will provide the resources needed to hire three senior faculty, one each in Psychology, Biology, and Veterinary Integrative Biosciences/Veterinary Medicine and Biomedical Sciences. The aim is to recruit faculty at the highest level, individuals who will bring strong grant support, outstanding records of research (top quarter of 1% in citations) and national recognition (National Academy of Sciences caliber). Funding from the IUMRI will also provide a base of graduate and administrative support for the program.

2. IMPACT ON TRAINING AND EDUCATION OF STUDENTS

The Texas A&M Institute for Neuroscience will serve as the administrative home for both the graduate program in Neuroscience and the undergraduate minor. At the graduate level, students working within the area of Neuroscience at Texas A&M University and Health Science Center have obtained their degrees within particular departments (e.g., Psychology, Biology, Neuroscience and Experimental Therapeutics [NExT], or Veterinary Medicine and Biomedical Sciences [VIBS]). Over 70 students are currently working with members of the Faculty of Neuroscience. With the approval of the Ph.D. program, current second and third year students have been given the option to transfer into the Neuroscience (NRSC) program and the Graduate
Recruiting Committee is now processing these requests. In addition, we anticipate accepting 6-8 new students per year into the program. Recognizing the strength of the program, and student interest, the program was recently granted 9-10 Heep Fellowships that will be used to recruit outstanding students. Our expectation is that the NRSC program within TAMIN will, within 3-5 years, have 25-30 graduate students obtaining their degree in NRSC.

Neuroscience has also encouraged undergraduate education in two ways. First, we have established a minor in NRSC that has undergone rapid growth since its inception a few years ago (with close to 100 students now participating). Second, many students benefit from the opportunity to do independent research, and senior research projects, in the area of Neuroscience. In the future, we hope to introduce a major in Neuroscience.

Both the graduate and undergraduate program build upon courses taught across a range of departments and colleges, providing a truly multidisciplinary educational experience. Coordinating these educational endeavors requires some central administration, and that will be provided by TAMIN.

TAMIN also benefits our students in other ways. Each year, Neuroscience provide travel awards for students to present their work at the Society for Neuroscience convention. A weekly seminar series brings in nationally recognized speakers and provides an opportunity for students to interact with top researchers from Harvard, Stanford, MIT, and other top-ranked programs. Finally, each year the students organize a symposium on a topic of their choice, which features 3 prominent scientists and short talks by current trainees.

3. SPACE, INFRASTRUCTURE AND SUPPORT

Neuroscience was identified as one of three areas to occupy the new Interdisciplinary Life Sciences Building. Eight laboratories (four each) have been allocated to researchers in the areas of Biology and Behavioral and Cellular Neuroscience (Psychology) on the third floor of the building. In addition, as part of the IUMRI, the remaining laboratory space on the third floor of the Life Sciences Building has been set-aside for the senior hires in Neuroscience. These facilities will provide a central hub for the Institute for Neuroscience and its administrative home. The Institute for Neuroscience will also benefit from the space allocated to Neuroscience and Experimental Therapeutics within the College of Medicine (approximately 2/3 of the new Medical Research Building [80,000 sq. ft.] on the new TAMHSC campus.

Through the IUMRI, TAMIN has been granted a base budget that will grow to approximately $270,000 by FY14. Most of this budget is directed to graduate training (approx. $197,000). These funds are required to support students during their first year of graduate study, when they are participating in laboratory rotations. After that, the bulk of graduate funding (approx. $492,000) will be provided through departments and investigators from research (grant support) and teaching assistantships. Graduate training is also supported by 9-10 Heep Fellowships, which provide $30,000/year for the first three years of graduate training.

The remaining IUMRI funds (approx. $74,000) are used to provide student travel awards and administrative support. These funds are supplemented by TAMU college contributions of
roughly $1,000 per faculty member per year, and funds from the HSC ($32,000 in FY10), yielding a budget of $172,000. These funds are used to cover student travel ($40,000), graduate recruiting ($8,000), seminar series ($30,000), symposium ($6,000), administrative supplement ($26,000), clerical and advising ($54,000), and IT ($8,000).

Grants in Neuroscience at TAMU/TAMHSC currently total more than $50 million. Roughly 14% of the NIH grants awarded to TAMU/TAMHSC are directed to faculty members of Neuroscience (CRISP, 12/16/08). Just a fraction of the indirect costs generated by these grants will cover the TAMU/TAMHSC support for TAMIN. In addition, the FNS participate in a NIH-funded Program Project in Biological Clocks and a T32 Training Grant to foster graduate training of under-represented groups. Both the National Science Foundation and the US Department of Agriculture also fund neuroscience-related research. A variety of Foundations (e.g., Howard Hughes Medical Institute, Alzheimer’s Association, National Multiple Sclerosis Society, Christopher and the Dana Reeve Foundation) sponsor research in selected areas of neuroscience.

4. GOVERNANCE AND ADVISORY STRUCTURE

4.1 Governance

TAMIN will be overseen by an Administrative Council that includes the Council of Participating Deans, the Deans of Graduate Studies (TAMU and TAMHSC), and the Vice Presidents of Research (TAMU and TAMHSC). Because approximately 3/4 of the faculty come from four colleges (Medicine, Veterinary Medicine, Liberal Arts, and Science), the Council of Deans has been led by:

- Ben Crouch, Liberal Arts
- Joseph Newton, Science
- Van Wilson, Medicine
- Eleanor Green, Veterinary Medicine

The remaining colleges have been supportive and are kept abreast of major developments. With the continued growth of TAMIN, and greater involvement of faculty from other colleges, we anticipate increased participation on the Council of Deans.

The internal governance of the program is dictated by a set of Policies, first adopted in 1998 and last revised in 2009. An elected Chair (currently, J. Grau) provides administrative leadership and will serve as the Director of the Institute. The Chair oversees the FNS with input from the Executive Committee, composed of the chairs of each standing committee (Curriculum, Recruiting, Membership, and Seminar) and a graduate student representative. Each standing committee is composed of a Chair and 4 additional members. All positions are elected for two-year terms, staggered across years (for current membership, see FNS Committees). To encourage coordination with Department-level activities, the Chair and Executive Committee will receive input from an Advisory Committee composed of Participating Department Heads.
Membership in the FNS requires review, and approval by, both the Membership Committee and the EC. Criteria have been developed to help assure that faculty remain active (see Policies), and being an ‘active’ member is a requirement for maintaining voting privileges and access to FNS resources.

4.2 Intra-System Collaboration

The TAMIN will integrate two system components, TAMU and TAMHSC. Neuroscience faculty within TAMU and TAMHSC have a long history of collaboration. A Memorandum of Understanding has been developed, and approved, to address the coordination of the joint degree program across system components. Other ties include multiple research grants, funded through the Research Foundation, which handles the distribution of IDC across system parts. Both TAMU and the TAMHSC contribute in a proportionate manner to graduate teaching, graduate stipends, administrative support, and service to the program. To simplify program administration, TAMIN will be housed and budgeted through a single system component (TAMU), with a transfer of support from the TAMHSC.

5. PERIODIC REVIEW AND SUSTAINABILITY

5.1 Sustainability

The Society for Neuroscience is just under 40 years old and is continuing to grow at a rapid rate (see Figure 1). Why—because the interdisciplinary structure of neuroscience works and provides value. By focusing research efforts on enduring societal issues, and offering solutions that promote human health, it has grown and flourished. Properly managed, it will remain an enduring model of a successful interdisciplinary program. Will it supplant department-level expertise? No, because creative solutions draw from expertise within specific domains and traditional areas/departments continue to provide a rich background from which to gain specialized training. What neuroscience adds is the capacity to bring these together to discover new solutions to enduring problems. Only when neurological diseases are cured, and the mind and brain are understood, will the discipline begin to wane. In terms of funding potential, few areas can compare to neuroscience. First, it addresses a key criterion at NIH—the need for translational research; to bring new discoveries in biochemistry, nanotechnology, pharmacology, and structural biology to practice. Second, donors readily appreciate the importance of research designed to address fundamental issues of health, such as autism, Alzheimer’s, spinal cord injury, addiction, and sleep disorders. Beyond NIH, dozens of private foundation focus on topics within Neuroscience (e.g., Mission Connect, Howard Hughes Medical Institute, Alzheimer’s Association, and National Multiple Sclerosis Society). Third, progress in neuroscience depends on drug discovery and new engineering solutions (e.g., nanotechnology), whose application will bring new opportunities for commercialization. Developing these technologies, and demonstrating their effectiveness in treating clinical disorders, will require integration across areas, from engineering and biochemistry to translational neuroscience and clinical testing. Combined, TAMU/TAMHSC has what is needed to meet these challenges, but doing so will require effective integration. TAMIN is designed to provide that essential structure.
While it is understood that many institutes at TAMU/TAMHSC are structured with a finite life, it is not evident that the same criterion applies to neuroscience. We anticipate that, for decades to come, students will seek training in the area of neuroscience, that neuroscience will remain a central theme for funding at the National Institutes of Health, and that an effective neuroscience program will depend on the strength of its multidisciplinary ties. In the absence of an alternative university structure, TAMIN will provide an essential resource that we envision lasting well beyond 2020.

### 5.2 Institute Activities

The proposed Texas A&M Institute for Neuroscience will foster the research enterprise through the following activities:

- Promoting the recruitment and hiring of internationally recognized faculty;
- Developing interdisciplinary research projects and enhanced grant support;
- A weekly seminar featuring internationally recognized leaders in the field of Neuroscience;
- Symposia on special topics (e.g., drug addiction, Alzheimer’s, neurodegenerative diseases, neurotrauma);
- Developing Areas of Concentration that foster collaboration in areas of excellence at Texas A&M;
- Providing recognized topics for seeking donor support;
- Development and teaching of interdisciplinary courses and programs in Neuroscience, at both the graduate and undergraduate level;
- Enhancing postdoctoral training opportunities;
- Graduate recruitment and support (through increased federal funding and training grants);
- Development and enhancement of out-reach programs.

### 5.3 Goals

Over the next 5 years, we aim to:

- Hire 3 senior faculty;
- Submit NIH training grant proposals to enhance T32 support;
- Develop an undergraduate major;
- Foster the submission of NRSA/NSF graduate training applications, with the aim of having 25% of our post-first-year students supported by fellowships;
- Increase NIH funding so that Neuroscience accounts for 18% of the funds awarded to TAMU/TAMHSC;
- Increase average publication rate per faculty from 2.5 to 3 articles per year, with an increased emphasis on submissions to top-tier journals (Nature, Science);
- Enhance ties to current faculty in areas such as biostatistics and bioengineering, and promote hiring in neuroscience (to gain a 20% increase in our faculty).
5.4 Evaluation and Review

The impact of the Institute for Neuroscience will be evaluated by: 1) number of publications and their impact (journal impact and citations); 2) grant support; 3) number of patents and licenses filed and awarded; 4) number of students graduating and placed; 5) number of post-doctoral fellows trained and placed; 6) faculty awards; 7) number of undergraduate students conducting research under the mentorship of Faculty members; and 8) faculty participation in grant panels.

Neuroscience at TAMU/TAMHS has been, and will continue to be, reviewed in two ways. Each year, an Annual Report is provided to the VPRs at TAMU/TAMHSC, the Council of Participating Deans, and the Advisory Board composed of Participating Department Heads. (see http://Neuroscience.tamu.edu/Administration.html for annual for the last 3 years). Continued funding for the program is contingent upon submission of the annual review and evidence that the program has met its performance objectives.

As a graduate program, Neuroscience is subject to a review process analogous to that used to review department-affiliated Ph.D. programs. Every 5 years, the Vice President of Research at TAMU/TAMHSC will seek a list of potential external reviewers and select 3-4 to visit campus to provide a review. This review will encompass all aspects of the research and graduate program. The purview of the external review team would be similar to programs housed within a traditional department and could recommend enhanced development or discontinuation.

The Ph.D. program was last reviewed in 2008. The external review team was composed of Rebecca Burwell (Brown University), Cedric Williams (University of Virginia), and Michael Zigmond (University of Pittsburgh). Both the review and the TAMU/TAMHSC response are available at http://Neuroscience.tamu.edu/Administration.html.
List of Neuroscience Faculty
(Name, Title, Department, Areas of Expertise)

All 74 members of the FNS are participants on this proposal. All individuals are members of the graduate faculty. Membership requires review and approval by both the membership Committee and the Executive Committee. To assure that all members remain active participants, the Membership Committee reviews the current membership on an annual basis.

The distribution of faculty by position is as follows: 34% full Professor, 32% Associate Professor, 31% Assistant Professor, 1.4% Research Assistant Professor and 1.4% Clinical Assistant Professor.

Louise Abbott, Associate Professor, Veterinary Integrative Biosciences
Developmental neurobiology, neurotoxicology

Marcel Amstalden, Assistant Professor, Animal Science
Neuroendocrine regulation of puberty and estrous cyclicity

Rafael Ballesteró, Associate Professor, Biology, TAMU-Kingsville
Nerve regeneration, apoptosis

Spencer Behmer, Assistant Professor, Entomology
Nutritional physiology, learning, and behavioral ecology

Luc Berghman, Associate Professor, Poultry Science
Neuroimmunology

Gregory Bix, Assistant Professor, Molecular and Cellular Medicine
Neurovascular function and stroke, extracellular matrix

Jennifer Bizon, Assistant Professor, Psychology
Aging, neurogenesis

Paul Brandt, Associate Professor, Neurosci. and Exp. Therapeutics
Hormones, calcium signaling

Gerald Bratton, Professor, Veterinary Integrative Biosciences
Neurotoxicology, reproductive/neuroendocrine function

John Buchanan, Associate Professor, Health and Kinesiology
Motor learning and performance in humans

Ginger Carney, Assistant Professor, Biology
Genes and behavioral/neural dev., reproductive beh. in drosophila

Antonio Cepeda-Benito, Professor, Psychology
Drug addiction, nicotine

Wei-Jung Chen, Associate Professor, Neurosci. and Exp. Therapeutics
Drug abuse, alcohol, brain/cognitive development

Yoonsuck Choe, Associate Professor, Computer Science
Neural networks, computational neuroscience

Suzette Chopin, Professor, Life Sci., TAMU-Corpus Christi
Efficacy of herbal remedies

Evangelos Christou, Assistant Professor, Health and Kinesiology
Neuromuscular mechs. and motor performance in humans

Tim Cudd, Professor, Vet. Physiology & Pharmacology
Neural development, alcohol
Les Dees, Professor, Veterinary Integrative Biosciences
  Neuroendocrinology
David Earnest, Professor, Neurosci. and Exp. Therapeutics
  Cellular/molecular mechanisms underlying biological clocks
Shoshy Eitan, Assistant Professor, Psychology
  Emotional development, opiates
Richard Finnell, Professor, IBT, Houston – TAMHSC
  Neurotoxins and the genetic control of embryonic development
Jonathan Friedman, Associate Professor, Neurosci. and Exp. Therapeutics
  Neural regeneration, spinal cord injury
Gerald Frye, Professor, Neurosci. and Exp. Therapeutics
  Alcohol neuropharm., neurodev, disorders, electrophysiology
Luis R. Garcia, Associate Professor, Biology
  Cellular and molecular regulation of motivated behaviors
Lisa Geraci, Assistant Professor, Psychology
  Memory and aging
Maribel Gonzalez-Garcia, Associate Professor, Chemistry, TAMU-Kingsville
  Regulation of apoptosis
James Grau, Professor, Psychology
  learning, spinal cord plasticity
William Griffith, Professor, Neurosci. and Exp. Therapeutics
  Neuropharmacology of aging, calcium signaling
Paul Hardin, Professor, Biology
  Molecular genetics of biological clocks
Eddie Harmon-Jones, Professor, Psychology
  Social neuroscience, psychophysiology
Paul Harms, Professor, Animal Science
  Reproduction, neural control of ovarian function
Michelle Hook, Research Assistant Prof., Psychology
  Recovery of function, spinal plasticity, drug addiction
Rachel Hull, Instructor Asst. Prof., Psychology
  Language dev., aging, neuropsychology
William Klemm, Professor, Veterinary Integrative Biosciences
  Learning and memory, educational neuroscience, cognition
Gladys Ko, Assistant Professor, Veterinary Integrative Biosciences
  Circadian reg. of ion channels, signal trans., synaptic plasticity
Kathryn Kotrla, Associate Professor, Psychiatry & Behavioral Sciences
  Neuroimaging, neural networks, behavioral neuroanatomy
Julian Leibowitz, Professor, Microbial & Mol. Pathogenesis
  Neuroimmunology, coronaviral infection, multiple sclerosis
Jianrong Li, Assistant Professor, Veterinary Integrative Biosciences
  Neuron glial interaction, oligodendrocyte dev. & cell death
Robyn Lints, Assistant Professor, Biology
  Genetic control of neural dev. & function, mating beh. (c. elegans)
Thierry Lints, Assistant Professor, Biology
  Molecular basis of song imitation in birds (zebra finches)
Enrique Massa, Associate Professor, Biology, TAMU-Kingsville
  Ion channels, neurobiol. actions of ethanol & inhaled organic solvents
U. Jack McMahlan, Full Professor & Head, Biology
  Molecular basis of synaptic transmission
Mary Meagher, Professor, Psychology
  Psychoneuroimmunology, multiple sclerosis, pain
Rajesh Miranda, Associate Professor, Neurosci. and Exp. Therapeutics
  Fetal brain development, microRNAs, stem cells, alcohol
Ian Murray, Assistant Professor, Neurosci. and Exp. Therapeutics
  Aging, protein misfolding, mass spec., Parkinsons, Alzheimers
Mark Packard, Professor, Psychology
  Neurobiological basis of memory
Vladislav Panin, Associate Professor, Biochemistry and Biophysics
  Developmental biology, glycosylation and cell interactions
Brian Perkins, Assistant Professor, Biology
  Genes and retinal development (zebrafish)
Michelle Pine, Clinical Assistant Professor, Veterinary Integrative Biosciences
  Reproductive/neuroendocrine function, neurotoxicology
Samba Reddy, Associate Professor, Neurosci. and Exp. Therapeutics
  Epilepsy, neurosteroids and new drug development
Cecil Reynolds, Professor, Educational Psychology
  Psychological assessment (memory, emotion, cultural bias)
Cynthia Riccio, Professor, Educational Psychology
  ADHD, learning disabilities, neuropsychology
Mendell Rimer, Assistant Professor, Neurosci. and Exp. Therapeutics
  Synapse formation/maintenance, developmental neurobiology
Gil Rosenthal, Assistant Professor, Biology
  Evolutionary genetics of mating behavior
Barry Setlow, Assistant Professor, Psychology
  Learning and memory, decisionmaking, drug addiction
Charles Shea, Professor, Education & Human Development
  Motor behavior
Michael Smotherman, Assistant Professor, Biology
  Neurobiol. animal communication, echolocation in bats
Farida Sohrabji, Associate Professor, Neurosci. and Exp. Therapeutics
  Women's health/aging, hormones, stroke, blood brain barrier function
Ian Steele-Russell, Professor, Psychiatry & Behavioral Sciences
  Visual plasticity, learning & memory, recovery of function
George Stoica, Professor, Veterinary Pathobiology
  Mechanisms of retroviral-induced neurodegeneration
Ralph Storts, Professor, Veterinary Pathobiology
  Comp. neuropathology, neurodeg. and demyelinating diseases
Louis Tassinany, Professor, Visualization
  Psychophysiology, person perception
Evelyn Tiffany-Castiglioni, Professor, Veterinary Integrative Biosciences
  Neurotoxicology, glial biology
Gerard Toussaint, Assistant Professor, Neurosci. and Exp. Therapeutics  
Glioma biology

Jyotsna Vaid, Professor, Psychology  
Language, neuropsychological aspects of bilingualism

Paul Wellman, Professor, Psychology  
Behavioral pharmacology, feeding, drug addiction

Gregg Wells, Associate Professor, Molecular and Cellular Medicine  
Protein structure and neurological disease, ion channels

Robert Wells, Professor, IBT, Houston – TAMHSC  
Genetic instabilities and neurological disease

Jane Welsh, Professor, Veterinary Integrative Biosciences  
Neuroimmunology, animal models of multiple sclerosis

Teresa Wilcox, Associate Professor, Psychology  
Infant development, neuroimaging

Ursula Winzer-Serhan, Associate Professor, Neurosci. and Exp. Therapeutics  
Nicotine, nicotinic receptors, and brain development

Keith Young, Associate Professor, Psychiatry & Behavioral Sciences  
Biol. basis mental illness: PTSD, depression, schizophrenia, autism

Danna Zimmer, Associate Professor, Veterinary Pathobiology  
Pathobiology of neurological disorders, calcium signaling

Mark Zoran, Associate Professor, Biology  
Dev. neurobiology, synaptic plasticity, biological clock
APPENDIX F

TAMIN Budget
March 11, 2010

Dr. James W. Grau  
Professor of Psychology and Mary Tucker Currie  
Professor of Liberal Arts  
Department of Psychology  
Texas A&M University  
4235 TAMU  
College Station, TX 77843-4235

Dear Dr. Grau:

Thank you for developing an implementation plan for Texas A&M Institute for Neuroscience (TAMIN). The Office of the Vice President for Research (VPR) has high expectations for the success of this initiative, and we want to be supportive of the initiative in the coming years as new faculty are hired and as programming unfolds. To move this initiative forward, this letter outlines the process for (1) hiring faculty using the funding allocated to TAMIN and (2) creating operating accounts to support programs by the initiative. Our office will assist you as the initiative moves forward. Please do not hesitate to call upon us during this process.

Faculty Hires

The Texas A&M Institute for Neuroscience is authorized to begin planning for new faculty hires as presented in the attached budget. These hires should take place during the fiscal years noted in the budget. As convener, please follow these enumerated procedures as you initiate the hiring process and make final recommendations for hiring new faculty:

1. Prepare a hard-copy memorandum requesting authorization for the search (or for a specific targeted hire). The memorandum should be directed as follows:

   To: Dr. Karan L. Watson  
      Interim Provost and Executive Vice President for Academics  

   Through: Dr. Jeffrey R. Seemann  
      Vice President for Research  

   Through: [The dean of the college in which the new position is being created.]  

   Through: [The head of the department in which the new position is being created.]  

   From: [The director or convener for the initiative.]  

   cc: Dr. Antonio Cepeda-Benito, Dean of Faculties and Associate Provost
2. The memorandum should provide a description of the search process, including:
   a. A description of the position
   b. The names of the individuals on the search committee
   c. A description of the search process for candidates
   d. A detailed description of efforts undertaken in the search to assure that candidates from underrepresented groups are (or were) considered for the position
   e. The estimated or targeted start date for the new hire

3. The memorandum should also include information about the position, including:
   a. Estimated salary for the new hire and the sources of funding, including allocated funding from the TAMIN budget and any supplemental funding from departments or colleges
   b. Space or laboratory needs for the position and who will provide these facilities
   c. Estimated start-up funding required for the hire

4. During the interview stage, please make arrangements with my office to schedule time for me to visit the candidates. I would like to underscore the importance of the Texas A&M Institute for Neuroscience and make a personal connection with the proposed senior hires.

5. When an offer is contemplated, the approval to hire memorandum and associated forms should be routed through the same individuals who approved the initial search as described above in item 1 and the Dean of Faculties. This memorandum should include the information normally required for making a senior hire (e.g., supporting documentation for arrival with tenure and DOF-required information for faculty hires). Additionally, final information must be provided regarding salary commitments (with college or departmental account numbers if applicable) and other start-up commitments being made to the candidate.

Operating Expenses for TAMIN

The Vice President for Research will allocate funding to support operating expenses identified in the attached budget. For the remainder of this fiscal year, the VPR’s office will administer the account. Please contact Ms. Annette Shenkir for information about our office’s fiscal policies and processes. We will make arrangements with you or a designated individual for administration of these operating expenses in FY11 and in subsequent years.

Follow-Up Reports from TAMIN

To gain a sense of the initiative’s progress, we will expect an annual report on activities and achievements by the Texas A&M Institute for Neuroscience. This report would include progress on faculty searches and hires, major initiatives and projects undertaken by the initiative, and high-impact successes in research, scholarship, or creative work. Supplemental information about impacts on graduate and undergraduate education will also be very important. As you prepare your annual report, please be mindful of the benchmarks concerning progress of the initiative as provided in your implementation plan. Our office will send you a request for this report in the 2011 spring semester.
Dr. James W. Grau  
March 11, 2010  
Page 3

Thank you for your support and cooperation as we move into the implementation phase of the Research Roadmap Committee’s recommendations. I join members of the committee and the entire Texas A&M University research community in wishing you much success.

Sincerely,

[Signature]

Dr. Jeffrey R. Seemann  
Vice President for Research

Attachment

cc:  Dr. Karan L. Watson  
Dr. Antonio Cepeda-Benito  
Dr. Charles A. Johnson  
Dr. G. Kemble Bennett  
Dr. Ben M. Crouch  
Dr. Eleanor M. Green  
Dr. Mark A. Hussey  
Dr. H. Joseph Newton  
Dr. Douglas J. Palmer  
Dr. Jorge Vanegas
Summary of Implementation Plan for the Texas A&M Neuroscience Institute

Convener(s): Dr. James W. Grau, Professor of Psychology and Mary Tucker Currie Professor of Liberal Arts

Deans: Dr. G. Kemble Bennett, Dean, The Dwight Look College of Engineering
Dr. Ben M. Crouch, Interim Dean, College of Liberal Arts
Dr. Eleanor M. Green, Carl B. King Dean of Veterinary Medicine
Dr. Mark A. Hussey, Vice Chancellor and Dean for Agriculture and Life Sciences
Dr. H. Joseph Newton, Dean, College of Science
Dr. Douglas J. Palmer, Dean, College of Education and Human Development
Dr. Jorge Venegas, Dean, College of Architecture

New Faculty Hires:

Three senior hires will be accomplished as soon as possible. The hires will be in the Departments of Psychology, Biology, and Veterinary Integrative Biosciences. The Colleges of Liberal Arts, Science, and Veterinary Medicine will provide 25% top-up funding for each of their respective hires. Start-up costs for each hire will be negotiated individually with the appropriate department, college, and the Vice President for Research (VPR), with the understanding that the VPR will make major contributions to the start-up packages and that the VPR contribution will not exceed $1 million per hire. Start-up costs may include the costs for completion of some laboratory space.

Space and Facilities:

Each of the three hires is projected to occupy space on the third floor of the Interdisciplinary Life Sciences Building. Finishing of currently shelled space on the third floor will be funded by a combination of monies from the Vice President for Research, respective college funds, and other university sources, with the understanding that VPR funding will be substantial.

Additional Commitments Supplemented University Funding:

Participating colleges will provide additional funding for graduate assistantships, administrative staff, and other operating expenses or special requests as outlined in the attached budget spreadsheet.

Budget Allocation for FY10 – FY14

See attached budget sheet.

Governance and Administrative Oversight:

The chair of the Neuroscience Faculty will serve as director of the Texas A&M Neuroscience Institute and will have management responsibility for the institute. The institute will have a council of participating deans (COPD) to include the deans of Agriculture and Life Sciences, Architecture, Education and Human Development, Engineering, Liberal Arts, Science, and Veterinary Medicine and Biomedical Sciences. The VPR or his designate will also serve on the COPD. Provisions will be made for involving the College of Medicine and other interests in the Texas A&M Health Science Center.
Markers for Success and Achievement:

1. Neuroscience currently accounts for approximately 14% of the NIH funds awarded to Texas A&M University and the Health Science Center. Our goal over the next five years is to increase this to 18%. This will require increasing net support by 28%, from $50 million to $64 million.
2. Scholarship among current neuroscientists is strong, with an average of 2.5 articles per year. In five years, we aim to move this average to three articles per year, with an increased emphasis on reaching the top-tier journals (e.g., *Nature, Science, Neuron, Journal of Neuroscience*).
3. Our graduate program should have nearly 25 students within five years (perhaps more, if funding allows) and approximately 80-160 undergraduate minors. Within five years, we will offer a new undergraduate major in Neuroscience.
4. Top-tier research programs maintain a high proportion of postdoctoral trainees. Over the next five years, we hope to increase the number of post docs by 36%, from 22 to 30.
5. College- and department-level support will be demonstrated through both their continued financial support of the program and through increased hiring in the area of Neuroscience. Fueled by an agreement with the VPR to provide increased support for start-up costs, we envision continued hiring at the assistant professor level. This, coupled with our efforts to reach out to additional faculty in areas such as bioengineering and biostatistics, should bolster our faculty by 20% by 2015.
### WORKSHEET FOR TEXAS A&M INSTITUTE FOR NEUROSCIENCE (TAMIN)

#### FACULTY HIRE BASE FUNDING BUDGET REQUEST FROM RESEARCH ROADMAP FUNDS

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<th>Initial UMRFs</th>
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<th>FY11</th>
<th>FY12</th>
<th>FY13</th>
<th>FY14</th>
<th>Total Hires</th>
<th>Allocation</th>
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#### OPERATING AND OTHER BUDGET REQUEST FROM RESEARCH ROADMAP FUNDS

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<th>Initial UMRFs</th>
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<th>FY11</th>
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<th>FY14</th>
<th>Allocation Target by FY14</th>
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#### FUNDS REQUESTED FROM OTHER SOURCES OF FUNDS

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<td>Faculty Startup - Junior Hires</td>
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<td>Rent, Travel</td>
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APPENDIX G

TAMIN Policies
I. Description and Purpose

The Texas A&M Institute for Neuroscience (TAMIN) is composed of the Faculty of Neuroscience at Texas A&M University (TAMU), an interdisciplinary faculty representing numerous departments/colleges within the University.

The purpose of the Faculty of Neuroscience at TAMU is to provide a multidisciplinary opportunity for undergraduate, graduate and postgraduate education and to stimulate research in the various sub-fields of the neurosciences represented at the University. Specific goals include:

1. provide interdisciplinary training in Neuroscience and to confer the graduate degrees of Master of Science and of Doctor of Philosophy in Neuroscience (The Neuroscience Graduate Program).
2. continue to develop the graduate neuroscience curriculum at TAMU.
3. initiate efforts to obtain increased private sector and public financial support for training and research programs of the Faculty of Neuroscience at the local, state and national levels.
4. enhance the recruitment of high caliber graduate and post-graduate students into the neurosciences.
5. continue to encourage the recruitment of high quality neuroscientists as faculty at TAMU.
6. identify and expand neuroscience research collaborations at TAMU.
7. develop a neuroscience curriculum for undergraduate students at TAMU as a foundation for the establishment and maintenance of an undergraduate major in Neuroscience.

II. Membership

A. Full Membership in the Faculty of Neuroscience requires:
   1. membership on the Graduate Faculty of TAMU.
   2. willingness and capability to supervise Ph.D. candidates in the Program.
   3. willingness and capability to teach courses related to the Neurosciences Graduate Program
   4. willingness to participate on Standing Committees and regularly attend general faculty meetings of the Faculty of Neuroscience (see Meetings below).
   5. submission of materials required for the annual review of the program.
Additional criteria that may be considered by the Membership Committee include participation in the Society for Neuroscience (at the national and local level) and the maintenance of an active research program in an area relevant to neuroscience.

B. **Adjunct Membership** is available to those with or without appointment in an academic unit of TAMU.

C. New members will be recommended by the Membership Committee and approved by majority vote of the Executive Committee of the Faculty of Neuroscience.

D. Membership in the Interdisciplinary Faculty of Neuroscience does not constitute an official appointment with TAMU.

E. All postdoctoral trainees and graduate students whose primary mentor is an active member of the Faculty of Neuroscience, and who are conducting research relevant to neuroscience, are considered student members of the faculty.

F. Members with significant teaching and/or service contribution to the Faculty of Neuroscience will receive letters documenting their contribution. The letters will be based on the annual membership reviews (conducted in the spring semester by the Membership Committee). Letters prepared by the Chair will be reviewed by the Membership Committee and distributed by April 30th to each faculty member, their department head and college dean. Service and teaching contributions to the Faculty of Neuroscience will be recognized according to the respective guidelines for interdisciplinary programs developed within TAMU.

### III. Organizational Framework

A. **Chair of the Faculty of Neuroscience**
   1. The chief administrative officer of the Faculty of Neuroscience and TAMIN will be designated the “Chair”.
   2. The responsibilities of the Chair of the Faculty of Neurosciences include:
      a. the general administration of the Faculty and Institute, including ex officio membership on all standing committees; chairing of all Faculty of Neuroscience meetings; distribution of meeting minutes to all members of the Faculty of Neuroscience within three weeks after approval by the Executive Committee.
      b. the development of the Neuroscience Graduate Program including student recruitment, curriculum, advising and examination (with the assistance of the appropriate standing committees).
      c. the maintenance of archival records of student performance and faculty service.
      d. the appointment of ad hoc committee members which must be approved by the Executive Committee.
e. the active solicitation of financial aid and other resources for Faculty of Neuroscience programs from departments, colleges, the University and other appropriate sources and approval of all expenditures of TAMIN/Faculty of Neuroscience funds.

f. supervision of TAMIN support staff.

B. The Vice Chair of the Faculty of Neuroscience will be elected annually by the Executive Committee (at the first meeting after September 1) from its membership. The Vice Chair will serve as chief officer of the Faculty of Neuroscience in the absence of the Chair or when designated by the Chair.

C. The Program Coordinator for TAMIN will serve as the Secretary of the Faculty of Neuroscience. The Secretary will take attendance and minutes at faculty meetings, distribute the minutes to the faculty, and assist the Chair in maintaining an archive of Faculty of Neuroscience business and the recording of faculty votes.

D. The Executive Committee will consist of eight faculty elected from the full members of the Faculty of Neuroscience. These include the Chair of the Neuroscience Faculty and the Chairs/co-Chairs of the six standing committees. These Executive Committee members will be elected with a specific responsibility to chair (or co-chair) a specific standing committee (see below, Membership, Recruiting, Graduate Program, Undergraduate Program, Finance or Seminar Committees). In addition, the current President of the local chapter of the Society for Neuroscience and one graduate student elected by neuroscience graduate students will serve as voting members. The Committee will advise the Chair of the Faculty of Neuroscience concerning the direction and administration of the TAMIN, the associated Faculty of Neuroscience, and the graduate/undergraduate programs.

E. Elections and Terms of Service

1. The Chair of the Faculty of Neuroscience will provide a list of positions to be filled to the Membership Committee by April 15th. The Committee will recommend two or more candidates, who have agreed to serve, for each vacancy. Further nominations may be made from the floor at the annual meeting of the Faculty of Neuroscience, held at the end of the spring semester. No individual can serve on more than two standing committees nor chair more than one standing committee, respectively. The Chair cannot concurrently serve as a department head, college dean, associate head/dean, or other higher-level office. Should this occur, the Chair will step down and the vice-Chair will assume the position of Chair for the remainder of the term.

2. Elections of faculty to the Executive Committee and standing committees will be conducted by ballot to be distributed to all Full Members of the Faculty of Neuroscience immediately after the fall semester meeting by May 30. Each member can vote for one candidate
for each position to be filled. The individual receiving the largest fraction of the total vote for each position will be elected to that post (note: the graduate student representative to the Executive Committee will be elected by graduate students in the Neuroscience Program). Votes will be tallied by the Executive Committee. The Chair of the Faculty of Neuroscience will not vote unless it is necessary to resolve a tie. Election results will be mailed to all Faculty of Neuroscience members and graduate students promptly.

3. The Chair of the Faculty of Neuroscience, chairs of standing committees, standing committee members and the graduate student representative to the Executive Committee will serve two-year terms. They may be re-elected. Elections to fill the positions of Chair of the Faculty of Neuroscience, TAMU chair of the Graduate Program, Undergraduate Program, and Seminar Committees, two members of each standing committee and the graduate representative will be held on odd years, and the remaining positions will be elected in even years. Elected members will assume their duties on September 1.

4. Vacancies that occur on the Executive Committee or standing committees between elections will be filled through an appointment made by the Chair of the Faculty of Neuroscience that is approved by a majority of the Executive Committee.

F. Meetings and Policy Decisions

1. Meetings of the Faculty of Neuroscience will be called by the Chair, or at the request of a standing committee chair. The frequency of meetings should be at least once per semester. A quorum will consist of half of the active membership plus one. For purposes of the quorum, the active membership consists of those full members in regular attendance (defined as not missing three successive meetings). Only active members are eligible for committee assignments and to receive Faculty of Neuroscience resources (e.g., to host colloquia speakers, graduate support [stipends or travel awards]).

2. Academic and Operational Policies will be decided at meetings of the Faculty of Neuroscience. Proposals for policy changes may be made by the Chair, standing committees or individual members (including student representatives). Policy proposals should be acted upon, first, by the appropriate standing or ad hoc committee, then by the Executive Committee before being brought to a vote by the Faculty of Neuroscience. Written notification of proposed policy changes will be provided to the Faculty of Neuroscience at least two weeks before the meeting at which the change is to be voted upon. Policies can be changed by a two thirds majority vote of a quorum of the full members of the Faculty of Neuroscience. Votes that do not require a change in policies can be administered electronically. Again, written notification must occur two weeks prior to the vote and a quorum is defined as stated above.
G. Standing Committees
   1. Membership Committee
      a. The Membership Committee will consist of a Chair and 4 additional members elected by the Faculty of Neuroscience. These individuals should be representative of the major areas of neuroscience at TAMU. The Committee Chair will serve on the Executive Committee of the Faculty of Neuroscience. At least three members of the Membership Committee will not be members of the Executive Committee.
      b. The Committee will examine the credentials of faculty wishing to become members of the Faculty of Neuroscience and make recommendations for full or adjunct membership.
      c. The Committee will make recommendations concerning removal of members from the Faculty of Neuroscience.
      d. The Committee will recommend two or more faculty candidates, who have agreed to serve, for each vacant standing committee position or for the Chair of the Faculty of Neuroscience.
      e. The Committee will solicit nominations for the graduate representative to the Executive Committee.
      f. The Committee will review the membership annually to assure that records are up to date and that faculty members continue to meet the criteria for membership. At the start of the spring semester, the Chair will distribute a request to the faculty for materials to be used for both the Annual Report and for review by the Membership Committee. These materials should be directed to the Chair of the Membership Committee and will be due by February 1st. At a minimum, these materials should include an up-to-date biosketch (NIH format), a separate list of interdisciplinary projects, grants, and papers from the last year, a list of predoctoral/postdoctoral trainees, and a summary of teaching/service contributions to the Faculty of Neuroscience. Submission of these materials is required to remain an active member of the faculty. Faculty that have made significant service/teaching contributions will receive letters from the Chair acknowledging their contribution. It is recognized that an individual’s service/teaching contribution will vary across years and that some active members who are willing and capable of contributing may not have the opportunity to do so.
      g. Membership review may be initiated if a faculty member fails to submit the required materials and/or remains inactive for two or more years. The Membership Committee will forward the names of any individuals who fail to meet the criteria for membership to the Executive Committee. If the Executive Committee upholds the decision, the Chair of Neuroscience
will inform the faculty member that questions have been raised regarding their continued participation and provide the faculty member two weeks to respond. The response may involve submission of missing materials, explanation of continued involvement, or withdrawal of membership. Upon further review by the Membership Committee, a recommendation for dismissal can be made to the Executive Committee, which must confirm the decision.

2. Graduate Recruiting Committee
   a. The Recruiting Committee will be composed of a Chair and 6 additional members elected by the Faculty of Neuroscience. These individuals should be representative of the major areas of neuroscience at TAMU. The Committee Chair will serve on the Executive Committee of the Faculty of Neuroscience.
   b. The Recruiting Committee will advertise the training program, screen applicants and make recommendations for admission or rejection of applicants. Further, the Recruiting Committee will make recommendations concerning initial stipend awards from Faculty of Neuroscience Resources.

3. Graduate Program Committee
   a. The Graduate Program Committee will include two co-Chairs (one from TAMU and one from the HSC) and 4 additional members elected by the Faculty of Neuroscience. These individuals should be representative of the major areas of neuroscience at TAMU. The Committee Chair will serve on the Executive Committee of the Faculty of Neuroscience.
   b. One co-Chair will be elected from the TAMU faculty and will serve as the Graduate Advisor for TAMU students in the Neuroscience program. The other co-Chair will be elected from the HSC faculty and will serve as the Graduate Advisor for HSC students in the Neuroscience program. Each chair will serve as the representative for the graduate Neuroscience program within their system component, assuming the duties outlined for that position within the program proposal (including participation in TAMU graduate curriculum committees).
   c. The Graduate Program Committee will make recommendations concerning course selection, scheduling and staffing relevant to the graduate neuroscience curriculum. In addition, it will make recommendations concerning development and approval of new courses; will monitor and evaluate courses, using student and faculty feedback; and will recommend modification or deletion of courses.
   d. The Committee will evaluate graduate student performance and make recommendations concerning student progress.
e. The Committee will recommend Faculty representatives on an annual basis to serve as Examiners in the Doctoral Comprehensive Examination (see Curriculum).

4. Undergraduate Program Committee
   a. The Undergraduate Program Committee will include a Chair and 4 additional members elected by the Faculty of Neuroscience. Because the undergraduate program must be administered through TAMU, the chair and committee members must be from the TAMU faculty. The Committee Chair will serve on the Executive Committee of the Faculty of Neuroscience.
   b. The Committee will make recommendations concerning degree requirements, scheduling and staffing relevant to the undergraduate neuroscience curriculum. In addition, it will make recommendations concerning development and approval of new courses; will monitor and evaluate courses, using student and faculty feedback; and will recommend modification or deletion of courses.
   c. The Committee will oversee the undergraduate minor and develop the undergraduate curriculum with the goal of establishing a neuroscience major at TAMU.

5. Seminar Committee
   a. The Seminar Committee will include a Chair and 1 Vice Chair elected by the Faculty of Neuroscience. These individuals should be representative of the major areas of neuroscience at TAMU. The Committee Chair will serve on the Executive Committee of the Faculty of Neuroscience.
   b. The Seminar Committee chair will solicit the names of potential speakers or topics of interest from members of the Faculty of Neuroscience; organize a series of formal seminar presentations; and work with the Vice Chair to insure smooth transition once two year term is complete.
   c. The Seminar Committee Vice Chair will be expected to serve as the Seminar Committee Chair once the two year term is complete; assist Chair in soliciting names of potential speakers or topics of interest from the members of the Faculty of Neuroscience; assist Chair in organizing the seminar presentations.

6. Finance Committee
   a. The Finance Committee will include a Chair and a Vice Chair elected by the Faculty of Neuroscience. These individuals should be representative of the major areas of neuroscience at TAMU. The Committee Chair will serve on the Executive Committee of the Faculty of Neuroscience.
   b. The Finance committee will review the TAMIN budget and advise the Chair and Executive Committee on all financial
matters. The Finance Committee will also seek to identify new sources of funding, through university, federal, and private sectors, both to foster research and support graduate training.

H. *Ad Hoc* Committees

The Faculty of Neuroscience Chair, with the approval of the Executive Committee, may appoint *Ad Hoc* Committees for specific purposes and for limited terms. These committees will report directly to the Chair who will then present the results of said committees to the Executive Committee and to the Faculty as a whole.
APPENDIX H

Duties of EC
Administrative Positions
EC Reviewed (Spring/Summer, 2011)

Chair of Neuroscience
- general admin. of the Faculty and Institute
- w/Admin. Coordinator, assures effective distribution of meeting materials
- oversees standing/ad hoc committees, to assure effective student recruitment, curricula, and program implementation
- works w/Admin. Coordinator to maintain archival records
- appoints ad hoc committees
- solicits financial resources to maintain the program and oversees expenditures
- serves as Chair of the Executive Committee
- w/Admin. Coordinator, assures completion of Univ./HSC paperwork associated with the implementation of the undergrad. and grad. program
- coordinates and submits yearly reports
- works to maintain effective faculty representation and coordinates activities/decisions through the EC
- recognizes TAMIN faculty service and provides letters of support as requested
- works with vice-Chair and Chairs of Oversight and Finance to promote large-scale funded projects (training and program projects)
- works with vice-Chair and Chairs of Oversight and Membership to promote develop/promote program strengths

vice Chair of Neuroscience
- serves as chief officer when Chair is not available
- in addn., represents other System component and helps to coordinate activites across TAMU/TAMHSC
- serves as vice Chair of the Executive Committee
- helps to coordinate external and internal reviews
- works with Chair of Neurosci., Oversight and Finance to develop large-scale funded projects (training and program projects)
- works with vice-Chair and Chairs of Oversight and Membership to promote develop/promote program strengths

Graduate Advisors
- serve as graduate advisors at TAMU/TAMHSC
- chair Graduate Program Committee
- chairs serve as the chair of the IDP and coordinate the implementation of the graduate program through graduate studies at the HSC and TAMU
- works with Neurosci. Chair to assure effective compensation for NRSC graduate teaching, to the program and participating faculty
- chairs serve as grad. advisor for students in their first year
- assists students in the development of initial degree plan and rotation schedule
- works to assure effective placement of students after their first year
- works with Admin. Coordinator to assure sufficient seats are available for NRSC grad. students in cross-listed courses
Undergraduate Advisor
- serves as undergraduate advisor at TAMU
- chairs the Undergraduate Program Committee
- represents undergrad. program at the University level
- works with Neurosci. Chair to assure effective compensation for Undergraduate teaching, to the program and participating faculty
- works with Admin. Coordinator to assure effective communication with NRSC undergrads. and participation in NRSC sponsored events (e.g., colloquia)

Graduate Student Representative
- represents trainee concerns on the Executive Committee
- coordinates the nomination and election of the Graduate Student Representative and the election of the Symposium Committee
- coordinates the Annual Neuroscience Symposium

SFN Chapter President
- chairs Oversight Committee
- develops and coordinates an Internal Advisory Council, to promote effective communication with Deans, Heads, and key administrators
- promotes the dev. of large-scale projects (training grants/program projects)
- serve as a liaison (in collaboration with Chair of NRSC) to VPRs and COPD re. program development

Administrative Coordinator
- takes minutes/attendance at faculty meetings
- distributes minutes
- works w/Chair to main archival records
- coordinates/tracks the posting of web material to assure that it remains up to date
- serves as undergrad. advisor, assisting students with courserelated issues and degree requirements
- works with Undergrad. Advisor to assure effective program implementation
- directs undergrad. to appropriate resources (e.g., for res. opportunities/acad. advising)
- works with graduate students to assure access to coursework, appropriate scheduling, and effective coordination with Graduate Advisors/home depts.
- works with seminar/colloquia committee to assure effective speaker scheduling
- works with membership committee to assure records are up to date
- works with finance committee to assure accurate record keeping
- works w/program Chair to assure that all TAMU/HSC paperwork/reports are completed in a timely manner
- assists the Chair in coordinating other TAMIINrelated activities
- assists ad hoc committees, as needed
- coordinates submission/posting of financial matters through FAMIS
Standing Committees

Finance
- reviews TAMIN budget
- advises re. expenditures
- seeks to identify new sources of funding through univ., federal, and private sectors
- work to foster research and graduate support
- works w/Foundation to develop potential donors (to match Heep funds)
- develops strategies to foster collaborative research projects and training grants
- works w/Chair of Neurosci. to assure faculty and program is fairly compensated for grant-related activities

Graduate Program
- coordinates curriculum
- makes recommendations re. new courses and coordinates cross-listing of graduate courses
- monitors curriculum and course effectiveness
- makes recommendation re. termination in the program
- oversees the implementation of core courses in Neurosci. (e.g., Principles of Neuroscience)

Graduate Recruiting
- advertises the training program
  - assures material on web (local and at SFN) is up-to-date
- screens applicants
- makes recommendations for admission/rejection into the program
- coordinates the annual recruiting visit
- works to assure that all faculty have an opportunity to recruit thru Neurosci.
- implements effective strategies for assuring a high rate of acceptance
- works to increase the number of high quality applicants
- works to assure a diverse program (gender and ethnicity)

Membership Committee
- reviews those wishing to become members of Neurosci.
- makes recommendations re. removal of indiv. from the FNS
  - forwarded to EC w/reasons & recommendation
- maintain records for individual members
  - biosketch, current funding, publications, trainees (grad. & postdoc), service
- coordinates FNS elections
- tracks graduate membership and placement
- administer FNS travel awards
- promotes postdoc training/integration/placement
- assures that listing of faculty interests/ties are accurate and up-to-date
- monitor/promote dev. of cohesive units/areas of concentration, with indices of area strength (e.g., publications, grant activity)
• works with Admin. Coordinator to assure that web material for above is up-to-date
• works with Admin. Coordinator to assure that student participation in TAMIN activities (e.g., colloquia series)

Oversight (proposed, ad hoc [till Policies are revised]; ? SFN chpt. redefined)
• reviews/develops effective synergies (Areas of Concentration)
  ○ in collaboration with the Membership Committee
• assures faculty are protected and credited for teaching/service contributions
• promotes national ties, both to SFN and via collaboration
• promotes the dev. of large-scale projects (training grants/program projects)
• serve as a liaison (in collaboration with Chair of NRSC) to VPRs and COPD re. program development
• develop an External Review Committee and coordinates the External Review

Seminar
• solicits names of speakers and topics
• organizes the formal seminar series
• schedule opportunities for faculty/students to meet w/visting speakers
• work with Admin. Coordinator, and host faculty, to assure itineraries are well-developed and we effectively showcase TAMU/HSC resources
• provides oversight to graduate students charged with organizing the Annual Symposium
• works to foster strong participation at colloquia and symposia
• develops events that showcase program strenghts
• allocates resources to bolster program synergy (w/select areas of concentration)

Undergraduate Program
• makes recommendations re. degree requirements and curriculum
• makes recommendation re. the dev. and approval of new courses (and cross-listing)
• monitors and evaluate courses w/in the curriculum and recommends changes as needed
• oversees the undergrad. minor, with the goal of developing a major in NRSC
• works to develop effective research opportunities for NRSC students
• works to integrate training opportunities with TAMHSC faculty
• works to build a program that emphasizes program quality and student placement
• committee members assist Admin. Coordinator to provide effective academic advising and placement
• develops/maintains participation in relevant undergrad. activities (e.g., Brainbowl)
TAMIN Ballot-July 2011

Instructions: Please indicate your selections below and return to Sylvia Bernal (SylviaBernal@tamu.edu) as an attachment via e-mail by 5:00 PM on 8/9/2011.

TAMIN Chair-Choose 1

_____ David Earnest (NExT/BIOL; HSC Curriculum, Admissions & Faculty Senate; SFN Chpt. Pres.)

Ph.D., Northwestern University, 1984; Professor in Department of Neuroscience and Experimental Therapeutics with joint appointment in TAMU Department of Biology. His administrative experience includes service on numerous committees: College of Medicine and HSC (Faculty Advisory Committee, Medical Admissions Committee, Curriculum Committee and HSC Faculty Senate); Faculty for Neuroscience (Recruiting Committee); local chapter of the Society for Neuroscience (President); Center for Biological Clocks Research (Executive Committee). He has contributed to the research training of 22 graduate students, 4 postdoctoral fellows, 5 undergraduates and 3 medical students. His research program provides for interdisciplinary collaborations with TAMIN faculty at the HSC, TAMU and the College of Veterinary Medicine & Biomedical Sciences. His vision is to promote further progress in the development of TAMIN by stimulating interdisciplinary collaborators among researchers at different components and enhancing success in the recruitment of top graduate students.

_____ Jane Welsh (VIBS; current Chair: Seminar; member: Membership, Finance; TAMIN submitter)

I received a B.Sc. in Microbiology and a Ph.D. in Immunology from the University of London, UK. Then I was a postdoctoral researcher at King’s College Hospital, London and at the University of Cambridge. I came to Texas A&M in 1989 and have been researching the pathogenesis of multiple sclerosis, focusing on: 1) the blood-brain barrier; 2) understanding the mechanisms of estrogen therapy in MS with Dr. Sohrabji and interferon tau with Dr. Bazer; and 3) the role of psychological stress in the development of MS with Dr. Meagher. I have been involved in the Faculty of Neuroscience ever since its inception and also the TAMU Chapter of Neuroscience as Secretary/Treasurer and then President. For the future of TAMIN, I would envision continuing the excellent current events and also developing venues to allow interactions that would lead to more collaborative research efforts and the professional development of graduate students and postdoctoral scientists.

GRADUATE PROGRAM COMMITTEE

coChair (Grad. Advisor for TAMU; 9/11-8/13)-Choose 1

_____ Mike Smotherman (BIOL; coChair: Graduate Program; member: Seminar)
_____ Teresa Wilcox (PSYC; member: Graduate Program)

Member (9/11-8/13)-Choose 2

_____ Gladys Ko (VIBS; member: Graduate Program, Undergraduate Program)
_____ Thierry Lints (BIOL; organizes Princ. Neurosci. 1 & 2)
_____ Ian Steele-Russell (Psychiatry & Beh. Sci.)
_____ Teresa Wilcox (PSYC; member: Graduate Program)

MEMBERSHIP/NOMINATION COMMITTEE

Member (9/11-8/13)-Choose 2

_____ Tim Cudd (VTTP)
_____ Lisa Geraci (PSYC)
_____ Paul Hardin (BIOL)
_____ Keith Young (Psychiatry & Beh. Sci.)
GRADUATE RECRUITING COMMITTEE

Member (9/11-8/13)-Choose 2
____ Scott Dindot (VTPB)
____ Mark Harlow (BIOL)
____ Mary Meagher (PSYC; TAMIN submitter; Behavioral & Cellular Neuroscience Program Coordinator)
____ Mendell Rimer (NExT; member: Graduate Recruiting)

SEMINAR COMMITTEE

Chair (9/11-8/13)-Choose 1
____ Paul Wellman (PSYC; member: Finance)
____ Jane Welsh (VIBS; current Chair: Seminar; member: Membership, Finance; TAMIN submitter)

Member (9/11-8/13)-Choose 2
____ Hubert Amrein (Mol. Cell. Med.)
____ Spencer Behmer (ENTO)
____ Cynthia Riccio (EPSY; TAMIN submitter)
____ Reddy Samba (NExT; TAMHSC-COM)

UNDERGRADUATE CURRICULUM COMMITTEE

Chair (9/11-8/13)-Choose 1
____ Louise Abbott (VIBS; chair: Undergraduate Program; member: Membership)
____ Rachel Hull (PSYC; Director of PSYC advising and honors; PSYC undergrad curriculum committee)

Member (9/11-8/13)-Choose 2
____ John Buchanan (HLKN; member; Coordinator UG Motor Behavior track-HLKN)
____ Yoonsuck Choe (CSCE; member: Undergraduate Curriculum; TAMIN submitter)
____ Michelle Hook (PSYC; member: Undergraduate Program)
____ Rajesh Miranda (NExT, Former executive committee member of the Faculty for Neuroscience)

FINANCE COMMITTEE

Member (9/11-8/13)-Choose 2
____ Federico Bermúdez-Rattoni (PSYC)
____ Rene Garcia (BIOL; TAMIN submitter)
____ Jim Grau (PSYC; chair: TAMIN; TAMIN submitter)
____ Caurnel Morgan (NFSC; member: Finance; President-elect: SFN Chpt.)
Current Committees (Sept. 2011)

Chair: Jane Welsh, VIBS (9/11-8/13)

GRADUATE PROGRAM COMMITTEE
co-Chair (TAMU) - Mike Smotherman, Biology (9/11-8/13)
co-Chair (HSC) - Farida Sohrabji, NExT (9/10-9/12)
Member 1 - Gladys Ko, VIBS (9/11-8/13)
Member 2 - Brian Perkins, Biology (9/10-8/12)
Member 3 - Thierry Lints, Biology (9/11-8/13)
Member 4 - Louise Abbott, VIBS (9/10-8/12)

MEMBERSHIP COMMITTEE
Chair - Ursula Winzer-Serhan, NExT (9/10-8/12)
Member 1 - Paul Hardin, Biology (9/11-8/13)
Member 2 - Ginger Carney, Biology (9/10-8/12)
Member 3 - Tim Cudd, VTTP (9/11-8/13)
Member 4 - Soshy Eitan, Psychology (9/10-8/12)

GRADUATE RECRUITING COMMITTEE
Chair - Gregg Wells, Mol. Cell. Med. (9/10-8/12)
Member 1 - Mary Meagher, Psychology (9/11-8/13)
Member 2 - Mark Zoran, Biology (9/10-8/12)
Member 3 - Mark Harlow, Biology (9/11-8/13)
Member 4 - Jianrong Li, VIBS (9/10-8/12)

SEMINAR COMMITTEE
Chair - Paul Wellman, Psychology
Member 1 - Hubert Amrein, Mol. Cell. Med. (9/11-8/13)
Member 2 - Jyotsna Vaid, Psychology (9/10-8/12)
Member 3 - Reddy Samba, NExT (9/11-8/13)
Member 4 - Eddie Harmon-Jones, Psychology (9/10-8/12)

UNDERGRADUATE PROGRAM COMMITTEE
Chair - Louise Abbott, VIBS (9/10-8/12)
Member 1 - Yoonsuck Choe, Computer Science (9/11-8/13)
Member 2 - Gladys Ko, VIBS (9/10-8/12)
Member 3 - Michelle Hook, Psychology (9/11-8/13)
Member 4 - Marcel Amstalden, Animal Science (9/10-8/12)

FINANCE COMMITTEE
Chair - Mark Zoran, Biology (9/10-8/12)
Member 1 - Rene Garcia, Biology (9/11-8/13)
Member 2 - Dave Earnest, NExT (9/10-8/12)
Member 3 - Jim Grau, Psychology (9/11-8/13)
Member 4 - Paul Wellman, Psychology (9/10-8/12)

SFN Chapter Chair: Caurnel Morgan, NFSC (9/11-8/12)

Graduate Student Representative: to be elected
APPENDIX I

Institutional Profile for DOE
External Program Accreditation Docs.
January 2, 2017

TO: External Program Reviewers and Program Accreditors

FROM: Michael T. Stephenson
Associate Provost for Academic Affairs and SACSCOC Accreditation Liaison

RE: Information required for USDOE Accrediting Bodies

Texas A&M University is accredited by the Southern Association of Colleges and Schools Commission on Colleges to award baccalaureate, master’s, and doctoral degrees. Consistent with comprehensive standard 3.13.1, the following provides the institution’s official position on its purpose, governance, programs, degrees, diplomas, certificates, personnel, finances, and constituencies and is published in official university documents as noted.

Purpose

Classified by the Carnegie Foundation as a Research Doctoral University (Highest Research Activity), Texas A&M embraces its mission of the advancement of knowledge and human achievement in all its dimensions. The research mission is a key to advancing economic development in both public and private sectors. Integration of research with teaching prepares students to compete in a knowledge-based society and to continue developing their own creativity, learning, and skills beyond graduation.

The institution’s official mission statement, published both on the institution’s web page as well as in its annual university catalog, is:

Texas A&M University (Texas A&M) is dedicated to the discovery, development, communication and application of knowledge in a wide range of academic and professional fields. Its mission of providing the highest quality undergraduate and graduate programs is inseparable from its mission of developing new understandings through research and creativity. It prepares students to assume roles in leadership, responsibility and service to society. Texas A&M assumes as its historic trust the maintenance of freedom of inquiry and an intellectual environment nurturing the human mind and spirit. It welcomes and seeks to serve persons of all racial, ethnic and geographic groups, women and men alike, as it addresses the needs of an increasingly diverse population and a global economy. In the twenty-first century, Texas A&M University seeks to assume a place of preeminence among public universities while respecting its history and traditions.

Governance

The governance of the institution was described in the 2012 certification of compliance submitted to SACSCOC.
Texas A&M University at College Station, the flagship institution of the Texas A&M University System, has branch campuses located in Galveston, Texas and Doha, Qatar. A ten-member Board of Regents, appointed by the Governor, directs the Texas A&M System. The appointment of each Regent follows Texas Education Code (TEC, Chapter 85, Section 21).

TEC outlines the duties and responsibilities of the Board of Regents. These responsibilities are also defined in System Policy 02.01 Board of Regents and TEC 51.352. The Board elects two officers: Chair and Vice Chair. There are four standing committees: Audit, Academic & Student Affairs, Finance, and Buildings & Physical Plant. Special committees may be appointed by the Chair with Board approval.

At Texas A&M University the President is the chief executive officer; the President is not the presiding officer of the Board of Regents. The President reports to the state-appointed Board of Regents through the Chancellor of the Texas A&M University System. System Policy 2.05 Presidents of System Member Universities defines the duties of the President. The appointment of the President follows conditions set forth in System Policy 01.03 Appointing Power and Terms and Conditions of Employment, section 2.2.

**Personnel**

The institution is led by the President and members of his cabinet:

Michael K. Young, President  
Karan L. Watson, Provost and Executive Vice President  
Jerry R. Strawser, Executive Vice President for Finance and Administration and CFO  
Michael Benedik, Vice Provost  
M. Dee Childs, Vice President for Information Technology and CIO  
Michael G. O’Quinn, Vice President for Government Relations  
Dr. Douglas Palmer, Interim Vice President and COO, TAMU-Galveston  
Barbara A. Abercrombie, Vice President for HR & Organizational Effectiveness  
Jessica Rubie, Associate Vice President for Strategic Initiatives  
Christine Stanley, Vice President and Associate Provost for Diversity  
Amy B. Smith, Senior Vice President and Chief Marking & Communications Officer  
Glen A. Laine, Vice President for Research  
Carrie L. Byington, Senior Vice President TAMU Health Science Center, Dean of the College of Medicine, and Vice Chancellor for Health Services  
Daniel J. Pugh, Sr., Vice President for Student Affairs  
Gen Joe E. Ramirez, Jr. Commandant, Corps of Cadets  
Amy B. Smith, Senior Vice President and Chief Marketing and Communications Officer  
Scott Woodward, Director of Athletics

**Programs, Degrees, Diplomas, and Certificates**

See the Institutional Summary submitted to SACSCOC

**Finances**

See the Financial Profile 2016 submitted to SACSCOC
Name of Institution  Texas A&M University

Name, Title, Phone number, and email address of Accreditation Liaison
Michael T. Stephenson
Associate Provost for Academic Affairs and SACSCOC Accreditation Liaison
979.845.4016
mstephenson@tamu.edu

Name, Title, Phone number, and email address of Technical Support person for the Compliance Certification
Alicia M. Dorsey
Assistant Provost for Institutional Effectiveness
979.862.2918
amdorsey@tamu.edu

IMPORTANT:

Accreditation Activity (check one):

☒  Submitted at the time of Reaffirmation Orientation
☐  Submitted with Compliance Certification for Reaffirmation
☐  Submitted with Materials for an On-Site Reaffirmation Review
☐  Submitted with Compliance Certification for Fifth-Year Interim Report
☐  Submitted with Compliance Certification for Initial Candidacy/Accreditation Review
☐  Submitted with Merger/Consolidations/Acquisitions
☐  Submitted with Application for Level Change

Submission date of this completed document:  September 29, 2015
EDUCATIONAL PROGRAMS

1. Level of offerings (Check all that apply)
   - Diploma or certificate program(s) requiring less than one year beyond Grade 12
   - Diploma or certificate program(s) of at least two but fewer than four years of work beyond Grade 12
   - Associate degree program(s) requiring a minimum of 60 semester hours or the equivalent designed for transfer to a baccalaureate institution
   - Associate degree program(s) requiring a minimum of 60 semester hours or the equivalent not designed for transfer
   - Four or five-year baccalaureate degree program(s) requiring a minimum of 120 semester hours or the equivalent
   - Professional degree program(s)
   - Master's degree program(s)
   - Work beyond the master's level but not at the doctoral level (such as Specialist in Education)
   - Doctoral degree program(s)
   - Other (Specify) _____

2. Types of Undergraduate Programs (Check all that apply)
   - Occupational certificate or diploma program(s)
   - Occupational degree program(s)
   - Two-year programs designed for transfer to a baccalaureate institution
   - Liberal Arts and General
   - Teacher Preparatory
   - Professional
   - Other (Specify) _____

GOVERNANCE CONTROL

Check the appropriate governance control for the institution:

- Private (check one)
  - Independent, not-for-profit
    - Name of corporation OR
    - Name of religious affiliation and control: _____
  - Independent, for-profit *
    - If publicly traded, name of parent company: _____
Academic Program Review, May 2017

- Public state *(check one)*

- Not part of a state system, institution has own independent board
- Part of a state system, system board serves as governing board
- Part of a state system, system board is super governing board, local governing board has delegated authority
- Part of a state system, institution has own independent board

* If an institution is part of a state system or a corporate structure, a description of the system operation must be submitted as part of the Compliance Certification for the decennial review. See Commission policy “Reaffirmation of Accreditation and Subsequent Reports” for additional direction."

INSTITUTIONAL INFORMATION FOR REVIEWERS

**Directions:**

*Please address the following and attach the information to this form.*

1. **History and Characteristics**
   Provide a brief history of the institution, a description of its current mission, an indication of its geographic service area, and a description of the composition of the student population. Include a description of any unusual or distinctive features of the institution and a description of the admissions policies (open, selective, etc.). If appropriate, indicate those institutions that are considered peers. Please limit this section to one-half page.

2. **List of Degrees**
   List all degrees currently offered (A. S., B.A., B.S., M.A., Ph.D., for examples) and the majors or concentrations within those degrees, as well as all certificates and diplomas. For each credential offered, indicate the number of graduates in the academic year previous to submitting this report. Indicate term dates.

3. **Off-Campus Instructional Locations and Branch Campuses**
   List all locations where 50% or more credit hours toward a degree, diploma, or certificate can be obtained primarily through traditional classroom instruction. Report those locations in accord with the Commission's definitions and the directions as specified below.

   **Off-campus instructional sites**—a site located geographically apart from the main campus at which the institution offers 50% or more of its credit hours for a diploma, certificate, or degree. This includes high schools where courses are offered as part of dual enrollment. For each site, provide the information below. **The list should include only those sites reported and approved by SACSCOC.** Listing unapproved sites below does not constitute reporting them to SACSCOC. In such cases when an institution has initiated an off-campus instructional site as described above without prior approval by SACSCOC, a prospectus for approval should be submitted immediately to SACSCOC.
**Institutions with off-campus instructional sites** at which the institution offers 25-49% credit hours for a diploma, certificate, or degree—including high schools where courses are offered as dual enrollment—are required to notify SACSCOC in advance of initiating the site. For each site, provide the information below.

<table>
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<tr>
<th>Name of Site</th>
<th>Physical Address (street, city, state, country) Do not include PO Boxes.</th>
<th>Date Notified SACSCOC by SACSCOC</th>
<th>Date Implemented by the institution</th>
<th>Educational programs offered (specific degrees, certificates, diplomas) with 25-49% credit hours offered at each site</th>
<th>Is the site currently active? (At any time during the past 5 years, have students been enrolled and courses offered? If not, indicate the date of most recent activity.)</th>
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**Branch campus**—an instructional site located geographically apart and independent of the main campus of the institution. A location is independent of the main campus if the location is (1) permanent in nature, (2) offers courses in educational programs leading to a degree, certificate, or other recognized educational credential, (3) has its own faculty and administrative or supervisory organization, and (4) has its own budgetary and hiring authority. The list should include only those branch campuses reported and approved by SACSCOC. Listing unapproved branch campuses below does not constitute reporting them to SACSCOC. A prospectus for an unapproved branch campuses should be submitted immediately to SACSCOC.

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<th>Date Implemented by the institution</th>
<th>Educational programs (specific degrees, certificates, diplomas) with 50% or more credits hours offered at the branch campus</th>
<th>Is the campus currently active? (At any time during the past 5 years, have students been enrolled and courses offered? If not, indicate the date of most recent activity.)</th>
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</table>

### 4. Distance and Correspondence Education
5. **Accreditation**

(1) List all agencies that currently accredit the institution and any of its programs and indicate the date of the last review by each.

(2) If SACS Commission on Colleges is not your primary accreditor for access to USDOE Title IV funding, identify which accrediting agency serves that purpose.

(3) List any USDOE recognized agency (national and programmatic) that has terminated the institution’s accreditation (include the date, reason, and copy of the letter of termination) or list any agency from which the institution has voluntarily withdrawn (include copy of letter to agency from institution).

(4) Describe any sanctions applied or negative actions taken by any USDOE-recognized accrediting agency (national, programmatic, SACSCOC) during the two years previous to the submission of this report. Include a copy of the letter from the USDOE to the institution.

6. **Relationship to the U.S. Department of Education**

   Indicate any limitations, suspensions, or termination by the U.S. Department of Education in regard to student financial aid or other financial aid programs during the previous three years. Report if on reimbursement or any other exceptional status in regard to federal or state financial aid.

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**Document History**

Adopted: September 2004  
Revised: March 2011  
Revised: January 2014
1. History and Characteristics

Provide a brief history of the institution, a description of its current mission, an indication of its geographic service area, and a description of the composition of the student population. Include a description of any unusual or distinctive features of the institution and a description of the admissions policies (open, selective, etc.). If appropriate, indicate those institutions that are considered peers. Please limit this section to one-half page.

**History.** Texas A&M University was established in 1871 as the state’s first public institution of higher education and opened for classes in 1876. We are now one of a select few institutions in the nation to hold land grant, sea grant (1971) and space grant (1989) designations. We are also one of few universities to host a presidential library; the George Bush Presidential Library and Museum opened in 1997. A mandatory military component was a part of the land grant designation until 1965 and today we are one of only three institutions with a full-time corps of cadets, leading to commissions in all branches of service. We have two branch campuses, one in Galveston, Texas, (established in 1962, officially merged with Texas A&M in 1991) and one in Doha, Qatar (established in 2003). In 2001 we were admitted to the Association of American Universities (AAU) and in 2004 to Phi Beta Kappa. We are classified by the Carnegie Foundation as a Research University (very high research activity).

**Mission.** Texas A&M University is dedicated to the discovery, development, communication, and application of knowledge in a wide range of academic and professional fields. Its mission of providing the highest quality undergraduate and graduate programs is inseparable from its mission of developing new understandings through research and creativity. It prepares students to assume roles in leadership, responsibility and service to society. Texas A&M assumes as its historic trust the maintenance of freedom of inquiry and an intellectual environment nurturing the human mind and spirit. It welcomes and seeks to serve persons of all racial, ethnic and geographic groups as it addresses the needs of an increasingly diverse population and a global economy. In the 21st century, Texas A&M University seeks to assume a place of preeminence among public universities while respecting its history and traditions.

**Enrollment Profile.**
77.42% Undergraduate, 18.41% Graduate, 4.02% Professional, and 0.14% Post-Doc Certificate

**Undergraduate Students:**
93.58% Texas Residents, 3.96% non-Texas Residents, 2.46% non-Texas, non-US Residents; 62.41% White, 3.11% Black, 22.33% Hispanic, 6.21% Asian

**Graduate Students:**
45.09% Texas Residents, 16.57% non-Texas Residents, 38.34% non-Texas, non-US Residents

Admissions Process. Selective. Automatic admission for Texas resident applicants in the top 10% of their high school graduating class; automatic admission for applicants who rank in the top 25% of their high school graduating class and achieve a combined (old) SAT math and SAT critical reading score of at least 1300 with a test score of at least 600 in each component, or combined (newly redesigned) SAT math and SAT evidence based reading and writing (EBRW) score of at least 1360 with a test score of at least 620 in Math and 660 in EBRW, or 30 composite on the ACT with a 27 in the math and English components; review of all other applicants based on academic potential, distinguishing characteristics, exceptional circumstances and personal achievements.

**Peer Institutions.** Georgia Institute of Technology, Ohio State University, Pennsylvania State University, Purdue University, University of California- Berkeley, Davis, Los Angeles, San Diego, University of Florida, University of Illinois – Champaign/Urbana, University of Michigan, University of Minnesota, University of North Carolina – Chapel Hill, University of Texas – Austin, and University of Wisconsin – Madison.
# 2. List of Degrees

List all degrees currently offered (A. S., B.A., B.S., M.A., Ph.D., for examples) and the majors or concentrations within those degrees, as well as all certificates and diplomas. For each credential offered, indicate the number of graduates in the academic year previous to submitting this report. Indicate term dates.

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Academic Program Review, May 2017

Texas A&M Institute for Neuroscience
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### Off-Campus Instructional Locations – 50% or more.

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<th>Name of Site</th>
<th>Physical Address (street, city, state, country) Do not include PO Boxes.</th>
<th>Date Approved by SACSCOC</th>
<th>Date Implemented by the Institution</th>
<th>Educational programs offered (specific degrees, certificates, diplomas) with 50% or more credits hours offered at each site</th>
<th>Is the site currently active? (At any time during the past 5 years, have students been enrolled and courses offered? If not, indicate the date of most recent activity.)</th>
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<td>Texas A&amp;M Health Science Center</td>
<td>8441 State Highway 47 Clinical Building 1, Suite 3100 Bryan, TX 77807</td>
<td>2000</td>
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<td>EDUCATION FOR HEALTHCARE PROFESSIONALS MEDICAL SCIENCES MD MEDICAL SCIENCES MS MEDICAL SCIENCES PHD MEDICINE MD NURSING BSN NURSING EDUCATION MSN PHARMACY PHMD FAMILY NURSE PRACTITIONER MSN</td>
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<td>City Centre</td>
<td>842 West Sam Houston Parkway North, Suite 200 Houston, Texas 77024-3920</td>
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<td>ANALYTICS BUSINESS ADMINISTRATION MBA</td>
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<td>College of Dentistry</td>
<td>3302 Gaston Ave. Dallas, TX 75246</td>
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<td>Name of Site</td>
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<td>Is the site currently active? (At any time during the past 5 years, have students been enrolled and courses offered? If not, indicate the date of most recent activity.)</td>
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<td>Institute of Biosciences and Technology</td>
<td>2121 W. Holcombe Blvd, Houston, TX 77030</td>
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<td>Rangel College of Pharmacy</td>
<td>1010 W. Avenue B, Kingsville, TX 78363</td>
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<td>College of Medicine - Temple</td>
<td>2401 S. 31st Street, Temple, TX 75050</td>
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<td>Clinical Learning Resource Center</td>
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<td>Rural Public Health - McAllen</td>
<td>2101 South McColl Road, McAllen, TX 78503</td>
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<td>Texas A&amp;M University School of Law</td>
<td>1515 Commerce St, Fort Worth, TX 76102</td>
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<td>Houston Methodist Hospital</td>
<td>6670 Bertner Avenue, R2-216, Houston, TX 77030</td>
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**Off-Campus Instructional Locations – 25%-49%**

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<th>Physical Address (street, city, state, country) Do not include PO Boxes.</th>
<th>Date Notified SACSCOC</th>
<th>Date Implemented by the Institution</th>
<th>Educational programs offered (specific degrees, certificates, diplomas) with 25-49% credit hours offered at each site</th>
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<td>Department of State Health Services</td>
<td>1100 West 49th Austin, TX. 78756</td>
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**Branch Campuses**

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<th>Date Implemented by the Institution</th>
<th>Educational programs (specific degrees, certificates, diplomas) with 50% or more credits hours offered at the branch campus</th>
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### 4. Distance and Correspondence Education

Provide an initial date of approval for your institution to offer distance education. Provide a list of credit-bearing educational programs (degrees, certificates, and diplomas) where 50% or more of the credit hours are delivered through distance education modes. For each educational program, indicate whether the program is delivered using synchronous or asynchronous technology, or both. For each educational program that uses distance education technology to deliver the program at a specific site (e.g., a synchronous program using interactive videoconferencing), indicate the program offered at each location where students receive the transmitted program. Please limit this description to one page, if possible.

#### Initial Approval in February 2000

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<tr>
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</tr>
<tr>
<td>BIOLOGICAL AND AGRIC ENGINEERING</td>
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<td>Synchronous course offered worldwide via PC or LMS</td>
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<td>EDUC HUMAN RESOURCE DEVELOPMENT</td>
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<td>EDUCATION FOR HEALTH CARE PROFESSIONALS</td>
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<td>ENGINEERING SYSTEMS MANAGEMENT</td>
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<td>EPIDEMIOLOGY</td>
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<tr>
<td>FAMILY NURSE PRACTITIONER</td>
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<td>MJ</td>
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<td>MARITIME ADMINISTRATION &amp; LOGISTICS</td>
<td>MMAL</td>
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<tr>
<td>MATHEMATICS</td>
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<td>NATURAL RESOURCES DEVELOPMENT</td>
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<td>PLANT BREEDING</td>
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<td>PLANT BREEDING</td>
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<tr>
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<td>PUBLIC SERVICE AND ADMINISTRATION</td>
<td>MPSA</td>
<td>College Station, TX</td>
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<tr>
<td>RECREATION &amp; RESOURCES DEVELOPMENT</td>
<td>MRRD</td>
<td>College Station, TX</td>
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<tr>
<td>SAFETY ENGINEERING</td>
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<td>Academic Program Review, May 2017</td>
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<td>STATISTICS</td>
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<td>WILDLIFE SCIENCE</td>
<td>MWSC</td>
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<td>MILITARY LAND SUSTAINABILITY</td>
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<td>ADVANCED INTERNATIONAL AFFAIRS</td>
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<td></td>
<td>AGRICULTURE E-LEARNING DEVELOPMENT</td>
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<td>APPLIED BEHAVIOR ANALYSIS</td>
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<td>EDUCATION FOR HEALTHCARE PROFESSIONALS</td>
<td>CERT</td>
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<tr>
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<td>CERT</td>
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<td>INDUSTRIAL DATA ANALYTICS</td>
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<td>NATIONAL SECURITY AFFAIRS</td>
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<td>PUBLIC HEALTH</td>
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<tr>
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<td>REGULATORY SCIENCE IN FOOD SYSTEMS</td>
<td>CERT</td>
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<td>SAFETY ENGINEERING</td>
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<tr>
<td></td>
<td>APPLIED STATISTICS</td>
<td>CERT</td>
</tr>
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5. Accreditation

<p>| Accreditation Council for Pharmacy Education | The pharmacy professional degree program | Last Review: April 2014 |
| American Council for Construction Education | The B.S. and M.S. curriculum in construction science | Last Review: 2011 (B.S.) and 2012 (M.S.) |
| American Psychological | The clinical psychology program | Last Review: April/May 2015 |</p>
<table>
<thead>
<tr>
<th>Association</th>
<th>in the Department of Psychology and the counseling psychology and school psychology program in the Department of Educational Psychology</th>
<th>Last Review: 2013</th>
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<tbody>
<tr>
<td>American Veterinary Medical Association Council on Education</td>
<td>The veterinary medicine degree program</td>
<td>Last Review: 2013</td>
</tr>
<tr>
<td>Association to Advance Collegiate Schools of Business (AACSB)</td>
<td>The business baccalaureate, master’s, and doctoral programs in Mays Business School</td>
<td>Last Review: Fall 2012</td>
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<tr>
<td>Commission on Accreditation for Dietetics Education</td>
<td>The dietetic track in the nutritional sciences curriculum and the dietetic internship program</td>
<td>Last review: January 2015</td>
</tr>
<tr>
<td>Commission on Accreditation of Athletic Training Education (caATE)</td>
<td>Athletic Training (College of Education)</td>
<td>Last Review: 2013</td>
</tr>
<tr>
<td>Commission on Accreditation of Healthcare Management Education</td>
<td>The Master of Health Administration</td>
<td>Last Review: Fall 2010</td>
</tr>
<tr>
<td>Commission on Collegiate Nursing Education and the Texas Board of Nursing</td>
<td>The nursing degree programs</td>
<td>Last Review: July 2013</td>
</tr>
<tr>
<td>Commission on Dental Accreditation. (CODA)</td>
<td>The degree programs in dentistry and dental hygiene and the certificate programs in the ten advanced dental graduate education programs</td>
<td>Last Review: August 2013</td>
</tr>
<tr>
<td>Commission on English Language Program Accreditation (CEA)</td>
<td>The English Language Institute</td>
<td>Last review: 2013</td>
</tr>
<tr>
<td>Computing Accreditation Commission of ABET</td>
<td>The computer science program</td>
<td>Last review: 2010</td>
</tr>
<tr>
<td>Council of the Section of Legal Education and Admissions to the Bar of the American Bar Association</td>
<td>Texas A&amp;M University School of Law</td>
<td>Last review: 2010</td>
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<tr>
<td>Council on Education for Public Health</td>
<td>The School of Public Health degree programs</td>
<td>Last Review: April 2011</td>
</tr>
<tr>
<td>Engineering Accreditation Commission of ABET</td>
<td>Undergraduate programs in aerospace, biological and agricultural, biomedical, chemical, civil, computer, electrical, industrial, mechanical, nuclear, ocean, petroleum and radiological health engineering</td>
<td>Last Review: 2010-2011 (College Station) and 2015 (Qatar)</td>
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<td>Engineering Accreditation Commission of ABET</td>
<td>Maritime systems engineering (Offshore and Coastal Systems Engineering) – TAMU Galveston</td>
<td>Last review: 2010-11</td>
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<tr>
<td>Program Name</td>
<td>Description</td>
<td>Last Review</td>
</tr>
<tr>
<td>--------------------------------------------------</td>
<td>-----------------------------------------------------------------------------</td>
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<tr>
<td><strong>Engineering Technology Accreditation Commission of ABET</strong></td>
<td>The electronic systems engineering technology program, the manufacturing and mechanical engineering technology program,</td>
<td>2013-2014 (College Station) and 2015 (Qatar)</td>
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<tr>
<td><strong>Engineering Technology Accreditation Commission of ABET</strong></td>
<td>marine engineering technology – TAMU Galveston</td>
<td>2013-14</td>
</tr>
<tr>
<td><strong>Forensic Science Education Programs Accreditation Commission (FEPAC)</strong></td>
<td>The forensics and investigative sciences program</td>
<td>Last Site Visit: October 2011 Accreditation dates: 1/2012-1/2017</td>
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<tr>
<td><strong>Institute of Food Technologists</strong></td>
<td>The food science and technology curriculum</td>
<td>Last Review: December 2011</td>
</tr>
<tr>
<td><strong>Landscape Architectural Accreditation Board</strong></td>
<td>The curriculum in landscape architecture</td>
<td>Last Review: July 2015</td>
</tr>
<tr>
<td><strong>Liaison Committee on Medical Education</strong></td>
<td>The medical education degree program</td>
<td>Last Review: August 2012</td>
</tr>
<tr>
<td><strong>National Architectural Accrediting Board</strong></td>
<td>The curriculum in architecture</td>
<td>Last Review: March 2013</td>
</tr>
<tr>
<td><strong>Network of Schools of Public Policy, Affairs, and Administration</strong></td>
<td>The Master of Public Service and Administration degree in the Bush School of Government and Public Service</td>
<td>Last review: April 2014</td>
</tr>
<tr>
<td><strong>National Recreation and Park Association</strong></td>
<td>The curriculum in recreation, park and tourism sciences</td>
<td>Last Review: June 2010</td>
</tr>
<tr>
<td><strong>Planning Accreditation Board</strong></td>
<td>The Master of Urban Planning curriculum</td>
<td>Last Review: 2013</td>
</tr>
<tr>
<td><strong>Society for Range Management</strong></td>
<td>The curriculum in rangeland ecology and management</td>
<td>Last Review: 2006</td>
</tr>
<tr>
<td><strong>Society of American Foresters</strong></td>
<td>The curriculum in forestry</td>
<td>Last Review: 2013</td>
</tr>
<tr>
<td><strong>State Board of Educator Certification Texas Education Agency</strong></td>
<td>Programs in professional education and degrees conferred by Texas A&amp;M University</td>
<td>Last review 2011</td>
</tr>
</tbody>
</table>

(2) If SACS Commission on Colleges is not your primary accreditor for access to USDOE Title IV funding, identify which accrediting agency serves that purpose.

Not applicable.

(3) List any USDOE recognized agency (national and programmatic) that has terminated the institution’s accreditation (include the date, reason, and copy of the letter of termination) or list any agency from which the institution has voluntarily withdrawn (include copy of letter to agency from institution).

None.
(4) Describe any sanctions applied or negative actions taken by any USDOE-recognized accrediting agency (national, programmatic, SACSCOC) during the two years previous to the submission of this report. Include a copy of the letter from the USDOE to the institution.

None.

6. Relationship to the U.S. Department of Education.

Texas A&M University does not have any limitations or suspensions, nor have we been terminated by the U.S. Department of Education in regard to student financial aid or other financial aid programs during the previous three years. We are not on reimbursement nor do we have any other exceptional status in regard to federal or state financial aid.
APPENDIX J

Texas A&M University Vision 2020
Twelve Imperatives of Texas A&M University’s Vision 2020

1. Elevate Our Faculty and Their Teaching, Research, and Scholarship

The world today is knowledge-based and constantly changing. In such a world, the quality research university is "a creator, organizer, preserver, transmitter, and applier of knowledge." The foundation of these functions is an excellent faculty in adequate numbers. We need to increase substantially the size of our faculty (perhaps by half), and we must attract and retain many more top scholars, teachers, and researchers. We will have to review and strengthen hiring and tenure policies, enhance compensation, focus our scholarship, and transform our administrative culture. We cannot achieve our goal without a nationally recognized faculty with a passion for teaching and an academic environment that values and rewards innovation, great ideas, and the search for the truth.

2. Strengthen Our Graduate Programs

We must have a shift in our thinking about the role of graduate education to attain the level of excellence we desire. A substantially expanded graduate studies effort is critical to our academic aspirations and to our effectiveness as a great research university. Outstanding professors attract superior graduate students and, in many instances, the money to help support their research. But these professors by themselves will not be enough. We must create a dynamic, exciting, discovery-driven intellectual environment that will draw superior graduate students, comparable to those in the nation's best graduate programs.

3. Enhance the Undergraduate Academic Experience

The core of Texas A&M University must be a residential, learner-centered community that attracts excellent students and provides quality learning and mentoring experiences. We must better prepare learners for lives of discovery, innovation, leadership, and citizenship by better inculcation of writing, thinking, and self-expression skills. Texas A&M University is proud of its history of developing student leaders. Our co-curricular programs are already an area of true distinctiveness, but we must continue to strengthen their substance and reputation and extend their benefits to a greater percentage of the student body. While our retention rate is the highest in Texas, it is low relative to the best national institutions; we must make an institutional commitment to graduate those we enroll. We must emphasize education more than training and significantly improve our student-faculty ratio. We must provide more opportunity for intellectual exchange between distinguished faculty and undergraduates. Our recruiting should be more proactive and produce a more broadly representative student body. We need to expand our honors, study/live-abroad, interdisciplinary studies, and course-assistance programs.

4. Build the Letters, Arts, and Sciences Core

Texas A&M University has historically placed less emphasis on the letters and arts. While many of our basic science disciplines are nationally acclaimed, the best public universities have stronger and deeper liberal arts programs and a fuller range of such programs with a significantly higher institutional commitment. Such strengthening is necessary for the true, enduring education of our graduates and the enrichment of their lives. It is abundantly clear
that we will never be seen as a premier institution nationally without a far stronger letters, arts, and sciences program.

5. **Build on the Tradition of Professional Education**

Undergraduate education in all areas, including professional education, has been our traditional strength at Texas A&M University. At the heart of Vision 2020 is a belief that we will not only sustain but also continually strengthen our professional programs at both the undergraduate and the graduate levels. We expect that these programs will be the first (as some already are) to represent Texas A&M University solidly and firmly in the top ten nationally. Our professional programs must also recognize the necessity to prepare their graduates more broadly for entry into a complex, changing, and unpredictable world.

6. **Diversify and Globalize the A&M Community**

The time has passed when the isolation of the Texas A&M University campus served a compelling utilitarian function. Information, communication, and travel technology have produced a highly connected global society. The ability to survive, much less succeed, is increasingly linked to the development of a more pluralistic, diverse, and globally aware populace. It is essential that the faculty, students, and larger campus community embrace this more cosmopolitan environment. The university’s traditional core values will give us guidance and distinctiveness, while preparing us to interact with all people of the globe. Texas A&M University must attract and nurture a more ethnically, culturally, and geographically diverse faculty, staff, and student body.

7. **Increase Access to Knowledge Resources**

Despite recent progress, the intellectual assets represented by Texas A&M University library holdings are underdeveloped and must be increased. Coincidentally, we must recognize that the technology related to the storage, access, and distribution of knowledge resources has changed as much in the last decade as in the 550 years since the invention of movable type. Texas A&M University must invest rapidly, but wisely, to gain parity with its academic peers. It must lead, not just grow, in forcefully developing new methods and measures of success in this rapidly changing arena. The wedding of communications and computer technology will, no doubt, yield the most formidable change in academe by 2020. Texas A&M University must lead the adaptation.

8. **Enrich Our Campus**

The physical environment of our campus should be conducive to scholarly work and study. Texas A&M University has an efficient and well-maintained campus. However, during our rapid growth over the past four decades, the physical unity of the campus has been diminished by the presence of Wellborn Road and the railroad tracks. Innovative planning and bold leadership are needed to redress this division for reasons of safety and convenience as well as aesthetics. West Campus has not maintained the human scale that exists on the Main Campus. Through judicious planning we need to attain the same pedestrian-friendly scale and green space that gives the Main Campus its character. The use of large areas for surface parking needs to be reconsidered so that the unity of the campus is maintained as new building occurs to accommodate growth. As more of the university’s
current land holdings are consumed by non-agricultural uses, acquisition of land on or near the Riverside Campus for agricultural development should be a high priority.

9. Build Community and Metropolitan Connections

The way that we relate to the local community, Houston, and other metropolitan areas of the state will have a powerful impact on Texas A&M University and the communities supporting and supported by the university. In addition, it is critical that the community in which we live provide opportunities for families to work and grow. Spouses need high-quality employment opportunities. Faculty and researchers need private-sector sponsorships and commercialization support. As we attract a wider range of people to Texas A&M University, the enrichment provided through our connection to a large metropolitan area becomes increasingly important. Correctly choreographed, such a connection gives us the best of both worlds.

10. Demand Enlightened Governance and Leadership

Great universities have a clearly articulated vision, a stimulating intellectual environment populated by great faculty and students, and resources adequate to support quality offerings. One other characteristic often contributes to greatness: enlightened leadership. Clear, cooperative relationships between the university and the System must be the norm. To achieve our aspirations, strong, enlightened, stable, and forward-thinking leadership focused on academic quality is essential. We have made progress, but we must guard it zealously. Regents must continue to take the policy high ground. The System administration must acknowledge and nurture Texas A&M University’s role as a comprehensive research university with national peers. The university administration must be steadfast in its demand for quality in every decision. And finally, the university administration must make decisions through a process characterized by openness and appropriate faculty and staff participation. Our responsibility to the System as its flagship must be evidenced in all decision-making. Academic progress is fragile. Enlightened, shared governance and leadership are elemental to its achievement.

11. Attain Resource Parity with the Best Public Universities

The combination of rapid population growth, demand for government services and difficult economic times have placed a strain on the Texas treasury in recent years. A good and widely dispersed university system has provided access to a growing college-aged population. Access alone is no longer enough. Texas must have a few universities that offer opportunities equal to the best public universities, while taking complementary steps to maintain access. Competitive peer states have long recognized the economic necessity of comprehensive research universities in meeting the knowledge demands of an information society. States with the best universities are currently investing twice as much funding per student as at Texas A&M University.

Texas A&M University and the University of Texas are ideally positioned to achieve recognition as top national institutions because of the state’s historical, constitutional financial commitment to them. Texas may also need additional institutions of this caliber. The institutions designated to fill this role must be acknowledged and supported in a way that is consistent with national competition. They must be provided the flexibility and
exercise the wisdom and courage to price their offerings more in line with their value, while taking complementary steps to maintain access. Finally, they must use their historical strength to generate more private capital. Texas A&M University must attain resource parity with the best public institutions to better serve Texas.

12. Meet Our Commitment to Texas

Texas A&M University is a creation of the state and in its origin was designed to prepare educated problem-solvers to lead the state’s development. This fundamental mission, born out of the land grant heritage of service, remains today. Texas A&M University's aspiration to be among the best public universities in the country resonates with this historical mandate. The diverse population of Texas should have access to the best public education in America without having to leave the state. Texas A&M University must also reach out even more to help solve the most difficult societal problems, including those related to public education, crime, and the environment, and must honor its heritage of enhancing the economic development of all regions of the state. Texas A&M University, if it aspires to national prominence, must first stay committed to Texas.
APPENDIX K

TAMIN 101 Schedule
## TMIN 101 Schedule (8/14 - 8/25)

<table>
<thead>
<tr>
<th>Time</th>
<th>Location</th>
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</thead>
<tbody>
<tr>
<td>7:00 PM</td>
<td>3806 Ridgewood Street, Bryan, TX 77801</td>
</tr>
<tr>
<td><strong>Sunday 8/14/2016</strong></td>
<td></td>
</tr>
<tr>
<td>9:30 AM</td>
<td>Campus Tour - Misty will lead</td>
</tr>
<tr>
<td>12:00 PM</td>
<td>Lunch with TMIN graduate students and new recruits - Jason's Catering</td>
</tr>
<tr>
<td>1:30 PM</td>
<td>TMIN student presentations</td>
</tr>
<tr>
<td>3:30 AM</td>
<td>Get student ID's - Chris Horrax and Ian will accompany</td>
</tr>
<tr>
<td>7:00 PM</td>
<td>Potluck @ Chris Horrax's House</td>
</tr>
<tr>
<td><strong>Monday 8/15/2016</strong></td>
<td></td>
</tr>
<tr>
<td>9:30 AM</td>
<td>Meet at ILSB 3149 prior to tour</td>
</tr>
<tr>
<td>12:00 PM</td>
<td>ILSB 3216</td>
</tr>
<tr>
<td>3:30 AM</td>
<td>Student Business Services Building</td>
</tr>
<tr>
<td>9:00 AM</td>
<td>ILSB 3216</td>
</tr>
<tr>
<td>9:00 AM</td>
<td>ILSB 3216</td>
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<tr>
<td><strong>Tuesday 8/16/2016</strong></td>
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<tr>
<td>9-12 pm</td>
<td>LAR Conference Room</td>
</tr>
<tr>
<td>7:00 PM</td>
<td>Dinner with TMIN graduate students at Papa Perez (all volunteers invited)</td>
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<tr>
<td><strong>Wednesday 8/17/2016</strong></td>
<td></td>
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<tr>
<td>9am-2pm</td>
<td>LAR Conference Room</td>
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<td>9am-3pm</td>
<td>LAR Conference Room</td>
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<tr>
<td><strong>Thursday 8/18/2016</strong></td>
<td></td>
</tr>
<tr>
<td>9-12 pm</td>
<td>ILSB 1st Floor MIC</td>
</tr>
<tr>
<td>12:30-2pm</td>
<td>ILSB 3147</td>
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<tr>
<td><strong>Friday 8/19/2016</strong></td>
<td></td>
</tr>
<tr>
<td>All Day</td>
<td>New Graduate Student Orientation (Register at: <a href="http://ogaps.tamu.edu/New-Current-Students/New-Graduate-Student-Orientation">http://ogaps.tamu.edu/New-Current-Students/New-Graduate-Student-Orientation</a>)</td>
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<tr>
<td><strong>Saturday 8/20/2016</strong></td>
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<tr>
<td>All Day</td>
<td>Memorial Student Center</td>
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<tr>
<td><strong>Thursday 8/25/16</strong></td>
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<tr>
<td>10:30a-Noon</td>
<td>Orientation</td>
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<tr>
<td>12:30-2p</td>
<td>Welcome Lunch</td>
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<td>The Corner Bar/ 2nd Floor</td>
</tr>
</tbody>
</table>

For questions and/or concerns please contact: Ian Smith at ismith@bio.tamu.edu or Sylvia Bernal at sylviabernal@tamin.tamu.edu
APPENDIX L

Principles of Neuroscience I
Syllabus
NRSC 601 Principles of Neuroscience - Part I  Course Schedule Fall 2015 - as of 6/6/2016

3 credit hours; Mon, Wed & Fri 11:00-11:50 AM; Classroom: Rm 3143, ILSC, TAMU College Station Campus.

Course Coordinators:
Mendell Rimer: 4008 MREB; mjrimer@medicine.tamhsc.edu; http://medicine.tamhsc.edu/basic-sciences/next/faculty/mendell-rimer.html
Wesley Thompson: 3214A ILSB; wthompson@bio.tamu.edu; http://www.bio.tamu.edu/FACMENU/FACULTY/ThompsonW.php


The final course grade (A = 90-100, B = 80-89, C = 70-79 or F < 69) is based on 2 written examinations and 4 written assignments.

The two exams represent 80% of the course grade. The remaining 20% of the course grade is based on written roundtable discussion article critiques (15%) and roundtable discussion participation (5%). Written critiques are graded as follows: Appropriately prepared = 100; poorly prepared = 70; unprepared or absent = 0.

First year TAMIN Neuroscience Students taking this course must also register for NRSC 681 Seminar in Neuroscience, a Journal Club that will be run concurrently with NRSC601.

<table>
<thead>
<tr>
<th>DATE</th>
<th>WEEK</th>
<th>SUBJECT</th>
<th>INSTRUCTOR</th>
<th>contact hrs</th>
<th>Exam # hours</th>
<th>Exam grade (% all exams)</th>
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<td>Monday, August 29, 16</td>
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<td>Course Intro. Overview of &quot;big&quot; problems in Neuroscience</td>
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<td>CNS Cells: Neurons, Glia, Epithelial, . . . etc.</td>
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<td>Excitable Membranes &amp; Action Potentials</td>
<td>Thompson</td>
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<td>Active &amp; Passive Spread of Potentials</td>
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<td>Ion Channels</td>
<td>Lookless</td>
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<td>History of Synaptic transmission</td>
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<td>Neurotransmitters, Synaptic Vesicle Release, Receptors</td>
<td>DuBois</td>
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<td>Harlow</td>
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<td>Dendritic Integration</td>
<td>Griffith</td>
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<td>Activity and Gene Expression</td>
<td>Rimer</td>
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<td>Cytoskeleton</td>
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<td>Monday, October 3, 16</td>
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<td>Protein Synthesis and Processing</td>
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<td>Intracellular Vascular Transport</td>
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<td>Endocytosis and Excocytosis/Neurotransmitter release</td>
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<td>Round Table Discussion I</td>
<td>Rimer</td>
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<td>Round Table Discussion II</td>
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<td>The cell biology of myelination</td>
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<td>Neuroimmunology</td>
<td>Welsh</td>
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<td>Roles of glia in buffering and metabolic support</td>
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<td>Gliotransmission and the role of astrocytes in neural cell communications</td>
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<td>Neural patterning in development</td>
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<td>Axon guidance in development</td>
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<td>Methods for imaging neurons in action</td>
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<td>Synapse formation and remodeling at THE CNS</td>
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<td>Methods for manipulating neurons with light</td>
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Total hours = 38
All Exams (100.00%)

Afternoon Take Home Exam #1*

Exam 1 % of total

21 60

Exam 2 % of total

14 40

Total

Exam 1  Exam 2

25 75

100.00%

Academic Program Review, May 2017

Texas A&M Institute for Neuroscience 169
APPENDIX M

Principles of Neuroscience II
Syllabus
NRSC 602 (Biol 628) Principles of Neuroscience – Part 2 Course Schedule
Spring 2017

3 credit hours: Tuesdays, Thursdays  11:10-12:15; Conference Rm 3143, ILSB, TAMU
Course Coordinators:
Wesley Thompson (3214 ILSB) wthompson@bio.tamu.edu
Jim Grau (3149A ILSB) J-Grau@tamu.edu
Mike Smotherman (107 Biological Sciences Bldg. West) msmoth@bio.tamu.edu
Recommended Text: TBA

Final Grade Point Breakdown:
Midterm   45%
Final Exam   45%
Discussion/Participation 10%

Exams: Both exams will be take-home exams w/13 questions (roughly one for each lecture). Students will have 24 hours to complete the exam and must answer 12 of the 13 questions.

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<tr>
<th>Date</th>
<th>Week</th>
<th>Subject</th>
<th>Reading</th>
<th>Instructor</th>
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<tr>
<td>17-Jan</td>
<td>1</td>
<td>Organization of sensory systems</td>
<td>Squire Chap. 22</td>
<td>Smotherman</td>
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<td>19-Jan</td>
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<td>Information Coding in spike trains</td>
<td>Spikes/Arabzadeh handout</td>
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<tr>
<td>24-Jan</td>
<td>3</td>
<td>Chemical Senses</td>
<td>Squire Chap. 23</td>
<td>Smotherman</td>
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<td>Somatosensation</td>
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<td>31-Jan</td>
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<td>Hearing and Balance-Peripheral mechanisms</td>
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<td>Smotherman</td>
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<td>2-Feb</td>
<td>6</td>
<td>Hearing-Central pathways</td>
<td>Squire Chap. 25</td>
<td>Smotherman</td>
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<td>7-Feb</td>
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<td>Vision-Retina</td>
<td>Squire Chap 26</td>
<td>Thompson</td>
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<td>9-Feb</td>
<td>8</td>
<td>Vision-Central pathways</td>
<td>Squire Chap 26</td>
<td>Thompson</td>
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<td>14-Feb</td>
<td>9</td>
<td>Motor Systems-Central Pattern Generators</td>
<td>Squire Ch 29; Luo Ch 8</td>
<td>Thompson</td>
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<td>10</td>
<td>Spinal Cord and Brainstem</td>
<td>Squire Ch 27, 28</td>
<td>Thompson</td>
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<td>21-Feb</td>
<td>11</td>
<td>Basal Ganglia-Anatomy, Circuits, Disease</td>
<td>Squire, Ch 30</td>
<td>Thompson</td>
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<td>28-Feb</td>
<td>13</td>
<td>Autonomic Nervous System</td>
<td>Squire Chpt. 36</td>
<td>Wellman</td>
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<td>2-Mar</td>
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<td>Hypothalamus and SCN</td>
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<td>Zoran</td>
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<td>Motivation and Feeding</td>
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<td>Learning</td>
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<td>30-Mar</td>
<td>22</td>
<td>Neurobiology of learning &amp; memory</td>
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<td>Neurobiology of learning &amp; memory</td>
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<td>Addiction</td>
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<td>Bolanos</td>
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<td>Sensation &amp; perception</td>
<td>Squire Chpt. 51</td>
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<td>27</td>
<td>Sensation &amp; perception</td>
<td>Squire Chpts. 44 &amp; 45</td>
<td>Anderson</td>
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<td>20-Apr</td>
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<td>Neuroimaging Methods</td>
<td>Gazzaniga Chapter</td>
<td>Orr</td>
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<tr>
<td>25-Apr</td>
<td>29</td>
<td>Cognitive Neuroscience</td>
<td>Squire Ch. 46 &amp; 49</td>
<td>Worthy</td>
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<td>27-Apr</td>
<td>30</td>
<td>Executive Function</td>
<td>Squire Chpt. 50</td>
<td>Orr/Worthy</td>
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<td>2-May</td>
<td>31</td>
<td>Aging</td>
<td>Kandel &amp; Schwartz, Chpt. 59</td>
<td>Bernard</td>
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APPENDIX N

Graduate Annual Meeting Evaluation Form
Interdisciplinary Graduate Program in Neuroscience

Annual Advisory Committee Meeting Evaluation Form

Student Name__________________________ Meeting Date__________

The following items were discussed at this meeting:

On a scale of 1 to 5 please rate how this student is progressing in each of the five categories, with 1 being Insufficient and 5 being Excellent. Refer to the attached rubric for assessment criteria. The committee members should discuss each criteria separately and the chair documents the committee’s rating.

<table>
<thead>
<tr>
<th>Mastery of Fundamental Concepts</th>
<th>Good</th>
<th>Excellent</th>
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<table>
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<tr>
<th>Interpreting and Applying Scientific Literature</th>
<th>Good</th>
<th>Excellent</th>
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<th>Developing Hypotheses</th>
<th>Good</th>
<th>Excellent</th>
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<th>Experimental Design and Execution</th>
<th>Good</th>
<th>Excellent</th>
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<th>Communication</th>
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Specific comments and recommendations:

________________________________________    ______________________________________
Committee Chair         Student

________________________________________    Acknowledged:
Committee Member         Graduate Advisor

________________________________________
Committee Member

________________________________________
Committee Member

________________________________________
Committee Member

revised 9/24/2015
**Rubric for Assessing Categories**

**Mastery of Fundamental Concepts:** By the time the students have their first committee meeting they should have already completed the core coursework in neuroscience, Principles of Neuroscience I & II, and have received additional training and experience through elective courses, journal clubs, lab meetings and seminar attendances. For beginning (1st and 2nd year) students, competency of fundamental concepts should be assessed from the students presentation of their research interests and goals, and if necessary from additional questions presented by the committee members. For beginning students, a score of 3 or more indicates that the committee has confidence in the student’s mastery of fundamental concepts taught in the core course. As students advance and their exposure to the literature increases these scores are expected to increase reflecting mastery of concepts specific to their course of study.

**Interpreting and Applying Scientific Literature:** Beginning students should at minimum show evidence that they have surveyed the literature, are able to define the current state of their chosen field, and can identify recent publications that will guide and influence their research. This level of competence would be rated a 3. As students progress and develop more focused research proposals, a broader and deeper knowledge of the literature is expected to warrant higher ratings. During annual evaluation meetings, committee members should challenge the students’ ability to accurately reference relevant literature during their presentations and in response to specific questions.

**Developing Hypotheses:** A central component of the training program is to teach students the inherent importance of hypothesis-driven research. Students will be expected to identify key questions in their field, develop a testable hypothesis to address the question(s), and present a cogent rationale based on their interpretation of the literature. Beginning students should demonstrate that they are at least thinking about their research in the context of hypothesis testing to receive a rating of 3. As students advance in their research, evidence of experience developing and defending testable hypotheses should be apparent in their presentation. Additional questioning by the committee may be appropriate to help clarify the student’s skill level before assigning the rating.

**Experimental Design and Execution:** Beginning students should be evaluated based on their ability to identify the tools and techniques that they will need to pursue their research interests. A rating of 3 or better would indicate that the student has demonstrated a basic knowledge of experimental design and research planning. As students progress, they should demonstrate greater levels of competency in this area and direct evidence of having completed experiments. Note: a student may display sufficient knowledge of experimental design and execution to complete their research and graduate without necessarily being rated “excellent” in this skill.

**Communication:** The committee is asked to evaluate the student’s ability to communicate their research ideas and finding, verbally and written. A rating of 3 or better indicates that the student is making satisfactory progress towards becoming an effective writer/speaker. These scores are expected to improve with experience during the course of training: students do not need to be rated “excellent” to graduate, and critical feedback even in later stages of the degree program may help the students advance their careers.
APPENDIX O

TAMIN Seminar Series
<table>
<thead>
<tr>
<th>Date</th>
<th>Speaker</th>
<th>Title</th>
<th>Host</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sep 8</td>
<td>Lawrence Judson Chandler</td>
<td>Alcohol Abuse and the Prefrontal Cortex</td>
<td>Jun Wang</td>
</tr>
<tr>
<td></td>
<td>Medical Univ. of South Carolina</td>
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</tr>
<tr>
<td>Sep 15</td>
<td>Doo-Sup Choi</td>
<td>Adenosine and Glutamate Signaling in Alcoholism: Translation from Bench to Bedside</td>
<td>Jun Wang</td>
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<tr>
<td></td>
<td>Mayo Clinic</td>
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</tr>
<tr>
<td>Sep 22</td>
<td>Joseph M. Orr</td>
<td>Disruptions of Prefrontal Brain Dynamics During Novel Task Learning in Adolescents at Risk for Psychosis</td>
<td>Ursula Winzer-Serhan</td>
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<tr>
<td></td>
<td>Texas A&amp;M &amp; University</td>
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<td>NEXT</td>
</tr>
<tr>
<td></td>
<td>Psychology Department</td>
<td></td>
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</tr>
<tr>
<td>Sep 29</td>
<td>David Morlak</td>
<td>Prefrontal cortical function in models of psychiatric pathology and novel therapeutic efficacy</td>
<td>Rachel Smith</td>
</tr>
<tr>
<td></td>
<td>UT Health Science Center</td>
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<td>PSYC</td>
</tr>
<tr>
<td></td>
<td>San Antonio</td>
<td></td>
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<tr>
<td>Oct 6</td>
<td>Jeffrey Chamberlain</td>
<td>Vectors for Gene Therapy of DMD</td>
<td>Joe Kornegay</td>
</tr>
<tr>
<td></td>
<td>University of Washington</td>
<td></td>
<td>VIBS</td>
</tr>
<tr>
<td>Oct 13</td>
<td>Jonathan D. Hommel</td>
<td>Therapeutic Targets for the Addictive Dimensionality of Obesity</td>
<td>Paul Wellman</td>
</tr>
<tr>
<td></td>
<td>University of Texas Medical Branch</td>
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<td>PSYC</td>
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<tr>
<td>Oct 20</td>
<td>Emanuel C. Mora</td>
<td>Bat echolocation vs. moth hearing: evolution of tactics and counter tactics</td>
<td>U.J. McMahan</td>
</tr>
<tr>
<td>Oct 27</td>
<td>University of Florida Health</td>
<td></td>
<td>BIOL</td>
</tr>
<tr>
<td>Nov 3</td>
<td>Graduate Student Presentations</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nov 10</td>
<td>Lee Sweeney</td>
<td>Possible targets and translational challenges for muscular dystrophy therapies</td>
<td>Wes Thompson</td>
</tr>
<tr>
<td>Nov 17</td>
<td>Graduate Student Presentations</td>
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<td></td>
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<tr>
<td>Nov 24</td>
<td>Richard R. Dubielzig</td>
<td>Optic Nerve Pathology</td>
<td>Erin M. Scott</td>
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<tr>
<td>Dec 1</td>
<td>University of Wisconsin-Madison</td>
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<td>VSCS</td>
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<tr>
<td>Dec 6</td>
<td>Kimberly Nixon</td>
<td>Gliosis in models of alcoholism: Roles in neuro degeneration regenerati</td>
<td>Rajesh Miranda</td>
</tr>
<tr>
<td></td>
<td>University of Kentucky</td>
<td></td>
<td>NEXT</td>
</tr>
<tr>
<td>Dec 8</td>
<td>Hasan Ayaz</td>
<td>Functional near infrared spectroscopy: Basic Principles and Applications from Aerospace, Medicine, and to Clinical Life-saving Solutions</td>
<td>Ranjana Mehta</td>
</tr>
<tr>
<td></td>
<td>Drexel University</td>
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<td>PHEO</td>
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<tr>
<td>Jan 19</td>
<td>Jin Hyung Lee</td>
<td>Optogenetic fMRI and the investigation of global brain circuit mechanisms</td>
<td>Yoonsuck Choe</td>
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<td></td>
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<td>CSCE</td>
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<tr>
<td>Feb 2</td>
<td>Kimberly Nixon</td>
<td>Gliosis in models of alcoholism: Roles in neuro degeneration regenerati</td>
<td>Rajesh Miranda</td>
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<td></td>
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<td>NEXT</td>
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<tr>
<td>Feb 9</td>
<td>Hasan Ayaz</td>
<td>Functional near infrared spectroscopy: Basic Principles and Applications from Aerospace, Medicine, and to Clinical Life-saving Solutions</td>
<td>Ranjana Mehta</td>
</tr>
<tr>
<td></td>
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<td>PHEO</td>
</tr>
<tr>
<td>Feb 16</td>
<td>Jessica Bernard</td>
<td>Cerebellar Networks and Symptom Severity in Ultra-High Risk Adolescents and Patients with Schizophrenia</td>
<td>Ursula Winzer-Serhan</td>
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<td>Texas A&amp;M &amp; University</td>
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<tr>
<td>Feb 23</td>
<td>Sergiu P. Pasca</td>
<td>Piece by piece: building a human cerebral cortex in a dish</td>
<td>U.J. McMahan</td>
</tr>
<tr>
<td></td>
<td>Stanford University</td>
<td></td>
<td>BIOL</td>
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<tr>
<td>Mar 2</td>
<td>Marisa Roberto</td>
<td>Amygdala Dysregulation in Alcohol Dependence</td>
<td>Jun Wang</td>
</tr>
<tr>
<td></td>
<td>The Scripps Research Institute</td>
<td></td>
<td>NEXT</td>
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<tr>
<td>Mar 9</td>
<td>Hans Linsenbardt</td>
<td></td>
<td>Ian Smith</td>
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<td>University of Florida Health</td>
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<td>TMIN</td>
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<td>Mar 16</td>
<td>Graduate Student Presentations</td>
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<td>Mar 23</td>
<td>Jonathan B. Cohen</td>
<td>GABA,Rs and general anesthetics: illuminating drug-receptor interactions</td>
<td>Ayman Hamouda</td>
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<td>Harvard Medical School</td>
<td></td>
<td>PHAR</td>
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<td>Mar 30</td>
<td>Wei-Jung Chen</td>
<td>Research Reproducibility: Is It Real and How to Fix it?</td>
<td>Farida Sohrabji</td>
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<td>Texas A&amp;M Health Science Center</td>
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<tr>
<td></td>
<td>Neuroscience and Experimental</td>
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<td></td>
<td>Therapeutics</td>
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<tr>
<td>Apr 6</td>
<td>Maria Siemonow</td>
<td>Cellular therapies in reconstructive transplantation</td>
<td>Joe Kornegay</td>
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<td>University of Illinois at Chicago</td>
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<td>VIBS</td>
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<td>Apr 13</td>
<td>Marina Guizzetti</td>
<td>Astrocyte-neuron interactions in Fetal Alcohol Spectrum Disorders</td>
<td>Rajesh Miranda</td>
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<td>Oregon Health &amp; Science</td>
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<tr>
<td></td>
<td>University</td>
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<tr>
<td>Apr 20</td>
<td>Nicholas J. Priebe</td>
<td>Common Strategies for information processing in across the cerebral cortex</td>
<td>Wes Thompson</td>
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<td>University of Texas</td>
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<td>Apr 27</td>
<td>TMIN Business Meeting</td>
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<td>Jane Welsh</td>
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<td>Apr 28</td>
<td>9th Annual Spring Symposium</td>
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<td>Interdisciplinary Life Sciences Building</td>
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<td>May 15</td>
<td>TMIN Business Meeting</td>
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<tr>
<td>May 15</td>
<td>TMIN External Review</td>
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Society for Neuroscience, San Diego, CA
Nov 12-16, 2016

No Seminar—Thanksgiving
<table>
<thead>
<tr>
<th>Date</th>
<th>Speaker</th>
<th>Title</th>
<th>Host</th>
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<tr>
<td>Sep 03</td>
<td>Tamin Social Faculty Meeting</td>
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<td>Welsh</td>
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<tr>
<td>Sep 10</td>
<td>Rachel Smith</td>
<td>Texas A&amp;M University Stress-associated neural systems and addiction</td>
<td>Ursula Winzer-Serhan NExT</td>
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<td>Sep 17</td>
<td>Graduate Student Presentations</td>
<td>Joel Turtle C fiber activation undermines recovery after spinal cord injury</td>
<td>Jim Grau PSYC</td>
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<tr>
<td>Sep 24</td>
<td>Paul Garrity</td>
<td>Brandeis University Temperate Sensing and Pain Perception</td>
<td>Hubert Amrein MCMD</td>
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<tr>
<td>Oct 1</td>
<td>Greg Bix</td>
<td>University of Kentucky The role and therapeutic potential of the extracellular matrix and its receptors in stroke and vascular dementia</td>
<td>Farida Sohrabji NExT</td>
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<tr>
<td>Oct 8</td>
<td>Naoshige Uchida</td>
<td>Harvard University Arithmetic and neural circuits underlying dopamine prediction error</td>
<td>Rachel Smith PSYC</td>
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<tr>
<td>Oct 15</td>
<td>Lee Shapiro</td>
<td>Dept of Surgery &amp; Neurosurgery Texas A&amp;M HSC Peripheral and neuroinflammatory contributions to neuropathology following traumatic brain injury</td>
<td>Ursula Winzer-Serhan NExT</td>
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<tr>
<td>Oct 22</td>
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<tr>
<td>Oct 29</td>
<td>Gina Bertocci</td>
<td>University of Louisville Development of a neuromusculoskeletal computer simulation gait model to characterize functional recovery in dogs with intervertebral disk herniation</td>
<td>Jonathan Levine VIBS</td>
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<tr>
<td>Nov 5</td>
<td>Annemieke Kavelaars</td>
<td>Canceled</td>
<td>Mary Meagher PSYC</td>
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<tr>
<td>Nov 12</td>
<td>Kathryn Wagner</td>
<td>Johns Hopkins School of Medicine Myostatin Inhibition for Muscle Disease: Helpful or Harmful?</td>
<td>Joe Kornegay VIBS</td>
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<tr>
<td>Nov 19</td>
<td>Kendal Broadie</td>
<td>Vanderbilt Drosophila Modeling of Galactosemia Neurological Symptoms: Glycosylation Roles in Synaptogenesis</td>
<td>Vlad Panin BICH</td>
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<td>Nov 26</td>
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<tr>
<td>Dec 3</td>
<td>SFN Poster Session</td>
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<tr>
<td>Jan 28</td>
<td>Raquel Sitcheran</td>
<td>Department of Molecular &amp; Cellular Medicine Texas A&amp;M HSC NIK-ing mitochondria to promote glioma cell migration and invasion</td>
<td>Ursula Winzer-Serhan NExT</td>
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<tr>
<td>Feb 04</td>
<td>David Lovinger</td>
<td>NIAAA Synaptic Actions of Alcohol and Drugs of Abuse in Dorsal Striatum: Relation to Habit Formation</td>
<td>Jun Wang NExT</td>
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<tr>
<td>Feb 11</td>
<td>Nicole Wicha</td>
<td>UTSA Arithmetic in the Bilingual Brain Jyotsna Vaid PSYC</td>
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<tr>
<td>Feb 18</td>
<td>George Stoica</td>
<td>Vanderbilt Impact of Myosin 5a mutation in neurodegeneration: A rat animal model</td>
<td>Ursula Winzer-Serhan NExT</td>
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<td>Feb 25</td>
<td>Tamin Business Meeting</td>
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<td>Mar 03</td>
<td>Boris V. Zemelman</td>
<td>University of Texas Unexpected effects of presenilin dysfunction on neuronal plasticity and learning</td>
<td>Steve Maren PSYC</td>
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<td>Mar 10</td>
<td>Graduate Student Presentations</td>
<td>Mike Emery Miriam Aceves Tamin</td>
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<td>Mar 24</td>
<td>Michael N. Nitabach</td>
<td>Yale School of Medicine Unexpected effects of presenilin dysfunction on neuronal plasticity and learning</td>
<td>Hubert Amrein MCMD</td>
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<tr>
<td>Mar 31</td>
<td>Nace Golding</td>
<td>University of Texas at Austin Illuminating mechanisms of sensory coding and neureomodulation with light-sensitive channel blockers</td>
<td>Wes Thompson BIOI</td>
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<tr>
<td>Apr 07</td>
<td>Yan Dong</td>
<td>University of Pittsburg Synaptic remodeling of drug craving; the neural rejuvenation hypothesis</td>
<td>Jun Wang NExT</td>
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<td>2015 Annual Spring Symposium</td>
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<td>Apr 21</td>
<td>Maria Braga</td>
<td>Uniformed Services University of the Health Sciences The anticonvulsant and neuroprotective efficacy of GluK1 Kainate Receptor antagonists: a novel therapeutic against nerve agent-induced seizures and brain damage</td>
<td>Samba Reddy NExT</td>
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<td>Apr 28</td>
<td>Tamin Business Meeting</td>
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</table>
APPENDIX P

TAMIN Travel Grant Application Form
Application for TMIN Travel Award
Submit application with original signatures only; all applications must be typed

Name: ___________________________________________ UIN: ______________ Date: __________
Last          First          MI

Email: ________________________ Phone: ____________________ Dept: ________________

***Reimbursement for TMIN Travel Awards will be posted to your TAMU account in the form of a scholarship. If you have a balance due, it will be deducted from that posting. Any credit amount remaining will then be sent to you.

This travel is: _______ Domestic (within USA) or _________ International

Overview: Attach a copy of the letter of invitation to present your poster/paper; describe below 1) why it is important for you to present at this meeting and 2) how it relates to your degree program. (You may apply before receiving official acceptance to conference).

Title of Paper/Abstract: ______________________________________________________________

Society/Conference Name (no acronyms, spell out): ____________________________________________

Dates of travel: ____________________________ Location: _________________________________________

Itemized budget; be specific on the items below.
Airfare/transportation ____________
Hotel/housing ________________
Registration fee ____________
Meals ________________
Other ________________

Total Requested ____________________________

Approvals (Signatures here verify that the applicant’s PI is a member of the Faculty of Neuroscience)

Signature of applicant ____________________________ Printed Name ____________________________

Signature of PI ____________________________ Printed Name ____________________________

Signature of Dept Head/IDP Chair ____________________________ Printed Name ____________________________

For TAMIN use only

GPR: ____________  ___Approved ___ Not approved-why: __________________________________________
Full-Time: ________
Proposal Submitted ________ Amount Awarded $ ____________
Funds must be spent by: ____________________________ Approved by ____________________________ Date ____________________________
APPENDIX Q

TAMIN 18 Characteristics
### Characteristics of Texas Public Doctoral Programs

Texas A&M University

18 Characteristics of Texas Public Doctoral Programs

Programs included only if in existence 3 or more years. Program is defined at the 8-digit CIP code level.

<table>
<thead>
<tr>
<th>Department</th>
<th>Texas A&amp;M Institute for Neuroscience</th>
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</thead>
<tbody>
<tr>
<td>Doctoral Degree Program</td>
<td>Neuroscience</td>
</tr>
<tr>
<td>Contact Name</td>
<td>Sylvia M. Bernal</td>
</tr>
<tr>
<td>Contact Phone Number</td>
<td>979-458-0214</td>
</tr>
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</table>

#### Number of Degrees Per Year

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<tr>
<th>Year</th>
<th>Number of Degrees</th>
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<tbody>
<tr>
<td>2012-2013</td>
<td>4</td>
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<tr>
<td>2013-2014</td>
<td>2</td>
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<tr>
<td>2014-2015</td>
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#### Graduation Rates

Starting Cohorts: 2003-2005

<table>
<thead>
<tr>
<th>Year</th>
<th>% Graduating within 10 Years</th>
<th>Years with Cohort greater than 0</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>0%</td>
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</tbody>
</table>

#### Average Time to Degree

Students Starting 2003-2005

<table>
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<th>Year</th>
<th>Average Years to Degree</th>
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<tbody>
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<td>0.0</td>
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#### Employment Profile

(In field within one year of graduation). For each of the three most recent years, the number and percent of graduates by year employed, those still seeking employment, and unknown

<table>
<thead>
<tr>
<th>Year</th>
<th>Employed Number</th>
<th>Employed Percent</th>
<th>Still Seeking Employment Number</th>
<th>Still Seeking Employment Percent</th>
<th>Unknown Number</th>
<th>Unknown Percent</th>
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<tr>
<td>2012-2013</td>
<td>4</td>
<td>100%</td>
<td>0</td>
<td>0%</td>
<td>0</td>
<td>0%</td>
</tr>
<tr>
<td>2013-2014</td>
<td>2</td>
<td>100%</td>
<td>0</td>
<td>0%</td>
<td>0</td>
<td>0%</td>
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<tr>
<td>2014-2015</td>
<td>2</td>
<td>100%</td>
<td>0</td>
<td>0%</td>
<td>0</td>
<td>0%</td>
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#### Admissions Criteria

Overall admissions criteria are based on the entire record of the applicant and availability of departmental resources. Admission will be based upon the following criteria: 1) Hold a four-year baccalaureate degree from a college or university of recognized standing (i.e., a degree recognized as equivalent to a baccalaureate degree from an accredited institution in the U.S.), overall transcript evaluation, and grade point ratio in the last 60 hours of coursework. 2) Show promise of intellectual and academic ability, a minimum of three letters of recommendation from persons capable of judging the applicant's capabilities, and an evaluation of the Statement of Purpose essay. 3) Submit, with application, scores on the General Test of the Graduate Record Examination (GRE), which will be evaluated in the manner that complies with House Bill 1641. 4) Additional at the program level, an applicant from another country seeking admission to graduate studies must demonstrate the ability to read, write, speak and understand the English language. Prospective students whose native language is not English must take the Test of English as a Foreign Language (TOEFL), which is administered by the Educational Testing Service in over 200 centers around the world. All applicants from non-English speaking countries must present a computer based TOEFL score of at least 213 to be admitted to graduate studies at the University.

#### Percentage Full-time Students

FTS/number of students enrolled for the last three fall semesters.

<table>
<thead>
<tr>
<th>Year</th>
<th>Percentage</th>
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<tr>
<td>Fall 2012</td>
<td>100.0%</td>
</tr>
<tr>
<td>Fall 2013</td>
<td>100.0%</td>
</tr>
<tr>
<td>Fall 2014</td>
<td>100.0%</td>
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</tbody>
</table>

#### Average Institutional Financial Support Provided

For those receiving financial support, the average monetary institutional financial support provided per full-time graduate student for the prior year, from assistantships, scholarships, stipends, grants, and fellowships. Does not include tuition or benefits.

$21,209.74
### Percentage Full-Time Students with Institutional Financial Support

In the prior year, the number of full-time students with at least $1,000 of annual support/the number of full-time students = 100%

### Number of Core Faculty

Number of core faculty in the prior year = 97

### Student-Core Faculty Ratio

Three-year average of full-time student equivalent (FTSE)/three-year average of full-time faculty equivalent (FTFE) of core faculty. Core Faculty: Full-time tenured and tenure-track faculty who teach 50 percent or more in the doctoral program or other individuals integral to the doctoral program who can direct dissertation research.

### Core Faculty Publications

Three-year average of the number of discipline-related refereed papers/publications, books/book chapters, juried creative/performance accomplishments, and notices of discoveries filed/patents issued per year per core faculty member.

### Core Faculty External Grants

Three-year average of the number of core faculty receiving external funds, average external funds per faculty, and total external funds per program per academic year. All external funds received from any source including research grants, training grants, gifts from foundations, etc., reported as expenditures.

| Average of the Number of Core Faculty receiving | 35.33 |
| Average External Funds per Faculty | $261,658.3 |
| Total External Funds | $27,727,873.96 |

### Faculty Teaching Load

Total number of semester credit hours in organized teaching courses taught per academic year by core faculty divided by the number of core faculty in the prior year = 8.3

### Faculty Diversity

Core faculty by ethnicity (White, Black, Hispanic, Other) and gender, updated when changed

<table>
<thead>
<tr>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>White</td>
<td>42</td>
</tr>
<tr>
<td>Black</td>
<td>2</td>
</tr>
<tr>
<td>Hispanic</td>
<td>2</td>
</tr>
<tr>
<td>Other</td>
<td>18</td>
</tr>
</tbody>
</table>

### Student Diversity

Enrollment headcount by ethnicity (White, Black, Hispanic, Other) and gender in program in the prior year

<table>
<thead>
<tr>
<th>Fall 2014</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>White</td>
<td>9</td>
<td>2</td>
</tr>
<tr>
<td>Black</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Hispanic</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Other</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>

### Date of Last External Review

Date of last formal external review, updated when changed = N/A

### External Program Accreditation

Name of body and date of last program accreditation review, if applicable, updated when changed

| N/A - University Accreditation through SACS |

### Student Publications/Presentations

For the three most recent years, the number of discipline-related refereed papers/publications, juried creative/performance accomplishments, book chapters, books, and external presentations per year by student FTE

| 12.04 |
Please contact the TAMIN Office if additional information is needed regarding stats. Sylvia M. Bernal (979) 458-0214 sylviabernal@tamin.tamu.edu

**Notes:**
The sum of #14 (Faculty Diversity) could be less than #9 (Number of Core Faculty) if some faculty have chosen to keep their information confidential.

For this reporting cycle of Academic Year 2014-15, the enrollment data was pulled by G8 Classification, not including the G7 Students whose degree objectives were doctoral. The definition of G7 and G8 classification could be found at [http://catalog.tamu.edu/graduate/academic-expectations-general-degree-requirements/registration-academic-status/](http://catalog.tamu.edu/graduate/academic-expectations-general-degree-requirements/registration-academic-status/).
APPENDIX R

Student Handbook
# Institute for Neuroscience Graduate Program Handbook

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APPENDIX II: Office of Graduate and Professional Studies Forms
Online versions and instructions for all OGAPS forms can be found at
http://ogaps.tamu.edu/Buttons/Forms-Information
Texas A&M’s Interdisciplinary Program (IDP) in Neuroscience is administered by the Institute for Neuroscience (TAMIN). The program is dedicated to providing multidisciplinary training for future neuroscientists wishing to pursue careers in higher education, government, medicine or private industry. The Office of Graduate and Professional Studies (OGAPS) establishes the minimal University guidelines for all graduate degrees. TAMIN has established additional requirements that all students must satisfy. **It is the responsibility of the graduate student to ensure that all departmental and university requirements for the degree are met.** Please note that graduate students must fulfill the requirements of the catalog that is current during the semester they complete their degree requirements. This is the case for both University and TAMIN requirements. It is the student’s responsibility to keep up with changes in requirements.

This book provides the TAMIN requirements and a summary of University requirements; a complete description of the university requirements can be found in the Graduate Catalog (http://catalog.tamu.edu/graduate/academic-expectations-general-degree-requirements/). This handbook and a copy of the Graduate Catalog should be reviewed throughout the progress of the degree. Additional information can be obtained from the Office of Graduate and Professional Studies and the TAMIN Graduate Advising Office, located in ILSB Room 3148.

### DEADLINES for GRADUATE DEGREE REQUIREMENTS

TAMIN has established the following deadlines for students enrolled in the Neuroscience IDP.

<table>
<thead>
<tr>
<th>TAMIN DEADLINES</th>
<th>Ph.D. Degree</th>
</tr>
</thead>
<tbody>
<tr>
<td>Choice of Thesis Advisor</td>
<td>By the end of the 2nd Semester</td>
</tr>
<tr>
<td>Establish Advisory Committee</td>
<td>Before fall semester of 2nd Year</td>
</tr>
<tr>
<td>Degree Plan Filed with Department</td>
<td>Before fall semester of 2nd Year</td>
</tr>
<tr>
<td>Proposal Draft submitted to Advisor</td>
<td>End of spring semester of 2nd Year</td>
</tr>
<tr>
<td>Proposal Filed with Department</td>
<td>During the 5th Semester (see page 19)</td>
</tr>
<tr>
<td>Preliminary Exam</td>
<td>During the 5th Semester (see page 20)</td>
</tr>
<tr>
<td>Final Examination</td>
<td>Within Four Years of Completing the Prelim Exam</td>
</tr>
</tbody>
</table>

Note that the number of semesters does NOT include summer sessions.

### SUBMITTING REQUIRED PAPERWORK:

It is also requested that students submit copies of all completed paperwork (with signatures) to the TAMIN Graduate Advising Office. The Graduate Advising Office is available to review and provide assistance in filling out documents, obtain
signatures from the Program Chair or Graduate Advisor, log in the paperwork, make copies for your
departmental file, and as necessary serve as a liaison between the student and OGAPS. TAMIN-
specific forms, such as the TAMIN annual committee meeting evaluation form should only be
submitted directly to the TAMIN advising office.

*IT IS THE STUDENT’S RESPONSIBILITY TO OBTAIN ANY NECESSARY SIGNATURES
AND ENSURE THAT OGAPS RECEIVES COPIES OF ALL PAPERWORK. THE THESIS
ADVISOR IS RESPONSIBLE FOR ENSURING DELIVERY OF FINAL EXAM REPORT.*

The **GRADUATE ADVISOR** is the person currently serving as chair of the TAMIN Graduate
Program Committee. During the student’s first year, the Graduate Advisor serves in an advisory
capacity for all entering students in helping them identify and choose electives most likely to be
relevant to their area of interest, identify and initiate discussions with potential faculty advisors,
identify and coordinate suitable rotation opportunities, and otherwise address any questions or
issues that might arise before the student has chosen a thesis advisor (page 7). After the first year,
the Graduate Advisor continues to serve in an advisory capacity to both students and graduate
faculty as needed. Petitions to exempt coursework or any other requests to modify program
requirements are submitted to through the graduate advisor to the graduate program committee,
which evaluates each request.

The **GRADUATE PROGRAM COMMITTEE (GPC)** is a group of faculty members elected by
the TAMIN graduate faculty members to review and update guidelines for the NRSC graduate
program. The GPC is assigned the role of ensuring that NRSC requirements are consistent with all
University requirements and that the training program continues to meet the evolving needs of
TAMIN faculty and students. The GPC reviews and approves which courses may serve as
approved electives towards the NRSC graduate degree. The GPC evaluates all requests or petitions
for deviations from prescribed program requirements and also monitors at the programmatic level
how students are progressing towards degree completion. If students encounter problems or
concerns in their relationship with their thesis advisor or thesis committee, they are encouraged to
bring those concerns to the attention of the Graduate Advisor and/or GPC for advice on how to
resolve the problems. Students bringing concerns or complaints to the Graduate Advisor or GPC
may do so with assurance of confidentiality.

**PROGRESS TOWARDS THE DEGREE**

All graduate students must adhere to the requirements set forth by the program in order to
remain in good standing. This includes adhering to all major milestones, holding annual committee
meetings, submitting annual reports and regular attendance at courses and seminars. If a student has
not met the required TAMIN or University deadlines as specified in this handbook or the Graduate
Catalog, they will no longer be in good standing with the department and may be blocked from
registration the following semester and become ineligible for financial support. The block will not be
lifted until the requirement is met. Requests for exemptions will be considered by the Graduate
Advisor in consultation with the Graduate Programs Committee on a case-by-case basis.
GETTING STARTED

1. RESEARCH ROTATIONS

TAMIN requires all incoming students to complete two seven-week (half-semester) laboratory rotations during their first semester. Rotations acquaint new students with research programs in their areas of interest, broaden the student’s perspective on approaches and procedures used in modern neuroscience, provide hands-on experience with state-of-the-art technology, and can serve as the foundation for useful contacts in other labs. At the conclusion of rotations, a major professor is chosen by the mutual consent of the graduate faculty member and the student. To receive academic credit for rotations students should enroll in NRSC 685 for two credits in the fall. Students entering the PhD program with a Master’s degree or other advanced degree (PhD, MD, DO, DVM, etc) may waive the rotations requirement if they have already identified a thesis advisor and the advisor requests the exemption in writing to the Graduate advisor. An email providing justification for the exemption is normally sufficient but all requests will be reviewed by the Graduate Program Committee. Students must submit the Rotations Form to the TAMIN Graduate Advising office by the end of the second week of the semester identifying where they will do their rotations and including the signatures of the faculty mentors. Students may register for a third rotation in the spring semester if needed (1 credit of NRSC 685).

2. REQUIRED COURSE WORK FOR 1st-YEAR STUDENTS

All TAMIN graduate students are required to enroll in the following graduate courses during their first year.

a) Principles of Neuroscience I. NRSC 601/Biol 627 is a foundation course covering fundamental details in cellular, molecular and developmental neuroscience. Topics include membrane potentials, action potentials and the mechanisms of synaptic transmission. The course also requires participation in an affiliated weekly journal club (NRSC 681-602, Seminar: Principles in NRSC) that teaches students how to evaluate and critique recent advances in the neuroscience literature.

b) Principles of Neuroscience II. NRSC 602/Biol 628 is a fully integrated overview of nervous system functional organization and systems-level neurobiology. Topics include sensory systems, motor systems, neuromuscular control, central pattern generators, locomotion, homeostatic regulation, circadian rhythms, motivation, emotions, learning and memory, and cognition. The course also requires participation in an affiliated weekly journal club that teaches students how to evaluate and critique recent advances in the neuroscience literature.

c) NRSC 685, Directed Studies (Rotations), in order to receive credit hours for their participation in faculty-supervised laboratory rotations. Satisfactory/Unsatisfactory grades will be assigned by the Graduate Advisor based on faculty evaluation of a student’s performance in each rotation.

d) NRSC 681, Departmental Colloquium each semester. This is TAMIN’s weekly neuroscience seminar series featuring guest experts in the field presenting and discussing recent research. Students must sign the attendance sheet before the beginning of the seminar to receive
credit for attendance. If a student misses more than three seminars during the semester they will receive an unsatisfactory (U) grade and may be ineligible for travel funds or other forms of support for the succeeding semester/year. For excused absences, limited opportunities for make-up credit are available through the TAMIN advising office at the discretion of the Graduate Advisor.

3. ADDITIONAL COURSE REQUIREMENTS

a) Ethics and Responsible Conduct of Research. This should be a one-credit course that meets weekly to engage in discussions of how to recognize, and avoid committing, fraud in science. Topics should include scientific ethics, negotiation techniques, plagiarism, record keeping, data management, peer review, conflict management, and the regulations covering animal and human experiments. There are several acceptable options available through different participating departments on campus, including but not limited to BIOL 696, MSCI 609, VMID 686, or BIMS 5126. Contact the Graduate Advisor to determine if a new or related course meets this requirement. This requirement is intended to ensure students are eligible to receive federal support for training and research, and therefore courses must meet requirements as set forth by the NIH (https://oir.nih.gov/sourcebook/ethical-conduct/responsible-conduct-research-training). It is in the best interest of the student to complete this requirement as soon as possible, pending course availability, but must be completed prior to graduating.

b) Statistics and Experimental Design. Several approved options are available in participating departments that meet this requirement. Check with your home department and advisor to identify the course that best complements to research plan. This requirement may be waived for students entering the PhD program with an advanced degree that included formal training in statistics. To be exempted the student’s thesis advisory committee must review the students prior training and experience and approve the exemption. STAT 651, NRSC 671 and MSCI 611 are accepted options. Other courses may apply if recommended by the student’s thesis committee and approved by the TAMIN graduate program committee.

c) Four additional elective courses totaling at least 12 credit hours. The list of approved Neuroscience elective courses is on page 12. The purpose of this requirement is to ensure that students are broadly trained within their particular discipline. Courses selected to fulfill these requirements must appear on the degree plan.

d) NRSC 691 research credit hours. Once the core course requirements are met students should enroll in the appropriate number of NRSC 691 research credit hours under the section affiliated with their thesis advisor to achieve the required minimum number of credit hours per semester (9 cr for fall and spring, 6 for summer). If the thesis advisor does not have a section listed under NRSC 691, the advisor must contact the TAMIN advising office to create one.

4. REQUIRED TEACHING

There is no teaching requirement for any degree offered through TAMIN.
5. **CHOICE OF THESIS ADVISOR**

   All students must identify a Thesis Advisor by the end of their First year. The Thesis Advisor is the professor within whose laboratory the student will conduct their thesis research, and whom will provide guidance and oversight in choosing a thesis committee, developing a degree plan, ensuring the student maintains satisfactory academic progress throughout their academic tenure, and in the development and execution of a research project. The thesis advisor must be a member of the TAMIN Graduate Faculty. Students must inform the TAMIN Graduate Advisor of their choice of professor to serve as thesis advisor, and request that the professor notify the Graduate Advisor of their agreement. A sample memo is included in Appendix I. Since TAMIN is an interdisciplinary program, when students join a lab they will also become affiliated with their thesis advisor's home department. Faculty members are required to inform their home department's head and graduate advising office when they invite a TAMIN student to join their lab. Office space, building access (keys or door codes), phone service and Internet access must normally be obtained through the advisor's home department. The home department may have additional requirements or guidelines regarding participation in departmental functions, so be sure to discuss this with your thesis advisor.

6. **REQUIREMENTS FOR A CO-CHAIR COMMITTEE**

   Occasionally, a student can best complete his/her graduate program by working under the direction of two faculty members. Under these circumstances, a student may elect to be co-chaired by two Texas A&M faculty members. In general, students should request a co-chaired committee only if it is absolutely necessary for their graduate training. One of the co-chairs must be a member of the TAMIN graduate faculty. Both co-chairs should both provide ongoing intellectual contributions and be active mentors to the student. The co-chairs must be willing to act as a conduit to maintain lines of communication between the TAMIN graduate program, the advisory committee, and the student.

   One of the co-chairs must be a member of the TAMIN Graduate Faculty. The other co-chair may be a member of any department on campus. Students with co-chaired committees must satisfy all requirements for degrees and must take at least 50% of their 691 research credit hours as NRSC hours.

**Guidelines for Requesting a Co-Chaired Advisory Committee**

Requests for a co-chaired committee must be reviewed and approved by the TAMIN Graduate Program Committee (GPC) before the Graduate Advisor will approve a student's Degree Plan.

Requests for a co-chaired committee should be submitted to the Graduate Advising Office and must contain the following:

(1) **Student Statement of Purpose:**

   This letter, from the requesting student, should outline the reasons for requesting the co-chaired committee and the reasons for designating the specific faculty member as their choice of co-chair. The student should outline the role(s) each co-chair will take in guiding the students academic and research progress.

(2) **Letters from Faculty Co-Chairs:**
A letter is required from each co-chair outlining his or her contribution to the student’s academic endeavors and/or research projects and confirming their approval of the shared duties as co-chairs of the student’s advisory committee. The faculty co-chairs may be requested to meet with the GPC to discuss their contributions prior to approval of the request by the GPC.

7. ADVISORY COMMITTEE

An Advisory Committee supervises a student’s course work and research, examines a student’s progress, and approves all documents required for progress toward a degree. The Advisory Committee will approve the degree plan, read and critique the proposal and thesis or dissertation, and administer the oral exams. The Advisory Committee, chaired by the major professor, is a primary source of direction and intellectual support for a student’s research.

In order to provide the student with maximum input on course choices and research direction, the Advisory Committee should be constituted soon after the choice of major professor. Since the Advisory Committee plays an important role in helping the student choose their courses, and most of the student’s major coursework should be completed by the end of the second year, it is imperative that students meet with their Advisory Committee before the end of their first year.

The University requires that a graduate student’s Advisory Committee must include a total of at least three (for M.S. students) or four (for Ph.D. students) members of the TAMU graduate faculty. In addition to the University requirements, an NRSC graduate student’s Advisory Committee must include at least two (for M.S. students) or three (for Ph.D. students) tenured or tenure-track TAMIN graduate faculty. The University requires that one member of the Advisory Committee be from a department other than the student’s home department. The TAMIN graduate program accommodates this University requirement by specifying only that all of the committee members cannot be from the same department.

8. REQUIRED COMMITTEE MEETINGS

All graduate students are required to have at least one committee meeting each academic year. An Advisory Committee Meeting Report form must be submitted to the Graduate Advising Office no later than the end of summer term of each academic year. Failure to do so may result in a registration block for the Fall semester. The first committee meeting has a unique set of forms to be completed by the Advisory Committee, and subsequent meetings all use the Annual Advisory Committee Meeting Report. These can be obtained from the TAMIN Graduate Advising Office or downloaded from the TAMIN Graduate Program website; copies are included in the appendix of this manual.

9. FILING THE DEGREE PLAN

The Degree Plan lists the course work and research hours to be completed by a student during graduate study. The department or university cannot change the requirements for graduation once the Degree Plan is filed, and the student can only change the Degree Plan by filing a petition with OGAPS. The student, in consultation with the Major Professor and Advisory Committee, decides upon the courses included on the degree plan that are in addition to the
departmentally required courses. The list of approved courses starts on page 14; in addition, a certain number of seminars, lab rotations and other required courses are listed on pages 6-7. The minimum total number of hours required on a Ph.D. degree plan is 96 hours, however for students entering with a M.S. degree awarded in the U.S. (or its equivalent as determined by the Office of International Admissions) the minimum number of hours is 64. A minimum of 32 semester hours is required for the thesis M.S. degree and 36 semester hours for the non-thesis M.S. degree.

It is important that students review the limitations on the use of undergraduate courses, seminar hours, research hours, and transfer courses (detailed in the Graduate Catalog) prior to submitting a degree plan. Sample degree plans can be found in the appendix.

The degree plan must be filed electronically http://ogaps.tamu.edu/Buttons/Resources-for-Degree-Completion#0-SubmitDegreePlan. Instructions can be found at the Office of Graduate and Professional Studies website, http://ogaps.tamu.edu/.
GENERAL INFORMATION

Petitions

During the course of a student’s career it may be necessary to make requests for changes to the Office of Graduate and Professional Studies. These petitions (for changes of committee, program, courses, etc.) must be submitted to the TAMIN Graduate Advising Office on the appropriate OGAPS form (forms can be downloaded from the OGAPS web site) and with sufficient time to accommodate approval decisions. Please note that it can take OGAPS up to 3 months to process some requests.

Ombudsperson for Graduate Education

The Ombudsperson for Graduate Education assists graduate students, faculty, staff, and administrators to solve conflicts informally. The ombudsperson serves as a neutral listener, information resource, advisor, intermediary, and mediator. The ombudsperson advocates for the processes of graduate education by being equally open and accessible to all parties.

Ombudsperson contact information:
Ombudsperson for Graduate Education
1113 TAMU
College Station, TX  77842-1113
(979) 845-3631
ombuds@tamu.edu

Minimum Credit Hour Requirements

All students must remain in continuous enrollment throughout their graduate careers regardless of their source of support. Graduate students must enroll for at least one credit hour during every regular semester (Fall and Spring) while working towards their degrees. Enrollment for a minimum of one credit hour also is required in the Summer semester for all students using university facilities.

There are higher enrollment requirements for students receiving a graduate research or teaching assistantship. All graduate students receiving a graduate teaching or graduate research assistantship must register for a minimum of 9 semester credit hours during the Fall and Spring semester. In the Summer, students receiving a graduate assistantship must register for a minimum of 3 semester credit hours during the summer session in which they are employed or any combination of 6 semester credit hours during the entire Summer if they are employed for the entire summer. For example, 3 hours in SSI and 3 hours in SSII (total 6 hours) or 6 hours in the 10-week summer session.

Minimum GPR (Scholastic Deficiency)

A student’s Graduate GPR is expected to remain at or above 3.000 (on a 4.000 scale) during his or her graduate career. If a graduate student’s cumulative GPR falls below 3.000, he or she will be on scholastic probation and notified of this in writing by the Graduate Advisor. A copy of the memo will be sent to the student’s thesis advisor. The student will meet with his or her thesis advisor and advisory committee to develop a plan to overcome the scholastic deficiency. The plan should include the course(s) to be taken and the grade(s) the student must receive to return to good standing with the department. A copy of the plan signed by the student and the advisory committee
will be given to the Graduate Advising Office for the student’s file. If the student has not yet chosen a major professor, he or she will meet with the Graduate Advisor to develop such a plan, a copy of which will be put in the student’s file. The student will be given one semester (excluding summer terms) to raise his or her GPR above 3.000. If after one semester the student remains scholastically deficient, he or she will be informed of this in writing by the Graduate Advisor. The student may request the Graduate Program Committee for a second semester of academic probation. If the request is denied or if after two full semesters the student remains on scholastic probation, he or she will no longer be considered to be in good standing and may be asked to leave the graduate program: at the discretion of the GPC, the Graduate Advisor will submit a request to the Office of Graduate and Professional Studies that the student be dismissed from the University for scholastic deficiency.

Financial Support

Graduate students in the TAMIN graduate program are eligible to be supported by graduate teaching assistantships (GAT), graduate non-teaching assistantships (GANT), graduate research assistantships (GAR), or fellowships. GAR support is usually provided by individual faculty and is funded by research grants. Fellowship support may be provided by the University, Federal grants, or other sources and is awarded on a competitive basis.

In order to be eligible for support, students must be registered as full-time graduate students. In the Fall and Spring semesters, a minimum of 9 credit hours is required. For summer support, required registration is a minimum of 6 credit hours for the 10-week session or 3 credit hours per five-week summer session.

A&M Policy on the on maximum Doctoral (G8) Hours

A full-time doctoral student will be allowed to pursue his/her program for seven calendar years before a charge of out-of-state tuition is initiated. If a student is pursuing a doctoral degree on a part-time basis, he/she would have up to 99 semester hours before the university would begin to charge out-of-state tuition if they pass the seven year mark. Students who exceed these time limits will be charged out-of-state tuition to compensate for this lack of state support.

Graduate Students at affiliated TAMU campuses

Students undertaking research at affiliated campuses toward a Neuroscience Degree are required to adhere to all requirements, deadlines, etc. of the TAMIN Interdisciplinary Graduate Program. Residence on affiliated US campuses will satisfy the residency requirement for graduate students.

Participation in Programmatic Committees

Graduate students are encouraged to participate in program Committees. Regular elections are held to select graduate student representatives to the Graduate Programs, Graduate Recruiting and Admissions, and Outreach and Executive committees. These elections are held under the auspices of the Neuroscience Graduate Student Association (Students for Advancing Neuroscience Discovery and Innovation - SANDI). Students are encouraged to join and become active in SANDI, as it provides an organized means of communicating student concerns to the faculty and administration. SANDI officer elections are held at the beginning of the Fall semester.
Travel and Support

The Texas A&M Institute for Neuroscience Travel Award is a scholarship supported by the Texas A&M Institute for Neuroscience with funds provided by Texas A&M University. The purpose of the program is to support graduate student travel to make presentations by reimbursing students for some of the eligible expenses incurred. **Funds for these programs are provided at the budgetary discretion of TAMU and may vary annually, and therefore may not be available every year or for all students that apply.** TAIN will advise students of any unanticipated changes in the availability of travel funds so they may plan accordingly. All graduate students enrolled or affiliated with TAIN may request funds to travel to scientific meetings to make presentations. To be eligible for these awards students in their 2nd through 5th years must have all of their paperwork up to date and be identified as first/presenting author on the meeting abstract. Travel grants are limited to a maximum of $500 per trip.

Eligibility

1. The applicant must be in good academic standing (3.0 GPR) and registered as a full-time graduate student at TAMU - College Station campus at the time of application and at the time of receiving reimbursement.
2. Proposed presentations should relate directly to the student's degree program.
3. Students must be the presenting author of a poster or oral presentation.
4. Student's PI and research must be accomplished under the supervision of a member of the Faculty of Neuroscience who is in good standing.
5. Applicant must have completed one year of graduate study.
6. Student is allowed one TAIN Travel Award per Fiscal Year.
7. All application materials must be submitted prior to traveling (abstract).
8. Award recipients are expected to acknowledge the Texas A&M Institute for Neuroscience in their presentation.

Guidelines and Requirements

1. The applicant should clearly type (<10 point type will not be accepted) on the application form **(TAIN Travel Award form)** the nature and specific objectives of the proposed activities, with emphasis on how the requested funds will be used. Attach a copy of the conference program, invitation, or acceptance of abstract if presenting a paper and/or poster. Abstract acceptance is not mandatory at time of application if it has not been received by the time of your application, but it must be submitted before traveling.
2. The budget should be brief, but must list the specific items (including breakdown of proposed expenses for travel, lodging, and expenses, etc.) for which support is requested, giving evidence that the requested amount is realistic and the result of thoughtful planning. Give a clear justification if international travel is required. The applicant should not simply list a convenient figure or an overestimate; such budgets do not enhance the chances for favorable consideration and can cause an application to be disqualified. The maximum awards given for the various categories will be:
Presentation Award (travel within U.S) $400
( international travel) $500
TAMIN Students Based on Award Letter

Requesting Permission for Business Travel

Regardless of source of funding, or even if you pay your own costs, every graduate student making a professional trip to attend a meeting or conduct research must complete TAMU’s "Request for Business Travel" form per SAP 21.01.01.M0.02. These forms are essential to ensure that you will be appropriately covered by university insurance and your trip will be designated as professional business. The form can be obtained online (http://travel.tamu.edu/Forms) through the TAMIN Advising office or in your home departments business office.
GRADUATE DEGREE REQUIREMENTS
Interdisciplinary Program in Neuroscience
DOCTOR OF PHILOSOPHY

To earn a Doctor of Philosophy degree a student must meet the requirements of both the University and TAMIN. The TAMIN Graduate Program requirements are outlined below, along with a summary of the University requirements. Please refer to the Graduate Catalog for a complete description of University requirements and policies.

Please note that graduate students must fulfill the requirements of the catalog that is current during the semester they complete their degree requirements. This is the case for both University and TAMIN requirements. It is the student’s responsibility to keep up with changes in requirements.

REQUIREMENTS

A. Residence
Students who enter the doctoral degree program with a bachelor’s degree must spend two academic years in resident study at College Station or affiliated campus (Galveston, Temple, etc). If a Master’s degree has been awarded, one academic year is required. One academic year may include two adjacent regular semesters or one regular semester and one adjacent 10-week summer semester. See the Graduate Catalog for additional information on residence requirements.

B. Identify a Thesis Advisor
All NRSC graduate students must identify a professor by the end of their 2nd semester (excluding summer terms). Sponsorship by the Chair or Co-chair must be submitted in writing to the Graduate Advisor by the end of the 2nd semester.

The committee chair or one of the co-chairs must be a member of the TAMIN graduate faculty. Requests for a co-chair from outside of TAMIN must be approved by the Graduate Program Committee (see requirements on page 7).

C. Establish an Advisory Committee
The advisory committee, chaired by the thesis advisor, is a primary source of direction and intellectual support for a student’s research. The advisory committee should be constituted soon after the choice of major professor in order to provide the student with maximum input on course choices and research direction. The advisory committee will approve the degree plan, read, critique, and approve the proposal and dissertation, and administer the preliminary exam and the final defense.

The University requires that a doctoral student’s advisory committee be composed of no fewer than 4 members of the graduate faculty who are representative of the student’s field of study and research. The chair or one of the co-chairs of the advisory committee must be from the student’s major department, and at least one of the members must be from a department other than the student’s home department.
D. Degree Plan

The degree plan should be developed in consultation with the student’s advisory committee and submitted to the TAMIN Graduate Advising Office prior to registering for the 3rd semester (excluding summer terms). This deadline was established to ensure that students consult with their advisory committees about course work before beginning the second year of study.

For Ph.D. students, a minimum of 96 credit hours beyond the baccalaureate degree or 64 credit hours beyond the Master’s degree is required. Some Master’s degrees awarded in countries other than the U.S. are not equivalent to a Master’s degree awarded in the U.S. In these instances, the student will be required to have 96 hours on their degree plan.

The degree plan should include the course work required by TAMIN. These requirements are described in the following section. For limitations regarding the use of certain graduate courses and transfer credit see the TAMU Graduate Catalog. All doctoral degree plans must carry a reasonable amount of 691 (Research) hours.

The TAMIN Graduate Program Guide for the student’s particular degree must be submitted along with the degree plan (see section E, item 5).

E. Neuroscience Course Requirements

1) All Ph.D. students are required to complete the two core courses, Principles of Neuroscience 1 (fall) and 2 (spring) (NRSC 601 and 602), receiving a letter grade of at least a B. Each course is 3 credit hours.

2) All Ph.D. students are required to take a minimum of 4 approved elective courses. Each elective course is 3 or 4 credit hours. The graduate program committee considers exemptions of one or more electives if the student’s thesis committee considers the exemption justified based on previous coursework (for example if a student enters the program with an MS in a related field).

3) All first-year Ph.D. students are required to take NRSC 685 Neuroscience Rotations during the fall semester. Two half-semester rotations in the fall are required, but additional rotations in the spring semester are allowable as needed. Students receive 1 credit hour per rotation: students should enroll in 685 for a total of 2 credit hours in the fall.

4) All Ph.D. students are required to complete an approved course in Statistics and Experimental Design. Most options are 3 credit hour courses.

5) All Ph.D. students are required to complete an approved course in the Responsible Conduct of Research. 1 credit hour.

6) All Ph.D. students are required to be continuously enrolled in NRSC 681 Neuroscience Seminar. 1 credit hour per semester.

7) All student fill out the rest of their degree plan with research credit hours (NRSC 691). Students in their 3rd through 5th year typically take 6-8 research credit hours per semester, plus 1 credit hour each for a journal club and the weekly neuroscience seminar series. Students with co-chairs from outside of TAMIN must satisfy all TAMIN course requirements and must take at least 50% of their 691 research credit hours as NRSC hours.

F. Teaching requirement

There are no teaching requirements for the NRSC degrees.

G. Foreign Language

No foreign language is required.
H. Research Proposal

The Ph.D. student must prepare a research proposal for approval by his or her Advisory Committee. The Proposal describes the research that a student intends to undertake. The proposal is not a contract to perform the described research and significant research progress need not be completed at the time of proposal submission. The proposal is a mechanism to assist students in clarifying research goals early in their graduate program, to encourage students to become familiar with the primary literature in their field, to provide experience in scientific writing, and to facilitate research interactions between students and members of their Advisory Committee. In the proposal, the student describes the rationale for the research project, the objectives of the research to be performed, and outlines the methodologies to be used.

Students will prepare a proposal describing their planned research. The proposal format will be determined by the student's advisory committee during their first committee meeting and may vary depending on the student’s home department. Suggested formats include:

- NIH R01 applications (http://grants.nih.gov/grants/funding/phs398/phs398.html)
- NSF research proposals (http://www.nsf.gov/pubs/gpg/nsf04_23)
- NIH postdoctoral fellowships (http://grants1.nih.gov/grants/funding/416/phs416.htm)

A draft of the research proposal should be submitted to the student’s advisor by the end of the 4th semester. The proposal must be approved by the student’s advisor, then submitted to the entire advisory committee by the 3rd Monday in September of their 5th semester (excluding Summer). The advisory committee will evaluate the proposal and request any changes by the last business day in September. Students will complete any changes and gain approval by the committee to proceed with the preliminary exam by the 2nd Monday in October.

After revisions and approval by the advisory committee, the proposal should be submitted along with the signed official cover sheet to the TAIMN Graduate Advising Office. The official cover page is available on the OGAPS website http://ogaps.tamu.edu/Buttons/Forms-Information.

Students performing research involving human subjects, infectious biohazards, and/or recombinant DNA must attach a copy of the appropriate research compliance approval form to the proposal when proposal is submitted. Proposals that include research with vertebrate animals (including antibody generation in rabbits or mice) must include a copy of an approved Animal Use Protocol cover page. Information on Animal Use Protocols can be found at http://animal.tamu.edu/approval.html.

I. Preliminary Examination

The purpose of the preliminary examination is for the student’s advisory committee to determine whether the student has a mastery of the subject matter of all fields in the program, an adequate knowledge of the literature in these fields, and the ability to carry out bibliographical research. The preliminary examination is required. See the TAMU graduate catalog for additional details about University requirements at http://catalog.tamu.edu/graduate/academic-expectations-general-degree-requirements/degree-requirements/#Prelim.

Eligibility requirements for the preliminary exam.

- The student must be registered for at least 1 hour for the semester or 5-week summer term during which any portion of the preliminary exam may fall. If the entire exam falls...
between semesters, the student must be registered for the term immediately preceding the exam.

• An approved degree plan was on file with OGAPS at least 90 days prior to the first written examination.
• The student’s official GPR at the time of the examination must be at least 3.000.
• All English language proficiency requirements must have been satisfied.
• All committee members must have scheduled or waived the written portion and agree to attend the oral portion of the exam or have found a substitute. Only one substitution is allowed and it cannot be for the committee chair.
• At the end of the semester in which the exam is given, there are no more than 6 hours of formal course work remaining on the degree plan (except 681, 684, 690, 691, and 692). The TAMIN chair or head of the student’s department has the authority to approve a waiver of this criterion.
• The time span from the first written examination to the oral is no more than three weeks. (In cases of department-wide written examinations, this criterion is not applicable.) The head of the student’s department has the authority to approve a waiver of this criterion.

The preliminary examination includes both a written and an oral examination in which the student’s Advisory Committee tests a Ph.D. student’s mastery of his or her field of specialization. The preliminary examination will be administered, during the 5th semester (excluding Summer), by the student's advisory committee. University guidelines state that the Ph.D. preliminary examination consist of a written and an oral examination “unless otherwise recommended by the student's advisory committee and approved by the Office of Graduate and Professional Studies,”. The format and nature of the exam is at the discretion of the student’s advisory committee and may vary depending upon the student’s home department. Each member of the advisory committee is responsible for administering a written examination in his or her particular field, unless he or she chooses to waive participation in this part of the examination. Two or more members of the advisory committee may give a joint written examination. During this exam, students are expected to demonstrate that they: 1) have mastered fundamental concepts; 2) have gained detailed knowledge of scientific literature in their research area and the ability to critically evaluate it; 3) are able to formulate specific, plausible and testable hypotheses; 4) are able to design controlled experiments that distinguish among competing hypotheses; 5) can communicate effectively both in writing and in the oral presentation. Details of the exam format and requirements are as follows.

Preliminary examinations cannot be taken until all the core course requirements of the NRSC IDP have been completed and less than six hours of formal course work remain to be completed on the degree plan.

1) The student and committee chair will complete the Preliminary Exam Checklist. The committee chair will bring the Preliminary Exam Checklist to the TAMIN Graduate Advising Office, which will then submit the form to the Office of Graduate and Professional Studies. This MUST be submitted and the Exam scheduled 2 weeks prior to taking the Preliminary Exam.
2) Written exams will normally be taken during the week starting with the last Monday in October. Each student will arrange a time to take the written exam from each advisory committee member. Exams will be evaluated and returned to the committee chair, who will then forward the exams to the student. Students will have the opportunity to discuss any deficiencies in their exams with advisory committee members during the first full week of November. The nature and content of the written exams is at the full discretion of the Advisory Committee, and may include but are not limited to formal exam questions, literature reviews or major area papers. In circumstances where a major area paper (MAP) is required, this document does not replace or nullify the requirement for a separate and dedicated thesis proposal.

3) Oral exams will be taken during the second full week of November. Students are responsible for scheduling a mutually agreeable two-hour block of time for the committee to give the oral exam. The nature and content of the oral exams is at the full discretion of the Advisory Committee. In general, students are expected to prepare a 20-40 minute presentation on their proposal and will be examined on their proposal as well as their general knowledge of neuroscience. The committee will meet at the end of the exam and evaluate student performance. The student passes the preliminary exam if there is no more than one dissenting vote among advisory committee members. The committee will also discuss and complete the Annual Committee Meeting Evaluation form, which must be submitted to the TAMIN office.

4) In the event of a failure, the advisory committee has the option to allow a retake of the preliminary exam. The written and oral portions of the exam, administered as described above, must be completed within a three-week timeframe prior to Spring break. In the event of a second failure, no further retakes will be allowed. The student’s status in the NRSC graduate program will then be determined by the student and the advisory committee.

The results of the examinations should be reported on the Report of the Preliminary Exam form. The chair will bring the completed Report form to the TAMIN Graduate Advising Office, which will submit the form to the Office of Graduate and Professional Studies. Failure to submit the form to OGAPS within 10 working days of the exam will result in the preliminary exam being recorded as a failure. Copies of the official forms can be downloaded from the Office of Graduate and Professional Studies web site: [http://ogaps.tamu.edu/Buttons/Forms-Information](http://ogaps.tamu.edu/Buttons/Forms-Information).

After passing the preliminary examination, all degree requirements must be completed within four calendar years. Otherwise, the student will be required to repeat the preliminary exam.

**J. Admission to Candidacy**

For admission to candidacy for a doctoral degree, the student must have: (1) completed all formal course work on the degree plan with the exception of any remaining 681, 684, 690, and 691, (2) a 3.0 graduate GPR and a Degree Plan GPR of at least 3.0 with no grade lower than C in any course on the degree plan, (3) passed the written and oral portions of the preliminary exam, (4) submitted an approved dissertation proposal, and (5) met the residence requirements. The final examination will not be authorized for any doctoral student who has not been admitted to candidacy.
K. Continuous Registration

Once all coursework on the degree plan other than 691 (Research) is completed, a doctoral student must be in continuous registration until all further requirements for the degree have been completed. See the Graduate Catalog for additional information on the continuous registration requirement.

L. Pre-Defense Publication of Dissertation Material. Students should be aware of the agreement that is signed when a journal (hard copy or electronic) accepts an article for publication. At that time, the student assigns rights to the journal as publisher. The student must obtain written permission from the copyright holder to include the material in the thesis, dissertation, or record of study. Some journals and publishers have previously granted TAMU such rights, these can be found on the thesis office website.

M. Dissertation

The ability to perform independent research must be demonstrated by the dissertation, which must be the original work of the candidate. The dissertation describes the research performed by a student during graduate study and the unique contribution the student has made to advance the frontiers of knowledge. The student, in consultation with his or her Advisory Committee, determines the content of the dissertation. The dissertation must be approved by the student’s Advisory Committee. The dissertation should be submitted to the members of a student’s Advisory Committee at least two weeks prior to the Final Examination.

The dissertation must be original work, grammatically correct in a format consistent with that used in scholarly journals in the candidate’s field. The Office of Graduate and Professional Studies controls the format of the dissertation. Students must follow it exactly, or risk having it rejected by the Thesis Clerk. Instructions and the Thesis Manual is available on-line at http://thesis.tamu.edu/.

The student must submit an original copy of the dissertation in a form approved by the student’s Advisory Committee to the Graduate Advising Office in order to obtain the Program Chair’s approval and signature a minimum of two weeks prior to the Office of Graduate Studies deadline for submitting the dissertation to the Thesis Office. If the program chair deems the dissertation unsatisfactory, it will be given to the Graduate Program Committee for review. The Graduate Program Committee will make a recommendation of action to the program chair, student, and the members of the student’s Advisory Committee.

Students are required to submit an electronic thesis/dissertation (ETD) as a pdf file to the Thesis Office instead of using the traditional blue-line paper. Paper copies of these ETDs will not be sent to the library or to the departments. All electronically submitted manuscripts can be accessed from the Internet via http://etd.tamu.edu or through the library website, http://library.tamu.edu. Information on how to submit an electronic thesis/dissertation is available on the Thesis Office website: http://thesis.tamu.edu.

In addition, Tamin requires students to submit a copy of their dissertation printed on acid-free cotton bond paper to the Graduate Advising Office. Acid-free cotton bond paper can be obtained from the TAMIN Graduate Advising Office. TAMIN will have this document bound for the departmental archives.

Deadlines for submission of manuscripts to the Office of Graduate and Professional Studies are published each semester in the Office of Graduate and Professional Studies calendar. A copy of this calendar can be found at: http://ogaps.tamu.edu.Buttons/Calendars.
N. Time Limit

All graduate work must be completed within 10 consecutive calendar years. If within this time period a student does not complete all requirements for the degree sought, he or she cannot receive graduate credit for any course work that is more than 10 calendar years old at the time of the final examination.

O. Application for Degree

Graduate students who expect to complete their work at the end of a given semester must apply for graduation by submitting the electronic application for degree to the Office of the Registrar and by paying the required graduation fee at the Fiscal Department no later than the Friday of the second week of the fall or spring semester or the Friday of the first week of the first summer term. The electronic application for degree can be accessed via the website degreeapp.tamu.edu. Graduate students in interdisciplinary programs should attend the ceremony of their home academic department.

The TAMIN Graduate Advising Office should be notified when you apply to graduate so your file can be reviewed with time to identify and address any problems.

P. Final Examination/Dissertation Defense

In order to graduate at the end of a given semester the final exam for a doctoral degree must be passed by deadlines announced in the Office of Graduate and Professional Studies calendar. Students must be registered for at least one hour for the semester during the semester or summer term in which the final examination is held.

To be eligible to take the final examination, a student must be advanced to candidacy. The preliminary examination results and research proposal must have been submitted to the Office of Graduate and Professional Studies at least 14 weeks prior to the date of the defense. However the Final Examination must be held within three years of advancement to candidacy.

Request for permission to hold and announce the final oral examination must be submitted to the Office of Graduate and Professional Studies at least 10 working days before the requested exam date. This request must be approved by the student’s advisory committee, the TAMIN Graduate Advisor (or Department Head), and OGAPS. This announcement must be made on the official form, which can be downloaded from the OGAPS website. A sample form can be seen in Appendix II.

The student’s advisory committee will conduct the final examination/dissertation defense. The final examination is not to be administered until the candidate’s dissertation in substantially final form is provided to the Advisory Committee, and all concerned have had adequate time to review the document. TAMIN requires that the dissertation in substantially final form be submitted to the members of a student’s Advisory Committee at least two weeks prior to the Final Examination. In order to allow sufficient time for revisions and for Department Head approval, the Final Exam should be scheduled no later than 4 weeks prior to the OGAPS deadline for submission of the Dissertation.

All Ph.D. students receiving degrees through TAMIN’s NRSC Graduate Program will be required to present a seminar covering their dissertation research within the last year preceding their graduation. The student’s thesis committee may at their discretion require a private presentation and examination as part of the thesis defense, which satisfies TAMIN requirements. Ideally the student’s
thesis presentation should be a public seminar, which must be announced two weeks prior to the scheduled date and time (indicating that the student is a doctoral candidate), and be advertised by TAMIN and be open to all interested parties. Presentation of this seminar is to be followed by an open question period. Following the open question period. This public presentation may be coincident with but does not replace nor should it conflict with the student’s thesis defense, which will be conducted at the discretion of the student’s Advisory Committee. The Advisory Committee will conduct the Final Examination.

Whereas the final examination may cover the broad field of the candidate's training, it is presumed that the major portion of the time will be devoted to the dissertation and closely allied topics. Persons other than members of the graduate faculty may, with mutual consent of the candidate and the major professor, be invited to attend a final examination for an advanced degree. Upon completion of the questioning of the candidate, all visitors must excuse themselves from the proceedings when the Advisory Committee begins its deliberation on the results of the examination.

A positive vote by all members of the graduate committee with at most one dissension is required to pass a student on his or her exam.
Timeline Summary for Graduate Studies
Doctor of Philosophy
Interdisciplinary Program in Neuroscience

Year 1:
- Complete required courses, and seminars (NRSC 601, 602, 681)
- Complete rotations (NRSC 685)
- Choose advisor.
- Develop tentative degree plan with advisor
- Set up advisory committee
- Hold first committee meeting
  - Outline research project
  - Discuss degree plan
  - Obtain committee approval for degree plan
  - Submit Annual Committee Meeting progress report to TAMIN office
- Submit degree plan to the Office of Graduate and Professional Studies (OGAPS)

Year 2:
- Attend weekly seminars and attend symposia
- Complete remaining coursework (electives, ethics, statistics requirements)
- Research credit hours (NRSC 691)
- Submit draft of research proposal to thesis advisor by end of Spring semester
- Advisory committee meeting (by end of summer)
  - Submit Annual Committee Meeting progress report to TAMIN office

Year 3:
- Attend weekly seminars and attend symposia
- Research credit hours (NRSC 691)
- Complete preliminary exam*:
  - Submit research proposal to advisory committee (deadline: 3rd Monday in September)
  - Submit Preliminary Exam Checklist two weeks before preliminary exam commences (deadline: 3rd Monday in October)
  - Complete written and oral exams (deadline: Last working day of November)
- Advisory committee meeting (by end of summer)
  - Submit Annual Committee Meeting progress report to TAMIN office

Year 4 and beyond:
- Attend weekly seminars and attend symposia
- Hold committee meeting each year and submit progress report to TAMIN office.

Dissertation final exam:
- Permission to defend dissertation
  - Obtain permission to defend from advisory committee
Submit completed Permission to Defend Thesis form to the OGAPS two weeks before defense

Dissertation defense

- Distribute written dissertation to advisory committee at least two weeks before scheduled oral defense
- Present and defend dissertation to advisory committee
- Obtain committee approval for dissertation
- Submit dissertation to the OGAPS

*Some details regarding the nature and timeline of the preliminary exams may vary depending on the students home department.
GRADUATE DEGREE REQUIREMENTS
Interdisciplinary Program in Neuroscience
MASTER OF SCIENCE
Thesis Option

TAMIN does not recruit or accept applications from students into the NRSC graduate program solely interested in pursuing a Master’s degree in Neuroscience. However, current student in the midst of pursuing a PhD may choose to exit the program with a MS degree. To earn a Master of Science (thesis option) degree a student must meet the requirements of both the University and TAMIN. The TAMIN requirements are outlined below, along with a summary of the University requirements. Please refer to the TAMU Graduate Catalog for a complete description of University requirements and policies.

Please note that graduate students must fulfill the requirements of the catalog that is current during the semester they complete their degree requirements. This is the case for both University and TAMIN requirements. It is the student’s responsibility to keep up with changes in requirements.

<table>
<thead>
<tr>
<th>TAMIN DEADLINES</th>
<th>M.S. Degree</th>
</tr>
</thead>
<tbody>
<tr>
<td>Choice of Thesis Advisor</td>
<td>By the end of the 2nd Semester</td>
</tr>
<tr>
<td>Degree Plan Filed with Department</td>
<td>Before registering for first semester of 2nd Year</td>
</tr>
<tr>
<td>Proposal Filed with Department</td>
<td>During the 3rd Semester (see page 28)</td>
</tr>
<tr>
<td>Final Examination</td>
<td>Conclusion of Research / Thesis written</td>
</tr>
</tbody>
</table>

Note that the number of semesters does NOT include summer sessions.

REQUIREMENTS

A. Residence
In partial fulfillment of the residence requirement for the degree of Master of Science, the student must complete 9 residence credit hours during one regular semester or one 10-week summer semester. Upon recommendation of the student’s advisory committee and with approval of the Office of Graduate and Professional Studies, a student may be granted exemption from this requirement. However, such a petition must be approved prior to the student’s registration for the final 9 credit hours of required course work.

B. Identify a Thesis Advisor
All NRSC graduate students are required to identify a major professor by the end of the second full semester (excluding summer terms). M.S. candidates are encouraged to identify a major professor by the end of the first full semester. Sponsorship by the Chair or Co-chair must be submitted in writing to the Graduate Advisor by the end of the second semester.

The committee chair or one of the co-chairs must be a member of TAMIN graduate faculty. Requests for a co-chair from outside TAMIN must be approved by the TAMIN Graduate Program Committee (see requirements on page 7).
C. Establish an Advisory Committee

The advisory committee, chaired by the major professor, is a primary source of direction and intellectual support for a student’s research. The advisory committee should be constituted soon after the choice of major professor in order to provide the student with maximum input on course choices and research direction. The advisory committee will approve the degree plan, read and critique the proposal and thesis, and administer the final exam.

The University requires that a M.S. student’s advisory committee be composed of no fewer than 3 members of the graduate faculty who are representative of the student’s field of study and research. The chair or one of the co-chairs of the advisory committee must be from the student’s major department, and at least one of the members must be from a department other than the student's department. The NRSC IDP has established the following additional requirements. The advisory committee must contain at least two tenured or tenure-track member of the TAMIN graduate faculty.

D. Degree Plan

The degree plan should be developed in consultation with the student’s advisory committee and submitted to the TAMIN Graduate Advisor prior to registering for the 3rd semester (excluding summer terms). This deadline was established to ensure that students consult with their advisory committees about course work before beginning the second year of study.

For M.S. Thesis students, the degree plan must contain a minimum 32 semester hours of approved courses and research hours. TAMIN course requirements are described in the following section. There are limitations regarding the use of certain graduate courses including 681, 685, 689, 690, 691, 694, and 695 courses, certain transfer course work, extension courses, advanced undergraduate courses, and certain courses offered by the College of Medicine. The student is referred to the Graduate Catalog for the details of these limitations.

The TAMIN Graduate Program Guide for the student’s particular degree must be submitted along with the degree plan (see section E, item 5).

E. Departmental Course Requirements

1) All M.S. students are required to register for NRSC 681 and attend the weekly seminar series.

2) All M.S. students must register for at least one credit in a Neuroscience-related journal club. This may be the journal club accompanying the Principles of Neuroscience 1 and 2 or a journal club of their choice in the home department.

3) Graduate students are required to take the two core courses, Principles of Neuroscience 1 and 2 plus a minimum of 4 elective courses. Exemptions will be considered by the GPC. These courses are listed on page 12 at the beginning of this section.

4) M.S. students with a co-chair from outside of TAMIN must take at least 50% of their 691 research credit hours as NRSC credit hours.

F. Teaching Requirement

No teaching is required.

G. Foreign Language

No foreign language is required.
H. Research Proposal

The M.S. student must prepare a research proposal for approval by his or her Advisory Committee. The Proposal describes the research that a student intends to undertake. The proposal is not a contract to perform the described research and significant research progress need not be completed at the time of proposal submission. The proposal is a mechanism to assist students in clarifying research goals early in their graduate program, to encourage students to become familiar with the primary literature in their field, to provide experience in scientific writing, and to facilitate research interactions between students and members of their Advisory Committee. In the proposal, the student describes the rationale for the research project, the objectives of the research to be performed, and outlines the methodologies to be used.

Students will prepare a proposal describing their planned research. The proposal format will be determined by the student’s advisory committee during their first committee meeting. Suggested formats include:

- NIH R01 applications (http://grants.nih.gov/grants/funding/phs398/phs398.html)
- NSF research proposals (http://www.nsf.gov/pubs/gpg/nsf04_23)
- NIH postdoctoral fellowships (http://grants1.nih.gov/grants/funding/416/phs416.htm)

The proposal must first be approved the student’s advisor, then submitted to the advisory committee by the end of their 3rd semester (excluding summer). After revisions and approval by the advisory committee, the proposal should be submitted along with the signed official cover sheet to the TMIN Graduate Advising Office. The official cover page is available on the OGAPS website. http://ogaps.tamu.edu/Buttons/Forms-Information.

Students performing research involving human subjects, infectious biohazards, and/or recombinant DNA must attach a copy of the appropriate research compliance approval form to the proposal when proposal is submitted. Proposals that include research with vertebrate animals (including antibody generation in rabbits or mice) must include a copy of an approved Animal Use Protocol cover page. Information on Animal Use Protocols can be found at http://animal.tamu.edu/approval.html.

I. Pre-Defense Publication of Thesis Material.

Students should be aware of the agreement that is signed when a journal (hard copy or electronic) accepts an article for publication. At that time, the student assigns rights to the journal as publisher. The student must obtain written permission from the copyright holder to include the material in the thesis, dissertation, or record of study. Some journals and publishers have previously granted TAMU such rights, these can be found on the thesis office website.

J. Thesis

The thesis describes the research performed by a student during graduate study and the unique contribution the student has made to advance the frontiers of knowledge. The student, in consultation with the Advisory Committee, determines the content of the thesis. The thesis must be approved by the student’s Advisory Committee. The thesis should be submitted to the members of a student’s Advisory Committee at least two weeks prior to the Final Examination.

The thesis must be original work, grammatically correct in a format consistent with that used in scholarly journals in the candidate’s field. The Office of Graduate and Professional Studies controls

After approval by the Advisory Committee an original of the thesis must be submitted to the TAMIN Graduate Advising Office in order to obtain the Department Head's approval and signature a minimum of two weeks prior to the Office Graduate Studies deadline for submitting the thesis to the Thesis Office. If the Department Head deems the thesis unsatisfactory, it will be given to the Graduate Program Committee for review. The Graduate Program Committee will make a recommendation of action to the Department Head, student, and the members of the student's Advisory Committee.

Students are required to submit an electronic thesis/dissertation (ETD) as a pdf file to the Thesis Office instead of using the traditional blue-line paper. Paper copies of these ETDs will not be sent to the library or to the departments. All electronically submitted manuscripts can be accessed from the Internet via http://etd.tamu.edu or through the library website, http://library.tamu.edu. Information on how to submit an electronic thesis/dissertation is available on the Thesis Office website: http://thesis.tamu.edu.

In addition, TAMIN requires students to submit a copy of their thesis printed on acid-free cotton bond paper to the Graduate Advising Office. TAMIN will have this document bound for the departmental archives. Acid-free cotton bond paper is available in the Graduate Advising Office.

Deadlines for submission of manuscripts to the Office of Graduate and Professional Studies are published each semester in the Office of Graduate and Professional Studies calendar. A copy of this calendar can be found at http://ogaps.tamu.edu/Buttons/Calendars.

K. Time Limit

All requirements must be completed within seven consecutive calendar years. If a student does not complete all requirements for the degree sought by seven years, no course work will be applicable to the degree program that is more than seven calendar years old at the time of the final examination.

L. Application for Degree

Formal application for the degree must be filed in the Office of Graduate and Professional Studies not later than 90 days prior to the end of the semester (or 30 days in the summer term). Students must be registered in the semester in which the degree is conferred. If graduating at the end of the summer semester, the student must register during the first 5-week term of the summer session. There is a diploma fee that must be paid at the time formal application is submitted. The TAMIN Graduate Advising Office should be notified when the application for degree is filed so that the student’s file can be reviewed with time to address any problems.
M. Final Examination for M.S. students

The student should read the Graduate Catalog for a complete description of the University requirements.

1. The student must pass their final exam by deadline dates announced in the Office of Graduate and Professional Studies Calendar.
2. The student must be registered in the semester that the exam is taken.
3. The student’s GPR must be at least 3.000 for courses on the degree plan and for all courses completed at Texas A&M that are eligible to be applied to a graduate degree. There must be no un-absolved grades of D, F, or U in courses listed for credit on the degree plan. See the Graduate Catalog for information on how to absolve a deficient grade.
4. The student must have completed all course work on the degree plan with the exception of those hours for which the student is registered.
5. All English Language Proficiency requirements must be satisfied before the final examination is scheduled.
6. An approved research proposal must be on file with the Office of Graduate and Professional Studies by the published deadlines.
7. A request for permission to hold and announce the final examination must be submitted to the Office of Graduate and Professional Studies at least 10 working days in advance of the scheduled date for final examination.
8. The final examination covers the thesis and all course work on the degree plan. At the discretion of the Advisory Committee, the final examination may be written, oral, or both.
9. The final examination may not be administered until such time that the thesis is available to all members of the advisory committee in substantially final form and all members have had adequate time to review the document.
10. The final examination must be administered on campus (unless approved by the OGAPS).
11. There will be only one opportunity to retake the final examination. This must be accomplished within a time period that does not extend beyond the next regular semester (summer terms excluded).
GRADUATE DEGREE REQUIREMENTS
Interdisciplinary Program in Neuroscience
MASTER OF SCIENCE
Non-Thesis Option

To earn a Master of Science (non-thesis option) degree a student must meet the requirements of both the University and TAMIN. TAMIN requirements are outlined below, along with a summary of the University requirements. Please refer to the Graduate Catalog for a complete description of University requirements and policies.

Please note that graduate students must fulfill the requirements of the catalog that is current during the semester they complete their degree requirements. This is the case for both University and TAMIN requirements. It is the student's responsibility to keep up with changes in requirements.

REQUIREMENTS

All requirements for the non-thesis option Master of Science degree other than those specified below are the same as those for the thesis option degree.

Required course work

A minimum of 36 semester hours is required. The degree plan must be approved by the student's advisory committee and department head and is subject to the Limitations on the Use of Transfer, Extension and Certain Other Courses as described in the Graduate Catalog.

Students pursuing a non-thesis M.S. degree are not allowed to enroll in 691 (Research) for any reason and no 691 hours may be used for credit on the degree plan. A maximum of 4 credit hours of 684 (Professional Internship), 8 credit hours of 685 (Directed Studies), and up to 3 credit hours of 690 (Theory of Research) or 695 (Frontiers in Research) may be used toward the non-thesis option M.S. degree. In addition, any combination of 684, 685, 690, and 695 may not exceed 25% of the total credit hour requirement shown on the student’s degree plan.

TAMIN course requirements for the non-thesis option Master of Science degree are same as those for the thesis option degree.

Thesis

A thesis is not required. However, TAMIN requires non-thesis option students to either prepare and submit a library research paper as described in the following section or have already successfully completed the written exam portion of their preliminary exams (i.e. if the student has previously completed their preliminary exams and been admitted to candidacy before deciding to leave the program with a non-thesis Master's degree, their thesis committee may elect to accept the results of the written exams as having satisfied the written requirements for the non-thesis MS degree).

Library Research Paper

Students pursuing the non-thesis option Master of Science Degree are required to prepare and submit a library research paper. The purpose of this paper is to demonstrate that the student can do library research and read, understand, and integrate information from the primary literature. In
The scope of the paper is similar to the literature review that constitutes the first chapter of a thesis or dissertation. Typically, the paper is expected to be approximately twenty pages of double-spaced type, not including references and figures or tables. If the student has previously completed a major area paper as part of their preliminary exams and been admitted to candidacy, the thesis committee can elect to accept this paper as meeting the requirement for a library research paper.

The student’s Advisory Committee must approve this effort. An approved copy of this paper will be deposited with the Chair of the student’s advisory committee and a second soft-bound copy will be deposited in the departmental file of non-thesis papers located in the TAMIN Graduate Advising Office.

**Final Exam**

A final comprehensive examination is required for students seeking a non-thesis M.S. degree in Neuroscience. No exemptions are allowed. The requirements as to level of courses and examinations are the same as for the thesis option M.S. degree. As stated above, the student’s thesis committee may elect to accept the results of the student’s written preliminary exams if the student had previously completed these and been admitted to Ph.D. candidacy before deciding to leave the program with an MS degree.
NRSC GRADUATE COURSE LIST

Graduate students are required to take two Principles of Neuroscience courses and a minimum of 4 elective courses coming from the approved list below. Exemptions and alternative courses not yet evaluated will be considered on a case by case basis by the Graduate Program Committee (GPC). NOTE: not all courses are offered every semester or every year, so students are encouraged to check with the instructor of record about future offerings.

<table>
<thead>
<tr>
<th>Title</th>
<th>NRSC Prefix</th>
<th>Course # Home</th>
</tr>
</thead>
<tbody>
<tr>
<td>Principles of Neuroscience I</td>
<td>NRSC 601</td>
<td>BIOL 627</td>
</tr>
<tr>
<td>Principles of Neuroscience II</td>
<td>NRSC 602</td>
<td>BIOL 628</td>
</tr>
<tr>
<td>Neuroanatomy</td>
<td>NRSC 603</td>
<td>VIBS 603</td>
</tr>
<tr>
<td>Biomed. Neuroendo. Endocrine Disorders</td>
<td>NRSC 604</td>
<td>VIBS 604</td>
</tr>
<tr>
<td>Neuroanatomical Systems</td>
<td>NRSC 605</td>
<td>VIBS 606</td>
</tr>
<tr>
<td>Learning</td>
<td>NRSC 606</td>
<td>PSYC 606</td>
</tr>
<tr>
<td>Physiological Psychology</td>
<td>NRSC 609</td>
<td>PSYC 609</td>
</tr>
<tr>
<td>Molecular Biol. of Differentiation &amp; Dev.</td>
<td>NRSC 611</td>
<td>BIOL 611</td>
</tr>
<tr>
<td>Perceptual Processes</td>
<td>NRSC 615</td>
<td>PSYC 615</td>
</tr>
<tr>
<td>Advanced Developmental Neurotoxicology</td>
<td>NRSC 616</td>
<td>VIBS 616</td>
</tr>
<tr>
<td>Functional Neuroanatomy</td>
<td>NRSC 621</td>
<td>VIBS 621</td>
</tr>
<tr>
<td>Comparative Neurobiology</td>
<td>NRSC 634</td>
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</tr>
<tr>
<td>Biological Clocks</td>
<td>NRSC 635</td>
<td>BIOL 601</td>
</tr>
<tr>
<td>Signaling in Behavior and Development</td>
<td>NRSC 636</td>
<td>BIOL 615</td>
</tr>
<tr>
<td>Neurobiology</td>
<td>NRSC 640</td>
<td>VIBS 640</td>
</tr>
<tr>
<td>Principles of Neuropsychology</td>
<td>NRSC 641</td>
<td>PSYC 641</td>
</tr>
<tr>
<td>Neural Development</td>
<td>NRSC 644</td>
<td>BIOL 644</td>
</tr>
<tr>
<td>Seminar in Behavioral Neuroscience</td>
<td>NRSC 649</td>
<td>PSYC 649</td>
</tr>
<tr>
<td>Seminar in Neurobiology Journal Club</td>
<td>NRSC 650</td>
<td>BIOL 681</td>
</tr>
</tbody>
</table>
Other approved electives available at this time:
MSCI 601 Principles of Medical Sciences * not yet crosslisted
NEXT 603 Advanced Neuropsychopharmacology
MSCI 610D Pathogenesis of Human Disease * not yet crosslisted
PSYC 689 Neurobiology of Learning and Memory
VIBS 617 Cell Signaling
BIOL 609 Tools in Molecular Biology.

Special Topics courses (689’s):
These courses would cover current topics of interest and may or may not become permanent courses. Typically, they would be 1-3 credit literature-based courses and would be announced at the beginning of each semester. If these courses are well subscribed for 3 consecutive academic years then they could be moved up to the major course category and given a NRSC course number.

Undergraduate background courses:
If a graduate student enters the program without the background needed for a graduate course in a particular area, the student’s advisory committee may deem it necessary and appropriate for them to first take an undergraduate course. However, undergraduate (400-level) courses do not count towards the required 96/64 credit hours for completion of the degree.
APPENDIX S

Roadmap to the Ph.D.
Timeline Summary for Graduate Studies
Doctor of Philosophy
Interdisciplinary Program in Neuroscience

Year 1:
➤ Complete required courses, and seminars (NRSC 601, 602, 681)
➤ Complete rotations (NRSC 685)
➤ Choose advisor.
➤ Develop tentative degree plan with advisor
➤ Set up advisory committee
➤ Hold first committee meeting
  • Outline research project
  • Discuss degree plan
  • Obtain committee approval for degree plan
  • Submit Annual Committee Meeting progress report to TAIN office
➤ Submit degree plan to the Office of Graduate and Professional Studies (OGAPS)

Year 2:
➤ Attend weekly seminars and attend symposia
➤ Complete remaining coursework (electives, ethics, statistics requirements)
➤ Research credit hours (NRSC 691)
➤ Submit draft of research proposal to thesis advisor by end of Spring semester
➤ Advisory committee meeting (by end of summer)
  • Submit Annual Committee Meeting progress report to TAIN office

Year 3:
➤ Attend weekly seminars and attend symposia
➤ Research credit hours (NRSC 691)
➤ Complete preliminary exam*:
  • Submit research proposal to advisory committee (deadline: 3rd Monday in September)
  • Submit Preliminary Exam Checklist two weeks before preliminary exam commences (deadline: 3rd Monday in October)
  • Complete written and oral exams (deadline: Last working day of November)
➤ Advisory committee meeting (by end of summer)
  • Submit Annual Committee Meeting progress report to TAIN office

Year 4 and beyond:
➤ Attend weekly seminars and attend symposia
➤ Hold committee meeting each year and submit progress report to TAIN office.

Dissertation final exam:
➤ Permission to defend dissertation
  • Obtain permission to defend from advisory committee
➢ Submit completed Permission to Defend Thesis form to the OGAPS two weeks before defense

➢ Dissertation defense
  • Distribute written dissertation to advisory committee at least two weeks before scheduled oral defense
  • Present and defend dissertation to advisory committee
  • Obtain committee approval for dissertation
  • Submit dissertation to the OGAPS

*Some details regarding the nature and timeline of the preliminary exams may vary depending on the students home department.
APPENDIX T

Faculty Biosketches
BIOGRAPHICAL SKETCH

NAME: ABBOTT, Louise C.

eRA COMMONS USER NAME: LABBOTT

POSITION TITLE: Presidential Professor for Teaching Excellence

EDUCATION/TRAINING

<table>
<thead>
<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
<th>Completion Date MM/YYYY</th>
<th>FIELD OF STUDY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Whitman College, Walla Walla, WA</td>
<td>B.A.</td>
<td>B.A.</td>
<td>Biology</td>
</tr>
<tr>
<td>University of Washington, Seattle, WA</td>
<td>Ph.D.</td>
<td>Ph.D.</td>
<td>Developmental Biology</td>
</tr>
<tr>
<td>Washington State University, Pullman, WA</td>
<td>D.V.M.</td>
<td>D.V.M.</td>
<td>Veterinary Medicine</td>
</tr>
</tbody>
</table>

A. Personal Statement

My research interests include developmental neurobiology of the mammalian nervous system; neuroanatomy; neurochemistry; specific neurologic disorders including ataxia and epilepsy; and developmental neurotoxicology with special interest in the mechanisms of toxicity of methylmercury on the developing and adult brain. I have extensive experience in using both bright field and fluorescence labeling of neurons to both identify neuronal cell types as well as to identify specific cell types using double labeling. I also have extensive experience in preparing samples of tissues to be sectioned using the Knife Edge Scanning Microscope (KESM) as well as validating data obtained from the KESM. Much of my research has focused on the analysis of mouse brain development as well as adult mouse and rat brain neuroanatomy.

B. POSITIONS and HONORS:

Positions and Employment

1982 -1983  Postdoctoral Research Associate, Dept. of Veterinary & Comparative Anatomy, Pharmacology, Coll. of Veterinary Medicine, Wash. State University, Pullman, WA
1988 -1994 Faculty member, Neuroscience Program, Univ. of Illinois at U-C, Urbana, IL
1994 -1999 Assistant Professor, Dept. of Veterinary Integrative Biosciences, College of Veterinary Medicine, Texas A&M University, College Station, TX
1995 - present Faculty member, Texas A&M Institute for Neuroscience and the Toxicology Program, Texas A&M University, College Station, TX
1999 - 2012 Associate Professor, Dept. of Veterinary Integrative Biosciences, College of Veterinary Medicine, Texas A&M University, College Station, TX
2005 - present Member, Graduate Faculty of the Texas A&M University Health Science Center, College Station, TX
2012 - present Professor, Dept. of Veterinary Integrative Biosciences, College of Veterinary Medicine, Texas A&M University, College Station, TX
2014 - present Presidential Professor for Teaching Excellence, Texas A&M University, College Station, TX
Other Experience

1994  Fulbright Research Scholarship (Paris, France)
2012  NIH Special Emphasis Panel Member, ZGM1 TWD-A CB, June, 2012

Editorial Boards

2009-2011  Toxicology Letters – Editorial Board
2010-present  Journal of Applied Toxicology – Advisory Board
2011-2012  Anatomia, Histologia, Embryologia – Editorial Board
2012-present  Anatomia, Histologia, Embryologia – Associate Editor
2013-2014  Poultry, Fisheries and Wildlife Sciences – Editorial Board
2014-present  Poultry, Fisheries and Wildlife Sciences – Editor-in-Chief

Professional Memberships

1990-present  American Association of Veterinary Anatomists
2001-present  Sigma Xi
2005-present  Neurotoxicity Society
2005-present  Cajal Club
2008-present  Society of Toxicology

Honors

1975  Phi Beta Kappa Honor Society; Graduated magna cum laude & Honors in major study (B.A.)
1977 - 1981  Developmental Biology Graduate Training Program Fellowship (Univ. of Washington)]
1985  Member, Phi Zeta Honor Society
1988  Seattle-King County Veterinary Medical Assoc. Scholarship; Graduated cum laude (D.V.M.)
1989  Arnold O. Beckman Research Award (Univ. of Illinois)
1990  SAVMA Veterinary Medical Teaching Excellence Award in Basic Sciences (Univ. of Illinois)
1993  College of Veterinary Medicine Teaching Award (Univ. of Illinois)
1997 - 1998  Montague Center for Teaching Excellence Scholar (Texas A&M University)
1999  Samuel F. Scheidy Memorial Award, Veterinary Medical Foundation (Research Excellence)
2008  Nominated as a top reviewer in for Neurotoxicology and Teratology
2009  The Association of Former Students of Texas A&M University Distinguished Achievement Award at the College Level for Teaching
2010  The Association of Former Students of Texas A&M University Distinguished Achievement Award at the University Level for Teaching
2010  The Pfizer Carl J. Norden Distinguished Achievement for Teaching in Veterinary Medicine
2010  Selected as a top reviewer for Neurotoxicology Letters
2012-2015  TAMU Thammon Professorship in Undergraduate Teaching Excellence
2012  College of Veterinary Medicine & Biomedical Sciences Outstanding Graduate Student Mentor Award
2013  American Association of Veterinary Anatomists, 2013 Outstanding Anatomist Award
2014  TAMU Presidential Professor for Teaching Excellence
2015  College of Veterinary Medicine & Biomedical Sciences John H. Milliff ’28 Memorial Teaching Award
2015  Piper Professor of 2015 (State of Texas Teaching Award)
C. CONTRIBUTION TO SCIENCE:

Selected Peer-reviewed Publications (Selected from 85 peer-reviewed publications)


In this study, we analyzed the noradrenergic innervation of the mouse cerebellum and observed that in normal mice the analyzed axon terminals established synaptic junctions with apposed neural elements. Thus we determined that cerebellar noradrenergic innervation is of the nonjunctional modality. Up to this point, scientists had questioned whether this was, in fact the case. In examination of the agranular weaver mouse cerebellum we determined that despite the presence of innumerable free postsynaptic differentiations, mainly Purkinje cells dendritic spines, cerebellar noradrenergic innervation does not change from nonjunctional to junctional innervation as is the case for the cerebellar serotonergic system, indicating significant differences in organization and responses to remodeling between noradrenergic neurons and serotonergic neurons.


Methylmercury (MeHg) has cytotoxic effects on both animals and humans and a major target organ is the central nervous system. The developing nervous system is more vulnerable to methylmercury toxicity than the mature nervous system. However most studies published at the time of our 2008 study had not examined effects of very low methylmercury concentrations on brain development and behavior. We examined effects of very low concentrations of methylmercury exposure during in utero development in mice, specifically looking at behavior in young adult mice exposed prenatally to methylmercury. We observed that prenatal exposure to the lowest concentrations of methylmercury used at that time had long-lasting adverse motor and cognitive consequences in adult offspring. These observations have far reaching implications of exposure of pregnant women to low concentrations of methylmercury.


It was predicted that not only does methylmercury have more severe effects on the developing nervous system, but methylmercury exposure also has more severe effects on the aged central nervous system than observed in young adults. When we compared our results from methylmercury exposure in young adult mice (2007 manuscripts) to our 2012 study we reported, that the aged mouse nervous system was actually not more sensitive to methylmercury exposure than the young adult nervous system. This then suggests that chronic insult such as neurodegenerative diseases might in fact make the aged nervous system more vulnerable and not the aging process alone.


It has been demonstrated that methylmercury has severe effects on the developing nervous system, but methylmercury exposure. Our more recent studies have expanded the use of zebrafish embryos as a neurotoxicology model to study mechanisms by which exposure to low concentrations of mercury affect development.


Nicotine, which is the major psychoactive component in tobacco products interacts with nicotinic acetylcholine receptors (nAChR) in the nervous system. Nicotine has both neurotoxic and neuroprotective functions in the developing as well as aging brain. We carried out a series of studies examining the effects of nicotine exposure in the developing and adult mouse brain. We observed that the hippocampus is more sensitive to nicotine exposure than the cerebellum in the developing brain, which could explain adverse cognitive functions resulting from gestational nicotine exposure but not motor deficits. With respect to the reported neuroprotective functions of nicotine, especially in the adult brain, action on so called “longevity genes” was postulated to be one possible cause. Our 2011 study showed that nAChRs do not directly regulation the expression of major longevity genes in the aging mouse. However, we do postulate that loss of beta2 nAChRs could result in increased cellular stress, which could have an indirect effect on expression of several longevity genes, resulting in an adaptive response against neurodegeneration. These studies culminated in publication of our 2012 review paper.


The long-term research focus of the Brain Networks Laboratory is to better understand relationships between form and function in the mammalian brain. Our research group has been developing methodologies to allow detailed mapping of neural and vascular connections in the mammalian brain for over 10 years. We use the mouse brain as our model as it is a complex mammalian organ yet it is small enough to be sampled efficiently. An additional goal for our research group is to make data sets available to the larger neuroscience research community. The Brain Networks Laboratory, has developed the knife-edge scanning microscope (KESM), which is now becoming available for use by other research groups. The KESM provides high-throughput, high-resolution, high volume neuroanatomical data that are necessary to better understand the brain connectome. My role in this endeavor has been two-fold: 1) I developed methodologies to provide whole mouse brains stained with Nissl or Golgi-Cox stains and to use India ink to fill the entire central nervous system blood vascular system. These samples are embedded in plastic, sectioned on the KESM and the resulting data sets are analyzed. My second role is to provide neuroanatomical expertise when analyzing the resulting data sets.

D. RESEARCH SUPPORT

Ongoing Research Support

NIH/NLM
Large-scale reconstruction of microvascular networks
Sub-award to University of Houston (PI David Mayerich) 10/01/13 to 8/31/17
Louise Abbott – role – subcontract
This project is examining microvascular networks in the mouse brain and other organs as well as tumors.
Completed Research Support

IDBR-1215422 NSF
Enhanced Knife-Edge Scanning Microscopy for Sub-micrometer Imaging of Whole Small Animal Organs 04/01/13 to 03/31/16

**Co-PIs** - Yoonsuck Choe, John Keyser, Louise Abbott
This project continued to develop use of the knife-edge scanning microscope for imaging the three dimensional cellular and vascular structure of various organs using the mouse as an animal model.

NSF
Three dimensional analysis of mMRI
Sub-award to Kettering University on NSF MRI0 9/01/13 – 07/31/15

**Louise Abbott** – role – consultant
This project helped to elucidate three dimensional anatomy of the mammalian brain.

IOS-1208174 NSF
CRCNS: Data Sharing: Open Web Atlas for High-Resolution 3D Mouse Brain Data 09/01/12 to 08/31/15

**Co-PIs** - Yoonsuck Choe, John Keyser, Louise Abbott
This project helped develop mapping algorithms for developing high resolution three-dimensional brain maps of the mouse brain.

NIH/NLM
Large-scale reconstruction of microvascular networks 10/01/13 – 8/31/15
Sub-award to University of Houston (PI David Mayerich)

**Louise Abbott** – role – consultant
Role on grant was to help prepare brain and other organs for sectioning and analysis using the KESM.

IDBR-1215422 NSF
Enhanced Knife-Edge Scanning Microscopy for Sub-micrometer Imaging of Whole Small Animal Organs 04/01/13 to 03/31/15

**Co-PIs** - Yoonsuck Choe, John Keyser, Louise Abbott
This project helped enhance use of Knife Edge Scanning Microscope by developing its use with fluorescence microscopy

3R01NS042859 NIH - NINDS Louis (PI) 09/01/09 to 08/31/14
Pathogenesis of Essential Tremor: Cerebellar Metabolism
Role - consultant
This project helped elucidate the pathogenesis of essential tremor in humans.
A. PERSONAL STATEMENT

Our work over the last 15 years has largely focused on the role of chemosensory receptors in the model system *Drosophila melanogaster*. As an independent investigator at Duke University, my group and those of J. Carlson and R. Axel, identified the *Drosophila* gustatory receptors (Gr) genes. Shortly thereafter, we discovered that two members of this family encode pheromone receptors that control courtship behavior. At the same time, we initiated a major project with the goal to elucidate the role of the many *Drosophila* sugar receptors in sweet taste. In contrast to mammals, which have a single, broadly tuned, heterodimeric sugar receptor, *Drosophila* employs many sugar heteromeric sugar receptors, encoded by eight related sugar Gr genes. We undertook an extensive molecular genetic approach to assign specific functions for each of these Gr genes by generating null/expression alleles for all eight sugar Gr genes. These studies have indicated the need of a thorough re-calibration of the proposed model of insects taste sweet chemicals (see Scientific contributions). More recently, we have expanded our interest into other appetitive taste modalities, those of fatty acid taste, acid/sour taste and amino acid taste, studies proposed for this competitive renewal.

Two of the most important facts emerging from our work, and that of our colleagues, over the last ten years are the recognition that (i) taste coding is much more complex than anticipated from analogy to mammalian taste systems and (ii) taste receptors have critical, non-canonical functions in other sensory systems. Both these observations are the rationale for the current submission on taste coding of appetitive taste modalities, as well as a recently granted award on brain nutrient sensors. I am convinced that much of the exciting work by investigators studying taste processing in the higher brain will not only benefit from the findings that will emerge from this application, but indeed will need to rely on them for proper interpretations of their results and observations.

Publication output between 2009 and 2012 was very modest. The major reason for this reduced productivity was related to the illness of Isabel, my first-born daughter, who was diagnosed with a brain tumor shortly after moving my laboratory from Duke University to Texas A&M University. Isabel passed away in May 2011. Thanks to my team of dedicated students and postdocs, who stayed focused and kept producing high quality data, I have regained the enthusiasm for research, and we are poised to keep making impact in our field.

B. POSITION AND HONORS

Positions and Employment

1998-2005 Assistant Professor, Department of Molecular Genetics and Microbiology, Duke University Medical Center, Durham, NC
2005-2009  **Associate Professor** (with tenure), Department of Molecular Genetics and Microbiology, Duke University Medical Center, Durham, NC
2009-present  **Professor** (with tenure), Department of Molecular and Cellular Medicine, Texas A&M Health Science Center, College Station, TX
2012-present  **Associate Chair**, Department of Molecular and Cellular Medicine, Texas A&M Health Science Center, College Station, TX

**Patents, other professional experiences and memberships**

1999  # US7241881 **Identification of a family of genes encoding insect (fruit fly; *Drosophila melanogaster*) odorant receptors**. Awarded in 2005. Inventors: Leslie B. Vosshall, Richard Axel and Hubert O. Amrein

2003 - 2011  **Editorial Board Member** RNA Biology  Landes Biosciences Publishing
2004 – present  **Editorial Board Member** Current Biology  Elsevier Press
2009 – 2014  **Member**, Somatosensory and Chemosensory Systems study section
2009 – present  **Editorial Board Member** Frontiers in Neural Circuits  Frontiers Press
2011 – present  **Editorial Board Member** Brain and Behavior  Wiley Publishing
2014 – present  **Editor in Chief** Fly  Taylor & Francis Publishing

**Honors and Awards**

1984  **Short Term Fellow**, European Molecular Biology Organization  Laboratory of Dr. V. Pirrotta, EMBL Heidelberg, **Germany**
1989 – 1991  **Long Term Fellow**, European Molecular Biology Organization  Laboratory of Dr. T. Maniatis, Harvard University, Cambridge, **MA**
1991 – 1993  **Senior Fellow**, Swiss National Science Foundation  Laboratory of Dr. R. Axel, Columbia University, New York, **NY**
2014  **Presidential Symposium Speaker**, Association of Chemosensory Sciences, Annual Conference, **Fort Myers, FL**
2014  **Senior Faculty Award for Excellence in Research**, Texas A&M Health Science Center, **College Station, TX**

**C. SCIENTIFIC CONTRIBUTIONS**

1) Regulation of alternative splicing

My early work as a graduate student and postdoctoral fellow involved the molecular–genetic characterization of the sex-determining gene *transformer2* (*tra2*) in *Drosophila melanogaster*. Sex determination is a highly regulated process that involves a genetic cascade of five regulatory proteins. At the time, nothing was known about the molecular mechanism by which this cascade implemented the different developmental programs of female and male flies. I identified *tra2* and showed that it encodes an RNA binding protein/splicing factor necessary for alternative splicing of the *double-sex pre-mRNA*. I also characterized the different protein domains of Tra2 and identified specific protein–protein interaction domains, as well as RNA binding domains. These studies were seminal contributions that lead to the elucidation of the molecular mechanism of this important RNA regulatory mechanism.


2) Non-coding RNAs in Dosage compensation

While searching for the insect olfactory receptor genes during my postdoctoral studies with Richard Axel, I embarked on a second project: identifying sex-specific genes expressed in the brain, potential target genes of both the sex determination and the dosage compensation regulatory cascades in *Drosophila*. At the time, no sex-specific effector genes were known. I developed a strategy that used GFP-expressing GAL4 lines to label specific cells in the brain (sorted by FACS analysis from the brain mushroom bodies, a structure critical for learning and memory) of both sexes, and generated cDNA libraries. I identified two male-specific non-coding genes (*roX1* and *roX2*) that were localized in the male nucleus and associated specifically with the single male X chromosome. This discovery laid the groundwork for numerous investigators to uncover the basic mechanism of X-linked gene up-regulation in *Drosophila* dosage compensation.


3) Identification of *Drosophila* chemosensory receptor genes

My late postdoctoral early work and early work as an independent investigator were characterized by two critical gene discovery projects, the identification of olfactory receptor genes and gustatory receptor genes in the fly. The discovery of olfactory receptor genes in mammals almost 10 years earlier by Buck and Axel has directed the field of neuroscience into a molecular-genetic trajectory. At the same time, it became also clear that many of the most intriguing questions could be explored more easily in simpler model systems, providing an impetus to identify chemoreceptor genes in insects, leading to the discovery of the olfactory receptor genes in the fly in the late nineties. In my own lab, we quickly identified a second large group of chemoreceptor genes, the gustatory receptor genes (along with the Axel and the Carlson labs), providing the foundation for my career in this field. At the time, I was especially interested in the question how males and females use chemosensory cues for mate and status recognition and initiated a search for the elusive pheromone receptors, the first of which we identified and characterized in 2003 and 2008, respectively.


4) Sweet taste coding

The complete sequence of the *Drosophila* genome and expression studies using the GAL4 system initiated by several laboratories, combined with earlier electrophysiological studies, allowed the formulation of clear hypotheses as to the nature and function of specific *gustatory receptor* (*Gr*) genes. These, largely descriptive parameters suggested that flies use up to eight sugar receptors, a clear deviation from how mammals sense sweet foods. In addition, several genetic studies suggested that functional taste receptors (both for bitter and sugar chemicals) are comprised of two or more *Gr* subunits. However, a particular difficulty in studying the function and composition of sugar receptor was presented by the arrangement of six of the eight sugar *Gr* genes in the genome (*Gr64a* to *Gr64f*). These genes form a tight cluster, and consequently, specific mutation were lacking. Nevertheless, two groups promoted a model that has become largely accepted in which only two receptors (*Gr5a/Gr64a* and *Gr5a/Gr64f*) provide for sweet taste. Our extensive and detailed molecular genetic analysis (Reference d below) clearly rejects this model and instead indicates that all sugar *Gr* genes contribute to sweet taste. Specifically, our expression data, all based on precise gene-knock in alleles, and their phenotypic analyses, are inconsistent with *Gr64a* being a major component of a sweet taste receptor. Likely, the Carlson/Montell model, which was promoted in a
near ‘vacuum’ of cellular expression data, was based on over-interpretation of the few available Gr64 alleles used in these studies. Our investigations of the Gr64 genes have also identified additional likely roles for some of these sugar Gr genes in other chemosensory modalities, including olfaction and internal nutrient sensing (see below).


5) Brain Nutrient Sensors
One of the most exciting discovery during my career was the identification of the first, brain based nutrient sensor that is directly involved in feeding modulation (reference a below). Prompted by (at the time) anecdotal reports mostly from mammalian systems of chemoreceptors being expressed in the digestive and nervous system, we initiated a rather unconventional project. The genome resources (sequence data of many Drosophila species, as well as other arthropods) revealed that a few Gr genes are highly conserved, including Gr43, suggesting important functions for these receptors. Thus, we generated a GAL4 knock-in for Gr43 and found it to be expressed (among a few other sites) in neurons clustered in the posterior lateral protocerebrum of the brain. By developing a new ex vivo imaging method, we were able to show that this receptor functions as fructose sensor, and using Gr43a mutant and wild type flies, combined with hemolymph (blood) analysis, we found that this nutrient sensor senses fluctuating fructose levels in the hemolymph, brought about from meals of carbohydrate rich food. Moreover, we showed that the Gr43a brain fructose sensor regulates feeding behavior of the fly, based on satiation status. More recently, we identified a second brain nutrient sensor, expressed in the same cells as Gr43a. This sensor, Gr64a, was thought to be a major player in peripheral sweet taste (see reference d in section 4 above). Instead, our analysis has revealed that the function of this gene is likely sensing of yet another sugar present in the hemolymph/brain.


D. RESEARCH SUPPORT

Active:
2003 – 2020 Taste Receptor Genes and Sensory Coding NIH-1RO1GMDC05606-12 (PI H. Amrein)
Total award sum (direct costs): ~ $3,000,000 (entire lifetime of grant). The goal of this grant is to elucidate the specific functions of the putative sugar receptors (Gr5a, Gr61a and Gr64a-f) in sugar sensing. In addition, we seek to determine the molecular basis of other nutritious chemicals, including fatty acids, carboxylic acids and amino acids.

2014 -2019 Regulation of Feeding Behavior by Brain-based Nutrient Sensors NIH-1RO1 DC013967-02 (PI H. Amrein). Total award sum: ~ $1,250,000 (direct costs).
The goal of this grant is to determine the nature of brain nutrient sensor and to assess their role in feeding behavior and nutrient metabolism.

2016-2018 **Gustatory Receptors sense RNA and ribonucleic acid metabolites as nutrients and signaling molecules during rapid growth** NIH-R21DC15327-01 The goal of this grant is to characterize the role of six taste receptors in sensing ribose and ribose containing metabolites in taste perception of RNA and RNA precursors, as well as in sensing yet unknown internal metabolites in temperature and light sensing.

**Completed:**

2008 - 2013 **From Pheromone Receptor to Social Behaviors** NIH-1RO1 DC009014-01A1 (PI H. Amrein) Total award sum $1,075,800 (direct costs)

2006 – 2010 **Identifying Cardiomyopathy Genes by ENU Mutagenesis in Mice, Zebrafish and Drosophila** (PI H. Rockman). Total Award sum 1,250,000 (direct costs); Fraction to H.A. 30,000/year

2004 - 2007 **From Pheromone Receptor to Complex Mating Behavior** NSF 0349671 (PI H. Amrein), Award sum $367,722 (total)

2000 – 2003 **RNAs in Transcriptional Regulation and Dosage Compensation** NIH-1RO1GM60234-01; Total award sum $683,080 (direct costs) PI: H. Amrein
**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:** Brian A. Anderson

eRA COMMONS USER NAME (credential, e.g., agency login): bander33

**POSITION TITLE:** Assist. Professor of Psychology

**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.)*

<table>
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<th>Completion Date</th>
<th>FIELD OF STUDY</th>
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<td>Univ. of Maine at Augusta</td>
<td>B.A.</td>
<td>2006</td>
<td>Social Science</td>
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<tr>
<td>Villanova University</td>
<td>M.S.</td>
<td>2009</td>
<td>Psychology</td>
</tr>
<tr>
<td>Johns Hopkins University</td>
<td>Ph.D.</td>
<td>2014</td>
<td>Psychological &amp; Brain Sciences</td>
</tr>
<tr>
<td>Johns Hopkins University</td>
<td>Post Doc</td>
<td>2016</td>
<td>Psychological &amp; Brain Sciences</td>
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**A. Personal Statement**

In order for an organism to survive and thrive, attention must be directed to stimuli that are associated with rewarding outcomes so that these stimuli can be appropriately acted upon and the reward obtained. However, attention to reward-associated stimuli can become maladaptive when pursuing the associated reward conflicts with current goals, as in the case of desired abstinence from a substance of abuse. In my research, I investigate how selective attention is influenced by reward learning. I was among the first to argue that learned stimulus-reward associations have a direct impact on attention that is distinct from the well-documented mechanisms of goal-directed and stimulus-driven selection, what I have referred to as **value-driven attention**. The discovery that stimuli previously associated with reward automatically capture attention even when inconspicuous and task-irrelevant has now been replicated by several different research groups and has played an instrumental role in shaping recent theoretical developments concerning the control of attention. Much of my recent and ongoing research efforts focus on better understanding the mechanisms by which reward learning shapes attentional selection. I have also provided evidence linking this basic mechanism of attentional selection to psychopathology, specifically addiction, depression, and impulsive, high-risk behaviors. Individuals characterized by these conditions exhibit abnormal sensitivity to the influence of reward learning on attention. In addition, I have published several studies on goal-contingent attentional orienting and maintain an active line of research in this area. My expertise in goal-directed attention greatly enriches my insight into how and when reward learning interacts and interferes with goal-directed processes. In the proposed study, I seek to decomposed value-based attentional priority into separable underlying mechanisms. As a leader in this area, with demonstrated expertise in all aspects of the proposed research, I am well positioned to be successful in carrying out this important work. The findings from the proposed research will provide a firm foundation for beginning my career as a professor and independent scientist.


B. Positions and Honors

**Professional Experience**

2008-2009 Graduate Research Assistant, Villanova University  
2009-2014 Graduate Research Fellow, Johns Hopkins University  
2014-2016 Postdoctoral Scientist, Johns Hopkins University  
2016- Assist. Professor of Psychology, Texas A&M University

**Honors**

2007 Summa Cum Laude with Honors (University of Maine at Augusta)  
2007 Distinguished Social Science Student (University of Maine at Augusta)  
2008 Full Graduate Assistantship (Villanova University)  
2008 Travel Award (Villanova University)  
2008 Alumni Association Textbook Scholarship (Villanova University)  
2009 Ingeborg L. and O. Byron Ward Outstanding Thesis Award (Villanova University)  
2012 Walter L. Clark Collaborative Research Award (Johns Hopkins University)  
2012 Robert S. Waldrop Junior Investigator's Award (Johns Hopkins University)  
2013 G. Stanley Hall Scholar's Award (Johns Hopkins University)  
2014 Select-Speaker Award (Psychonomic Society)  
2015 New Investigator Award (American Psychological Association, Division 3)

C. Contribution to Science

1. Establishing a distinctly value-driven mechanism of attentional selection. My work in this area demonstrates that stimuli previously associated with reward automatically capture attention even when inconspicuous and task-irrelevant, which cannot be explained by other known mechanisms of attentional selection. This work contributes to basic theories of attentional control.


2. Characterizing the neural mechanisms underlying value-driven attention. Using functional magnetic resonance imaging (fMRI), I have implicated the visual corticostriatal loop in the signaling of value-based attentional priority, and have identified teaching signals in the visual attention system during reward learning. Using positron emission tomography (PET), I have directly linked value-driven attentional capture to the release of striatal dopamine.


3. Characterizing individual differences in the ability to override value-driven attentional capture. I have demonstrated that drug-dependent patients are less capable of ignoring irrelevant but previously reward associated stimuli, showing more pronounced attentional capture by such stimuli relative to healthy controls. Conversely, previously reward-associated distractors have a reduced impact on the attention of depressed individuals relative to controls. I also showed that value-driven attentional capture is negatively correlated with visual working memory ability, and positively correlated with impulsiveness. This work establishes the clinical significance of value-driven attention as a cognitive mechanism.


4. Clarifying the learning principles underlying value-driven attention. I have shown that value-driven attentional biases can be strongly modulated by context-specific learning, and that the influence of these biases can extend to the facilitation of overt behavior. I have also shown that prediction-error learning is critical for value-driven attentional biases to occur, which cannot be explained by the incentive properties of reward alone. This work establishes principles that describe when reward learning should and should not bias attention, which has both theoretical and translational implications.


5. Characterizing the precision of goal-directed attentional control. My work in this area demonstrates that the precision with which individuals are able to orient attention to a stimulus on the basis of whether it possesses a currently prioritized feature is limited. Individuals automatically orient attention to task-irrelevant stimuli that are similar in color to a searched-for target, and default to orienting towards physically salient objects rather than searching for multiple possible features simultaneously. This work contributes to basic theories of attentional control.


Complete List of Published Work:

D. Research Support

**Ongoing Research Support**

R01-DA041264 (PI: Cherie Marvel) 08/01/2016-07/01/2021 Co-investigator  
NIDA  
*HIV-Related Neuroplasticity and Attention-to-Reward as Predictors of Real World Function*  
The major goal of this project is to predict neurocognitive outcomes and risk-behaviors of HIV+ patients using behavioral and neural measures of reward-related attentional bias.

Landenberger Foundation Grant (PI: Cherie Marvel) 8/1/2015-7/31/2017 Co-investigator  
Landenberger Foundation  
*Identifying the Neurocognitive Determinants of HIV-Risk Behaviors*  
The major goal of this project is to characterize the neural correlates of value-driven attentional capture in drug-dependent and HIV+ populations, and how those correlates differ from those of healthy controls.

PESCA Grant Program 05/01/2017-04/30/2018 Co-PI  
Texas A&M University Division of Research  
*Neural Mechanisms of Attention to Pain Cues*  
Funding will support an fMRI study investigating the neural correlates of attentional capture by stimuli associated with an aversive outcome.

**Completed Research Support**

NIDA  
*Cortical and Subcortical Mechanisms of Human Cognitive Control*  
The major goal of this project was to characterize the behavioral and neural mechanisms by which individuals shift the focus of attention and the effect of reward learning on involuntary attentional selection.

P30-MH075673 (PI: Justin McArthur) 07/01/2013-06/30/2014 Co-investigator  
NIMH  
*Examining the Role of Attentional Bias on Risk Taking Behavior in HIV+ Patients*  
The major goal of this project was to relate attentional biases for reward-associated stimuli to HIV-risk behaviors and different aspects of HIV-associated neurocognitive disorder in an HIV+ sample.

F31-DA033754 (PI: Brian A. Anderson) 06/01/2012-05/30/2014 PI  
NIDA  
*Mechanisms of Value-Driven Attentional Capture*  
The major goal of this project was to characterize the cognitive and neural mechanisms by which previously reward-associated stimuli automatically capture attention in both healthy and drug-dependent populations, using both behavioral (psychophysics, eye tracking) and fMRI methodologies.
Biographical Sketch

Spencer T. Behmer
Professor
Department of Entomology, Texas A&M University, College Station, TX  77843-2475
Phone: (979) 845-7304
Email: s-behmer@tamu.edu; Webpage: http://behmerlab.tamu.edu/index.html

A. PROFESSIONAL PREPARATION

<table>
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<th>Institution</th>
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<tr>
<td>University of Nebraska-Lincoln</td>
<td>Biology</td>
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<td>University of Nebraska-Lincoln</td>
<td>Biology/Ecology</td>
<td>M.S., 1993</td>
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<tr>
<td>The University of Arizona</td>
<td>Entomology</td>
<td>Ph.D., 1998</td>
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B. ACADEMIC/PROFESSIONAL APPOINTMENTS

2014 - present  Professor, Department of Entomology, Texas A&M University
2011 - 2014  Associate Professor, Department of Entomology, Texas A&M University
2005 - present  Member, Faculty of Ecology & Evolutionary Biology, Texas A&M University (Chair (2014-present); Associate Chair (2011-2014))
2008 - present  Member, Institute of Neuroscience, Texas A&M University
2005 - 2011  Assistant Professor, Department of Entomology, Texas A&M University
2002 - 2003  Departmental Lecturer, Department of Zoology, University of Oxford
2001 - 2005  College Lecturer, Hertford College, University of Oxford

C. Products

Five Relevant Publications


**Five Additional Publications**


**D. FIVE SYNERGISTIC ACTIVITIES**

1) Editor-in-Chief, *Journal of Insect Physiology* (Jan 2013); Associate Editor, *Journal of Animal Ecology* (Nov 2012); Associate Editor, *BMC Physiology* (Jul 2010)

2) Elected Vice-Chair (2017) and Chair (2019) for the Gordon Research Conference on Plant-Herbivore Interactions

3) NSF IOS pre-proposal panel (2016); NIFA-USDA full proposal panel (2017)


5) Co-Chair for symposium “Integrative nutrition: from physiology to ecology and beyond” (XIV International Congress of Entomology, Deagu, South Korea; 18 invited speakers)
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: Bernard, Jessica Ann

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

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.)

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<td>Tufts University, Medford, MA</td>
<td>BS</td>
<td>05/2007</td>
<td>Biopsychology</td>
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<tr>
<td>University of Michigan</td>
<td>MS</td>
<td>12/2009</td>
<td>Psychology</td>
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<tr>
<td>University of Michigan</td>
<td>PHD</td>
<td>08/2012</td>
<td>Psychology</td>
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<tr>
<td>University of Colorado Anschutz Medical Campus</td>
<td>Postdoctoral Fellow</td>
<td>08/2013</td>
<td>Neurology and Aging</td>
</tr>
<tr>
<td>University of Colorado Boulder</td>
<td>Postdoctoral Fellow</td>
<td>07/2015</td>
<td>Psychology</td>
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A. Personal Statement

My role in this project is that of Co-PD/PI. I am an Early Stage Investigator making my first R01 application. I am a new Assistant Professor at Texas A&M University, and have been building a line of research investigating the cerebellum in motor and non-motor function across the lifespan and in psychopathology. Over the course of my graduate and post-doctoral research training, I have developed the expertise necessary to carry out the proposed project. I will serve as the project director and principal investigator on this proposal in direct collaboration with my colleague, the Co-PI Dr. Joseph Orr. We have assembled a team of talented Co-Investigators and Consultants who will provide critical support and insight with respect to different aspects of the project. As a doctoral student working with Dr. Rachael Seidler at the University of Michigan, I received training in transcranial magnetic stimulation, resting state functional connectivity MRI, as well as in structural brain imaging, and motor and cognitive behavioral assessments. I also gained a strong theoretical grounding in cerebellar function. I followed this with a post-doctoral fellowship at the University of Colorado working with Dr. Vijay Mittal. While working with Dr. Mittal, I gained further neuroimaging experience, and broadened my technical arsenal to include diffusion tensor imaging. I am currently the PI of a Brain and Behavior Research Foundation NARSAD Young Investigator Award. I am investigating differences in functional activation of the cerebellum in psychosis risk, with respect to internal model formation and disease progression. This grant has allowed me to conduct independent functional imaging work, and has provided me with important experience in grant and project management as PI. The present proposal builds directly off of my knowledge and experience investigating the cerebellum in non-motor behavior in young and older adulthood. As an independent investigator, I am expanding my investigations of the cerebellum to include its role in executive function, and its relationships with the prefrontal cortex. The current proposal stands to provide important new insight into the role of the cerebellum in non-motor behavior more generally, and into the circuits underlying executive function more specifically. The team of investigators including myself, Co-PI Orr, and Co-Investigator Ji, represent a team of individuals with strengths in cerebellar and prefrontal function, non-invasive brain stimulation, brain imaging and data analysis (both functional and white matter imaging), and data processing of large computationally demanding data sets. Together our technical expertise will allow us to investigate important new questions related to cerebellar-prefrontal interactions as they pertain to executive function, which has implications for our understanding of numerous disease states. We are well prepared to successfully carry out the research proposed in this application.


B. Positions and Honors

Positions and Employment

2015 -
Assistant Professor, Department of Psychology, Texas A&M University, College Station, TX

Honors

2009
Student of the Year Award, Michigan Center for Advancing Safe Transportation Throughout the Lifespan (MCASTL)

2009
Barbara A. Oleshansky Memorial Award, University of Michigan

2009
Graduate Research Fellowship Program Honorable Mention, National Science Foundation

2010
Barbara Perry Roberson Fellowship, University of Michigan

2012
Rackham One-Term Dissertation Fellowship, University of Michigan

2014
Clinical Loan Repayment Award, National Institutes of Health

2015
Young Investigator Travel Award, International Congress on Schizophrenia Research

2016
Clinical Loan Repayment Renewal Award, National Institutes of Health

C. Contribution to Science

1. Cerebellar Contributions to Age-Related Performance Differences. While it is well-established that older adults experience declines in both cognitive and motor performance, the status quo with respect to research seeking to better understand these declines was to focus on cortical underpinnings. Cerebellar contributions had been minimally investigated. For my doctoral dissertation, I investigated the role of the cerebellum in age-related motor and cognitive performance differences. This work demonstrated that in healthy older adults, there are decreases in cerebellar volume that impact the structure on a regional basis. Furthermore, there is decreased functional connectivity between the cerebellum and the rest of the brain; prefrontal and basal ganglia connections are particularly impacted. Both structure and connectivity contribute, at least in part, to motor and cognitive performance deficits in older adulthood. I have since synthesized these results into a hypothesis which purports that internal models of behavior are degraded in older adults, due to the cerebellar structural and connectivity declines, impacting behavior. Together, these studies have moved the field forward by emphasizing the contribution of the cerebellum to age related performance differences. This work has opened up new avenues of research with respect to the role of the cerebellum and aging, that would have otherwise remained closed. Further, this work has provided new insights into the role of the cerebellum in cognitive performance, which is highly relevant to this proposal.


2. Cerebello-Cortical Circuits and Development. Work using animal models has demonstrated that there are distinct connections between the cerebellum and motor and prefrontal regions of the cortex. This was at the level of the cerebellar cortex and lobules, but also in the cerebellar dentate nucleus. Furthermore, it had been suggested that regions that are more strongly associated with the prefrontal cortex, would develop more slowly than motor areas, consistent with cortical development. However, there were no direct investigations of these distinct cerebello-cortical circuits, or their development. As a doctoral student, and during my postdoctoral fellowship, I investigated these ideas. First, using resting state connectivity, I found
that there are distinct cerebello-frontal and motor circuits in the human cerebellar cortex and in the human dentate nucleus. Following up on these findings using a longitudinal developmental approach, I demonstrated that the cerebello-frontal circuits develop more slowly than the motor circuits. Structural analysis of distinct cerebellar regions also supports this. Thus, this work greatly advanced our knowledge of cerebello-cortical circuits in the human brain, and provided important insight into cerebellar development. This research lays important groundwork for both basic and clinical research investigating the cerebellum during development, given its known contributions to cognition and its purported role in a variety of diseases. However, it is notable that the connections with the prefrontal cortex were considered quite broadly; the current proposal builds off of these findings but stands to provide a more functionally relevant understanding of circuits between the cerebellum and prefrontal cortex.


3. Handedness and Age Influence Motor Cortical Representations. My early publications addressed factors that influence the representations of digits in the primary motor cortex. The primary motor cortex is highly plastic, and changes in motor cortical representations can be seen with practice, and after injury. However, there is also variability in these representations across individuals. Understanding factors that contribute to this variability is important when designing treatments or interventions related to injury. Both handedness, and normal aging may be contributing factors. Using transcranial magnetic stimulation to map hand muscle representations in conjunction with a behavioral measure of interhemispheric communication, I investigated the impact of handedness on these representations in young and older adults. This work demonstrated that handedness is a contributing factor to these representations, and is also related interhemispheric communication. Furthermore, the relationships with handedness change in advanced age, such that there is an interactive effect of age and handedness on motor cortical representations. Finally, as a part of this line of work I also demonstrated that older adults experience motor cortical dedifferentiation. The hand muscle representations were more diffuse, and this negatively impacted motor performance. Together this work vertically advanced the field by demonstrating important factors that influence the organization of the motor cortex, extended the notion of dedifferentiation with age to the motor cortex. Important for this proposal, I gained crucial experience with non-invasive brain stimulation.


4. Cerebellar Contributions to Psychosis and Disease Course in Psychosis Risk. The cognitive dysmetria hypothesis has posited that the cerebellum plays a role in the symptoms and cognitive deficits seen in patients with schizophrenia. In my postdoctoral work, I led a team of researchers with my mentor in investigations of the cerebellum with respect both symptomatology and disease course in youth at ultra-high risk for psychosis. Using meta-analysis, structural neuroimaging, and both structural and functional brain connectivity analyses, this work furthered our understanding of the role of the cerebellum in
psychosis. Cerebellar dysfunction is present during the performance of both motor and cognitive tasks. Furthermore, this work provided evidence for cerebellar impairments in structure, and with respect to its interactions with the rest of the brain, prior to the onset of formal psychosis in at-risk populations. This work is innovative in that it suggests a more causative role for the cerebellum in the pathophysiology of psychosis, and has opened up new avenues of research that investigate the cerebellum as a marker of disease progression and seeks to integrate this cerebellar cognitive dysmetria approach with other prominent hypotheses regarding the pathophysiology of psychosis.


b. Dean DJ, Kent JS, Bernard JA, Orr JM, Gupta T, Pelletier-Baldelli A, Carol EE, Mittal VA. Increased postural sway predicts negative symptom progression in youth at ultrahigh risk for psychosis. Schizophr Res. 2015 Mar;162(1-3):86-9. PMCID: PMC4339540.


Complete List of Published Work in My Bibliography:

D. Additional Information: Research Support and/or Scholastic Performance

Ongoing Research Support

Young Investigator Award, Donald and Janet Boardman Family Investigator, Brain and Behavior Research Foundation (NARSAD)

Bernard, Jessica Ann (PI)
01/15/15-01/14/17 (No-cost extension through 1/14/18)
Cerebellar-Prefrontal Involvement in Error Processing and Rule Learning in Youth at Ultra High-Risk for Psychosis
The goal of this investigation is to identify differences in cerebellar function during a non-motor task in youth at ultra-high risk for psychosis. Furthermore, cerebellar functional activation is being tested as a possible predictor of disease progression over the course of 12 months.
Role: PI

L30 MH104874-01
Bernard, Jessica Ann (PI)
07/01/14-06/30/17
Cerebellum and Psychosis Risk
Renewed through June 2017.
Role: PI

Completed Research Support

F32 MH102898-02
Bernard, Jessica Ann (PI)
09/30/13-07/31/15
Cerebellar Contributions to Disease Course in Youth At High-Risk of Psychosis
Role: PI
NAME: Bolanos, Carlos A.

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

POSITION TITLE: Associate Professor; Texas A&M University

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.)

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<td>California State University, San Bernardino, CA.</td>
<td>B.A.</td>
<td>05/1992</td>
<td>Psychology</td>
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<td>California State University, San Bernardino, CA.</td>
<td>M.A.</td>
<td>05/1995</td>
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<tr>
<td>Northeastern University, Boston, MA.</td>
<td>Ph.D.</td>
<td>05/2000</td>
<td>Exp. Psychology</td>
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<td>Yale Univ., Dept. Psychiatry, New Haven, CT.</td>
<td>Postdoc</td>
<td>08/2000</td>
<td>Neuroscience/Behavior</td>
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<tr>
<td>Univ. Texas Southwestern Medical Ctr., Dallas, TX.</td>
<td>Postdoc</td>
<td>07/2004</td>
<td>Molecular Neuroscience</td>
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A. PERSONAL STATEMENT

My research interests center on investigating how exposure to psychotropic drugs (NIDA R01DA026854) and stress (NIDA R21DA022351) modify the biochemical integrity of neural pathways involved in the regulation of mood and motivated behaviors, and how these pharmacological and/or environmental manipulations early in life affect functional outputs in adulthood. This work has focused on establishing causal relationships between early-life experiences, brain, biochemistry, and behavior. Specifically, my laboratory is guided by the clinical and epidemiological literature in generating clinically relevant questions using animal models. We have published on the lasting behavioral, biochemical and molecular consequences of psychotropic drug exposure such as methylphenidate (Bolaños et al., 2003, 2008; Legace et al., 2006; Wiley et al., 2009), fluoxetine (Iñiguez et al., 2010, 2014), and combined methylphenidate (MP) and fluoxetine exposure (Warren et al., 2011; Alcantara et al., 2014; Steiner et al., 2014) during adolescence. Our work has demonstrated that combined MPH+SSRI exposure during adolescence results in neurobiological profiles similar to those observed after exposure to cocaine, and that this combined treatment enhances behavioral and biochemical sensitivity to other psychostimulants such as nicotine and cocaine as measured by the conditioned place preference paradigm (CPP).


B. POSITIONS AND HONORS

Positions and Employment

2000  Postdoctoral Fellow, Yale University, Department of Psychiatry, New Haven, CT.
2000-04 Postdoctoral Fellow, University of Texas, Southwestern Medical Center, Dallas.
2004-11 Assistant Professor, Psychology and Neuroscience, Florida State University.
2011-16 Associate Professor, Psychology and Neuroscience, Florida State University.
2016– Associate Professor, Psychology and Neuroscience, Texas A&M University.

Advisory Panels

2007  CSR, NIMH, Special Emphasis Panel ZMH1SRC (99).
2009  CSR, NCAM-NIH, Special Emphasis Panel ZAT1 PK (09).
2009  U.S. Civilian & Development Foundation Independent States of Former Soviet Union (STCU #5008)
2010  CSR, NIMH, Special Emphasis Panel ZMH1 ERB-L (03).
2011  CSR, NIHCSR, ETTN - ZRG1 F02A-J (20) L, Study Section.
2011  CSR, NIHCSR, Special Emphasis Panel ZRG1 F02A-J 20L, Training Fellowships.
2012  CSR, NIHCSR – ZRG1-J (20) L, Study Section (NIMH/NIDA).
2012  National Hispanic Science Network on Drug Abuse (NHSN).
2012-present Editorial Board, Neuropsychopharmacology.
2013  CSR, NIHCSR – ZRG1-J (20) L, Study Section (NIMH/NIDA).
2013  CSR, NIHCSR – Biobehavioral Regulation, Learning and Ethology (BRLE), Study Section (NIH).
2014  CSR, NIHCSR – ZRG1-J (20) L, Study Section (NIMH/NIDA).
2014-20 Member, CSR, NIHCSR – BRLE, Study Section (NIH).
2016-present Associate Editor, Neuroscience Letters.

Fellowships and Awards

1995  Outstanding Graduate Student of the Year, CSUSB.
1995  Neuroscience Internship, Neuropsychiatric Institute, UCLA.
1996  Society for Neuroscience Scholar.
1997  Gordon Conference on Catecholamines Travel Award, New Hampshire.
1997-00 Pre-Doctoral Fellowship National Institute on Mental Health (NIMH).
1999  Gordon Conference on Catecholamines Travel Award, Oxford, UK.
2001  International Behavioral Neuroscience Society Travel Award, Cancún, Mexico.
2001-04 National Research Service Award (NRSA F32), National Institute on Drug Abuse (NIDA).
2002  Travel Fellowship, American College of Neuropsychopharmacology (ACNP).
2004  National Alliance for Research on Schizophrenia and Depression, Young Investigator.
2005  First Year Assistant Professor Award, Florida State University, Council for Research and Creativity.
2006  Early Career Investigator Travel Award, NIDA/APA Divisions 28 & 50, New Orleans, LA.
2009-11 Elected Associate Member to the American College of Neuropsychopharmacology (ACNP).
2012  Elected Full Member to the American College of Neuropsychopharmacology (ACNP).
2012  Developing Scholar Award, Florida State University.
2014  Nancy Marcus Professorship, Florida State University.
2017  Elected Fellow to the American College of Neuropsychopharmacology (ACNP).

C. CONTRIBUTIONS TO SCIENCE

1. My research interests have focused on an important, yet grossly understudied area of research:
understanding how early life pharmacological, environmental, and genetic perturbations alter brain
biochemistry to regulate functional outputs throughout the lifespan. My earlier work revealed that the
behavioral and biochemical responses to psychotropic drugs are age-dependent. Early postnatal
development and adult periods respond qualitatively similar to psychostimulants as measured by the
locomotor sensitization and conditioned place preference (CPP) assays, whereas the period of adolescence is marked by a significant decrease in sensitivity to drugs of abuse.


2. More recent contributions have centered on delineating the neurobiological consequences of exposure to drugs used for the management of attention-deficit hyperactivity disorder (ADHD; methylphenidate, MPH) and depression (fluoxetine, FLX) during adolescence. MPH exposure results in enhanced vulnerability to stress-eliciting situations, decreased responsivity to rewards, and decreased hippocampal neurogenesis. These deficits are mediated by dysregulation of dynorphin binding by the kappa-opioid system, such that activation with kappa drug doses that produce no effect in non-MPH-treated animals exacerbate, whereas blockade of kappa receptors, or treatment with FLX, reverse the functional deficits observed after MPH treatment. These results point to new molecular targets for the study and treatment of these central nervous system disorders, and complement/support our original observations of a depression-like phenotype because the hippocampus is believed to be a key brain area implicated in clinical depression in humans. My laboratory has also demonstrated that co-administration of MPH+FLX – drugs often prescribed together to manage behavioral and mood disorders associated with ADHD – may not result in a depression-like syndrome as observed in the MPH-exposed, but may increase vulnerability to the effects of drugs such as nicotine and cocaine (publications listed in section A).


3. In addition to the contributions described above, my work has also focused on investigating drug-induced alterations in intracellular signaling (CREB, ERK, PLCγ, ΔFosB; IRS2) in brain regions important for mood regulation and reward. This work has demonstrated that drug-induced and viral vector-mediated changes in transcription factor expression within the nucleus accumbens (NAc) and ventral tegmental area (VTA) are important in the formation of drug- and natural-reward associations. For instance, we have discovered that ERK2 activity decreases after chronic exposure to fluoxetine, whereas chronic exposure to stress increases its activity. Using viral vectors encoding ERK2, we have demonstrated that overexpression of ERK2 within the VTA increases vulnerability to stress, whereas a mutated form of ERK2 (to prevent its activity) decreases sensitivity to stress and drugs of abuse.


4. Very little is known about the neurobiology and long-term effects of physical and/or emotional stress exposure during periods prior to adulthood. My laboratory is engaged in addressing this gap in our basic knowledge by examining the short- and long-term behavioral and biochemical consequences of physical versus emotional stress in adolescent and adult rodents. We take advantage of our expertise using the social defeat model of chronic stress. In this model, a mouse ‘witnesses’ the exchange between an intruder and an aggressor mouse from the safety of an adjacent compartment. Our findings indicate that ‘witnessing’ aggression has long-term consequences in adult and developing mice, as they show behavioral deficits similar to those mice that experienced actual physical aggression.


D. RESEARCH SUPPORT

**Ongoing Research Support**

Texas A&M University          **Bolaños (PI)**          06/01/2016-05/30/2019

Generous start-up funds were provided to establish my laboratory at TAMU and fund various ongoing research projects in my lab. With these funds, we have calibrated all of our behavioral assays, which are fully functional.
Research Support Completed During the last Three Years

NIH R01DA026854      Bolaños (PI)      04/01/2010–03/31/2016
NIH/NIDA
“Ontogeny of Drug Exposure and Mood Dysregulation”
This study investigates the short- and long-term behavioral and biochemical consequences of early-life exposure to psychotropic drugs.
Curriculum Vitae

JOHN J. BUCHANAN  
Texas A&M University  
Department of Health & Kinesiology  
College Station, TX 77843-4243  
Phone: 979-862-3229  
Fax: 979-847-8987  
e-mail: jjbuchanan@tamu.edu

EDUCATION

<table>
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<tr>
<th>Institution</th>
<th>Degree</th>
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<th>Area</th>
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<tbody>
<tr>
<td>Florida Atlantic University</td>
<td>Ph.D.</td>
<td>1996</td>
<td>Complex Systems/Brain Science</td>
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<td>M.A.</td>
<td>1992</td>
<td>Experimental Psychology</td>
</tr>
<tr>
<td>Univ. of Texas at San Antonio</td>
<td>B.A.</td>
<td>1985</td>
<td>Cognitive Psychology</td>
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PROFESSIONAL EXPERIENCE

Professor of Kinesiology, Texas A&M University, 2011-present
Member, Graduate Faculty of the Health Science Center School of Graduate Studies, 2011-present
Member, Faculty of Neuroscience, Texas A&M University, 2007-present
Associate Professor of Kinesiology, Texas A&M University, 2005-2011
Assistant Professor of Kinesiology, Texas A&M University, 1999-2005
NIH Post-doctoral Fellow, R.S. Dow Neurological Sciences Inst., Oregon Health Sciences University, Human Posture and Postures Disorders Laboratory, 1996-1999
NIMH Pre-doctoral Fellow, Neuroscience Training Grant in Complex Systems and Brain Sciences, Human Brain and Behavior Laboratory, 1989-1995
Graduate Research Assistant, Department of Psychology, Florida Atlantic University, 1988

GRANTS: Current Funding

TAMU share: $199,976 for the period 9.1.14 to 8.31.17
PI A. Banerjee TAMU-DISE ($129,962), Co-PI J.J. Buchanan TAMU-HLKN ($70,014)
Other participating institutes: Cal State University Fullerton, Idaho State University

*Qatar National Research Fund*, Exoskeleton Based Stroke Rehabilitation with Augmented Reality. Total Award: $899,329 for the period 4.1.15 to 4.1.18
PI G. Langari TAMU-ETID, Co-PI J.J. Buchanan TAMU-HLKN
Other participating institutes: Cal State University Fullerton

PUBLICATIONS
(Bold text: current or former graduate student, *thesis or dissertation project)

Published manuscripts 2012-present


NAME: Rodolfo C. Cardoso

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

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.)

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<th>INSTITUTION AND LOCATION</th>
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<th>FIELD OF STUDY</th>
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<tr>
<td>Sao Paulo State University, Sao Paulo, Brazil</td>
<td>D.V.M.</td>
<td>12/2005</td>
<td>Veterinary Medicine</td>
</tr>
<tr>
<td>Sao Paulo State University, Sao Paulo, Brazil</td>
<td>M.S.</td>
<td>05/2009</td>
<td>Animal Reproduction</td>
</tr>
<tr>
<td>Texas A&amp;M University, College Station, TX</td>
<td>Ph.D.</td>
<td>08/2014</td>
<td>Physiology of Reproduction</td>
</tr>
<tr>
<td>University of Michigan, Ann Arbor, MI</td>
<td>Postdoctoral</td>
<td>06/2016</td>
<td>Reproductive Endocrinology</td>
</tr>
</tbody>
</table>

A. Personal Statement

My development as a scientist results directly from my training in reproductive biology, endocrinology and neuroendocrinology with outstanding mentors. My graduate training in physiology of reproduction and neuroendocrinology with Drs. Gary Williams and Marcel Amstalden as well as my postdoctoral training in reproductive endocrinology with Dr. Vasantha Padmanabhan (PI in this proposal) provide me with the experience, expertise and knowledge necessary to successfully carry out the proposed research project. I have a broad background in reproductive biology, with specific training in reproductive neuroendocrinology using female ruminants as animal models, directly relating to the proposed work. My peer-reviewed publications document my interests in the mechanisms underlying the environmental regulation of reproductive neuroendocrine function in female mammals. I have also been closely involved in the current studies led by Dr. Padmanabhan on the developmental origins of health and disease using sheep as a research model. My research experiences in working with ruminants and technical skills to perform the proposed neuroanatomical studies make me well-suited to contribute and support the proposed research. The current project builds upon my previous work at the University of Michigan as well as the current studies carried out in my laboratory at Texas A&M University.

B. Positions and Honors

Professional Positions

2014 – 2016 Postdoctoral Fellow, Department of Pediatrics, University of Michigan
2016 – present Assistant Professor, Department of Animal Science, Texas A&M University

Professional Memberships

2010 – present Society for the Study of Reproduction
2014 – present Endocrine Society
2016 – present Texas A&M Institute for Neuroscience
2016 – present Texas A&M Interfaculty of Reproductive Biology
C. Contribution to Science

1. **Effects of prenatal androgen excess on reproductive neuroendocrine function**: My research efforts focus on elucidating the mechanisms underlying reproductive neuroendocrine perturbations in females prenatally exposed to androgen excess. In conjunction with a team of outstanding researchers, I was able to demonstrate that postnatal endocrine imbalances, namely functional hyperandrogenism and hyperinsulinemia, play a critical role in promoting LH hypersecretion in prenatal testosterone-treated sheep, an animal model of polycystic ovary syndrome (PCOS). We have demonstrated that postnatal treatment with an insulin sensitizer (rosiglitazone) normalizes the GnRH-stimulated LH secretion in this sheep model, a finding that is likely to have important translational relevance to reproductive health in women with PCOS. We are currently using this knowledge to elucidate the epigenetic and transcriptional changes that occur during fetal development that may contribute to the development of LH hypersecretion in female sheep prenatally exposed to androgen excess.


2. **Effects of prenatal androgen excess on metabolic function**: In addition to reproductive perturbations, with the help of a team of collaborators, I documented the effects of prenatal androgen excess on adiposity and insulin resistance in prenatal testosterone-treated sheep. These studies demonstrated that prenatal androgen excess promotes changes in adipocyte size distribution that likely play a role in the development of insulin resistance in these animals. These studies also established the existence of a period of compensatory adaptation during the peripubertal development, when metabolic function is normal in prenatal testosterone-treated sheep. These studies clearly demonstrate the need of longitudinal studies when investigating the long-term effects of perinatal insults. Moreover, these findings suggest the existence of developmental windows in which preventive/treatment strategies may be more effective in preventing the progression of disease.


3. **Effects of perinatal nutrition on pubertal development:** Another important line of research in my laboratory investigates the effects of nutrition during perinatal development on reproductive maturation in females. Obesity during early childhood is associated with increased risk of precocious puberty in girls, which is a risk factor for reproductive cancers and fertility issues during adulthood. Using ruminant females and in collaboration with a strong group of researchers, I have demonstrated that increased nutritional input during the perinatal development promotes functional alterations in hypothalamic neurocircuits that are associated with an advancement in reproductive maturation. Focusing primarily on two leptin-responsive neuronal populations, the POMC/CART and the AgRP/NPY neurons, I have demonstrated that nutritional conditions that promote increased circulating concentrations of leptin result in transcriptional and morphological changes in the hypothalamus that facilitate the achievement of puberty in prepubertal bovine females. We are now investigating the potential interactions between maternal nutrition during gestation and early postnatal adiposity on pubertal development in female ruminants.


**Complete List of Published Work in MyBibliography:**
https://www.ncbi.nlm.nih.gov/sites/myncbi/1D3oxx998r0Qk/bibliography/50656895/public/?sort=date&dir=ascending

**D. Research Support**

**Ongoing Research Support**

<table>
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<tr>
<th>USDA – AFRI 003958</th>
<th>Williams/Amstalden (PI)</th>
<th>09/2013 – 08/2017</th>
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<tr>
<td>Prenatal control of nutritionally-accelerated puberty in heifers</td>
<td>$500,000 (total costs)</td>
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</table>

The major goal of this project is to determine the interactions between maternal nutrition during mid to late gestation and early postnatal nutrition on pubertal maturation in the female bovine. The proposed studies combine whole animal physiology with cellular and molecular approaches to better understand the neuroendocrine mechanisms involved in the nutritional acceleration of puberty.

Role: **Research Investigator**

**Completed Research Support**

<table>
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<tr>
<th>The Lalor Foundation Postdoctoral Fellowship</th>
<th>Cardoso (PI)</th>
<th>06/2015 – 05/2016</th>
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</thead>
<tbody>
<tr>
<td>Pathophysiological role of androgens and insulin in developmental programming of LH hypersecretion in prenatally testosterone-treated females</td>
<td>$50,000 (total costs)</td>
<td></td>
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</tbody>
</table>

The objective of this proposal was to dissect out the role of insulin (hyperinsulinemia) and androgen (functional hyperandrogenism) in promoting LH hypersecretion in a sheep model of PCOS.

Role: **Principal Investigator (Postdoctoral Fellow)**
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: Carney, Ginger Eileen

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

POSITION TITLE:

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.)

<table>
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<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
<th>Completion Date MM/YYYY</th>
<th>FIELD OF STUDY</th>
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<tr>
<td>University of Georgia, Athens, GA</td>
<td>B.S.</td>
<td>06/1991</td>
<td>Genetics</td>
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<tr>
<td>University of Georgia, Athens, GA</td>
<td>Ph.D.</td>
<td>06/1998</td>
<td>Genetics</td>
</tr>
<tr>
<td>Oregon State University, Corvallis, OR</td>
<td>Postdoctoral</td>
<td>07/2002</td>
<td>Behavioral Genetics</td>
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</tbody>
</table>

A. Personal Statement

I have more than 20 years of experience working in the areas of *Drosophila* genetics and molecular biology. I have been an independent investigator for 14 years and most recently have successfully completed projects funded by the NSF and the Norman Hackerman Advanced Research Program. My lab has numerous publications as a result of this funding, which include both undergraduate and graduate student co-authors. My experience as a PI has crystallized the importance of early focus on experimental design and the importance of regular communication with personal in this regard. I also have a realistic expectation of the time it takes to execute experiments, particularly those that require production of new reagents and strains.

My research goal is to understand how behaviors are regulated through the actions of genetic and physiological signals. The relationship between internal physiology and behavioral responses is an important area for study, but we have an incomplete mechanistic understanding of how an animal’s physiology influences its decision making processes. One approach to evaluate this relationship between physiology and behavior is to study reproductive behavior, the output of which is controlled at the level of the central nervous system (CNS) but is guided by an animal’s internal physiology as well as its prior experiences. Central to the interplay between physiology and behavior is crosstalk between adipose and neural tissues. *Drosophila melanogaster*, because of its genetics and the conservation of important neurological and physiological signaling pathways with those of other animals, is an ideal study model for testing hypotheses about how physiology affects reproductive behavior and choice.

In my capacity as a researcher and faculty member, I have extensive experience mentoring undergraduate and graduate students in research projects. Since 2003, I have mentored 37 undergraduates working on independent research projects, 23 of whom are from groups underrepresented in science (including 1 Howard Hughes and 4 summer minority REU students). Three students were Texas A&M (TAMU) Undergraduate Research Scholars (thesis). I have facilitated research presentation opportunities for Carney lab undergraduates and graduate students at local, regional, and national meetings, including presentations at the Annual Biomedical Research Conference for Minority Students (ABRCMS). At least 12 Carney lab undergraduates who earned their degrees enrolled in prestigious Ph.D., MD or MD/Ph.D. programs. Since 2011 my lab has published three peer-reviewed research articles with undergraduate authors; a fourth
article with undergraduate co-authors is in review. Providing research opportunities and mentoring undergraduates is a priority for me, so I accepted a position to serve as the College of Science Associate Dean for Undergraduate Research from 2013-2015. My goals in this capacity were to increase the numbers of students participating in research in the College of Science at Texas A&M University and to provide opportunities for professional growth for these students. I have overseen the college’s NSF Louis Stokes Alliance for Minority Participation (LSAMP) program since 2014 and am a faculty mentor in the LSAMP–Bridge-to-the-Doctorate program.

B. Positions and Honors

Positions
1998-2002 Postdoctoral Fellow, Department of Zoology, Oregon State University
2002-2004 Faculty Research Scientist II, School of Biology, Georgia Institute of Technology
2004-2010 Assistant Professor, Department of Biology, Texas A&M University
2006- Member, Faculty of Neuroscience, Texas A&M University
2010- Member, Faculty of Ecology, Evolution and Behavior, Texas A&M University
2010- Associate Professor, Department of Biology, Texas A&M University
2013-2014 Associate Dean for Undergraduate Research, College of Science (25% effort)
2014-2015 Associate Dean for Undergraduate Research and College Climate (25% effort)
2016- Associate Dean for Assessment and College Climate (25% effort)

Honors and Awards
1987-1991 Georgia Governor’s Scholarship
1987-1991 University of Georgia Alumni Scholarship
1990 Phi Beta Kappa National Honor Society
1991 B.S. Genetics, Magna cum laude, with honors
1992-1995 University of Georgia University-wide Fellowship for pre-doctoral training
1995-1998 NIH National Research Service Award for pre-doctoral training
1999-2002 NIH National Research Service Award for post-doctoral training
2004 Genes and Behavior Gordon Conference Travel Award
2008 Physicians Centre Hospital TAMU Faculty Guest Coach (student nominated)
2008 Center for Teaching Excellence 25th Anniversary W Course Teaching Award
2009 College of Science and Association of Former Students Distinguished Teaching Award
2010 Physicians Centre Hospital TAMU Faculty Guest Coach (student nominated)
2012 Outstanding Service and Leadership Award (TAMU Women’s Faculty Network)
2013-2014 NSF ADVANCE Administrative Fellow, College of Science
2015-2016 Fellow, Southeastern Conference Leadership Development Program

Other Experiences and Professional Memberships
1990-present Phi Beta Kappa National Honor Society
1995-present Genetics Society of America
2009, 2013a,b Member, NSF-IOS Proposal Review Panel
2014 Member, NIH Behavioral Neuroscience Fellowship Panel
2015 Member, NSF Graduate Research Fellowship Program Evaluation Panel
2015 Member, NIH Model Organisms Screening Center US4 Review Panel

C. Contributions to Science

1. My early contributions centered upon understanding the functions of one of the major insect hormones, ecdysone, during Drosophila melanogaster development as well as post-development. Ecdysone activity is mediated by binding to a heterodimeric nuclear receptor comprised of the EcR and Usp proteins. There are 3 isoforms of the EcR receptor, and I used P-element transgenesis to create the first mutations that specifically removed either EcR-A or a combination of the EcR-B1/B2 receptors, revealing previously unknown functions for each receptor. This work demonstrated for the first time that EcR-B1/B2 receptors are important for neuron remodeling during metamorphosis and that EcR-A receptors have vital post-developmental functions in female reproductive behavior. My laboratory extended this work recently with a more in-depth analysis of the
expression patterns and activities of the EcRs in adults of both sexes. (Undergraduate co-authors are indicated by an asterisk *)


2. In a mutant screen I identified logjam (loj), which encodes a p24 trafficking protein essential for egg laying behavior. We found that LOJ is expressed throughout development and localizes to the early secretory pathway. Adult or neural expression of LOJ are each sufficient to rescue oviposition defects of mutant females. This initial finding led us to identify a conserved family of nine p24 proteins in Drosophila, establishing the vinegar fly as a metazoan genetic model for investigating tissue and sex-specific functions for p24 proteins. Our group was the first to study p24 genes in Drosophila and to show that p24s have temporal, spatial and sex-specific distributions and functions within a multi-cellular organism. We hypothesize that particular p24 genes will be absolutely required for some processes (such as behavior or development), while others will have dispensable functions that can be supplied by other genes. Our observations and the reagents (antibodies and overexpression strains) we created provide a starting point for formulating additional testable hypotheses regarding functions of p24 proteins in specific cells and at particular developmental stages.


3. Another major theme in my laboratory has been to determine how the social environment affects gene expression and behavior in Drosophila melanogaster. We were the first to apply transcriptomics approaches to evaluating how animal social experience shapes its gene expression patterns and to link identified loci to specific reproductive behaviors in male and female Drosophila. We found that many socially-responsive genes are expressed in the fly adipose tissue and that adipose-specific knock-out or knockdown of these genes impacts a variety of courtship phenotypes. These findings that adipose tissue is a key player in behavioral performance led us to evaluate dietary effects on behavior. Our most recent work demonstrates that diet is an important factor in determining animal condition and mating behaviors, acting in a sex-specific fashion. These findings provide a new framework for exploring the genetic mechanisms that drive mating behavior, reproductive output, and, hence, trait evolution.


D. Research Support

Completed Research Support

National Science Foundation (IBN-0321473) Carney (PI) 09/01/03-08/31/07
“Characterizing a target locus of a behavioral genetic hierarchy”
The goal of this study was to dissect the role of individual genes in oviposition behavior by focusing on the logjam locus. This gene encodes a molecule with predicted function in intracellular protein trafficking.
Role: PI

Norman Hackerman Advanced Research Program Carney (PI) 05/15/08-12/14/10
“Identifying the neural circuits controlling a complex behavior”
The major goals of this study are to (1) Determine which LOGJAM (LOJ) neurons are required for oviposition, (2) Determine which p24s are required in LOJ cells for oviposition, and (3) Determine if p24s can functionally substitute.
Role: PI

National Science Foundation (IOS#1121517) Carney (PI) 08/15/11-07/31/16
“His fat made him do it: modulation of Drosophila courtship behavior by an adipose-expressed gene product”
The goal of this study is to determine the mechanism by which fat-expressed gene products modulate behavior.
Role: PI
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: Chen, Wei-Jung

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

POSITION TITLE: 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.)

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<td>National Taiwan University (Taiwan, ROC)</td>
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<td>1980-1984</td>
<td>Zoology</td>
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<td>State University of New York at Binghamton</td>
<td>M.A.</td>
<td>1987-1990</td>
<td>Exp. Psychology</td>
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<td>Neuroanatomy</td>
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A. Personal Statement

I received my graduate training in Experimental Psychology and Psychobiology, two research disciplines that emphasize training in experimental design, statistical analysis and complex behavioral theories. I was trained in a behavioral laboratory with an internationally renowned scientist, Dr. Norman Spear, and my training was specifically focused on learning and memory using behavioral paradigms such as latent inhibition and sensory preconditioning. As a faculty member at Texas A&M Health Science Center College of Medicine, I designed and taught a graduate course titled, Experimental Design for Biomedical Science (MSCI 611), and routinely consulted with faculty members across campus with respect to experimental design and data analyses questions. I am currently a member of the Advisory Committee for Research on Women’s Health (ACRWH), National Institutes of Health. Of importance, given my background in experimental design, I understand the limits of interpretation of the experimental findings based on design and data analyses. In addition to my thorough understanding of experimental design and the correct application of statistics to all levels of data (parametric and non-parametric), I have more than 15 years of independent animal research experience involving animal surgery and care, behavior testing, and animal euthanasia, and my service as a member to the Institutional Animal Care and Use Committee (IACUC) at Texas A&M University/Texas A&M Health Science Center over a 5-year period provides me with an unique opportunity to be familiar with the guidelines and practices.

Recent Presentations/Publications on Experimental Design and Statistical Analyses:

“Reproducibility of Research Findings: The Significance (p<0.05) of Well-Designed Experiments and Appropriate Statistical Analyses” Festschrift in Honor of Dr. Norman “Skip” Spear, Binghamton University – SUNY, Binghamton, NY, 2014 (4/30/2014)

“Enhancement of Research Findings: The Significance of Well-Designed Experiments” Methods and Techniques for Integrating the Biological Variable “Sex” in Preclinical Research, Office of Research on Women’s Health (ORWH), National Institutes of Health, Bethesda, MD, 2014 (10/20/2014)

B. Positions and Honors

Positions and Employment

1993-1996  Research Associate, Anatomy & Neurobiology, Texas A&M University, College Station, TX
1996-1999  Assistant Research Scientist, Anatomy & Neurobiology, Texas A&M Univ., College Station, TX
1998     Visiting Assistant Professor, Psychology, Texas A&M University, College Station, TX
1999     Research Assistant Professor, Anatomy & Neurobiology, The Texas A&M University System Health Science Center, College Station, TX
1999-2005  Assistant Professor, Anatomy & Neurobiology, The Texas A&M University System Health Science Center, College Station, TX
2005-2006  Associate Professor, Anatomy & Neurobiology, The Texas A&M University System Health Science Center, College Station, TX
2006-2012  Associate Professor, Neuroscience & Experimental Therapeutics, Texas A&M Health Science Center, College Station, TX
2013     Acting Associate Dean for Academic Affairs, Office of Academic Affairs, Texas A&M University Health Science Center, College of Medicine, Bryan, TX
1999- Faculty of Neuroscience/Texas A&M Institute for Neuroscience, Texas A&M Health Science Center/Texas A&M University
2010-2017  Assistant Dean for Student Affairs, Texas A&M Health Science Center College of Medicine, Temple, TX
2012-  Professor, Department of Neuroscience and Experimental Therapeutics, Texas A&M Health Science Center College of Medicine, Bryan, TX
2014-2017  Associate Dean for Faculty Affairs, Texas A&M Health Science Center College of Medicine, Bryan, TX
2017-  Associate Dean for Student Affairs, Texas A&M Health Science Center College of Medicine, Bryan, TX

Other Experience and Professional Memberships

2004     NIH/NIAAA, ZAA1 DD, Special Emphasis Panel
2004     NIH/NIAAA, ZRG1 1FCN-K, Ethanol, FAS, and Oxidative Stress
2005     NIH/NIAAA, CIFASD Review Committee
2005     NIH/NIDA, ZRG1 BBBP-J 03, Special Emphasis Panel
2005     Research Management Group, Philip Morris External Research Program
2006     Ontario Mental Health Foundation
2006-     Editorial Board Member, Alcohol - An International Biomedical Journal
2006/2007     Institutionally-Based Programs, US Army/Department of Defense
2009     NIH/NIAAA, ZRG1 IFCN-A (58), Special Emphasis Panel/Scientific Review Group
2009     PRMRP09 (Peer Reviewed Medical Research Program), Alcohol/Drug Abuse: Neurobiology-Therapy Panel, US Army/Department of Defense
2011     PRMRP11 (Peer Reviewed Medical Research Program), Drug Abuse Panel, US Army/Department of Defense
2016-2019  Member, Advisory Committee for Research on Women’s Health (ACRWH), National Institutes of Health

Honors

2002     Distinguished Teaching Award, College of Medicine, Texas A&M Health Science Center
2011     R. Kelly Hester Distinguished Teaching Award, College of Medicine, Texas A&M Health Science Center
2016     Elected Member, Gold Humanism Honor Society (GHHS), Arnold P. Gold Foundation.
2016     Distinguished Achievement Award College Level (Teaching), The Association of Former Students, Texas A&M University

C. Contribution to Science

1. Heavy smokers/tobacco users drink more alcohol than non- or light smokers/tobacco users. It has been hypothesized that the pharmacokinetic interaction between alcohol and nicotine may be partially responsible
for the co-use of these two drugs. Our lab demonstrated that the presence of nicotine increase alcohol metabolism, thereby decreasing the circulating alcohol in the physiological system which subsequently leads to a decrease in the expected alcohol effect, a phenomenon similar to “tolerance.” Interestingly, nicotine decreases blood alcohol concentration only when alcohol is administrated orally (intragastric intubation). Conversely, if alcohol is administered by intraperitoneal injection bypassing the first pass metabolism via gastric alcohol dehydrogenase (gADH), the presence of nicotine is unable to decrease the blood alcohol concentration. Clinically, nicotine-induced decreases in blood alcohol concentration may potentially promote more alcohol drinking to achieve the “expected effects.” This excessive alcohol drinking among smokers will jeopardize the health of these individuals, since more acetaldehyde, the primary alcohol metabolite which is highly toxic to the organ tissues, will be generated. I served as a PI or Co-I for these studies.

2. Offspring born with fetal alcohol syndrome (FAS) and fetal alcohol spectrum disorder (FASD) commonly exhibit a constellation of birth defects, including various degree of brain injuries. In order to develop interventions to at least reduce the severity of the damaging effects on the developing brain, it is critical to identify the risk factors that may potentially influence the severity of brain injury associated with FAS/FASD. Our lab has conducted various studies to identify whether blood alcohol concentration, polydrug use and timing of alcohol exposure are potential risk factors for developmental alcohol-induced brain damages. In addition, it has recently hypothesized that miRNA may play a role in mediating developmental alcohol induced damage. Our lab and the collaborators have shown that miRNA, miR-9, is involved in mediating teratogenic effects of alcohol. Identifying these risk factors and mechanisms is an important step to lessen the damaging effect of developmental alcohol on developing brains.

3. One of the most commonly proposed underlying mechanisms for developmental alcohol-induced brain injury is oxidative stress. Therefore, it is hypothesized that developmental alcohol exposure will lead to an increase in oxidative stress and the application of anti-oxidant is likely to reverse or ameliorate the severity of developmental alcohol-induced brain injury. Our lab found that developmental alcohol exposure leads to an increase in oxidative stress; however, various anti-oxidants failed to attenuate the damaging effects of developmental alcohol exposure. Based on these findings, it is further hypothesized that the timing of the application of anti-oxidants may be important to exert their therapeutic effects to lesson developmental alcohol-induced brain injury.

\[\text{References:}\]


\[\text{References:}\]


D. Research Support

Ongoing Research Support

RO1 AG041360 William Griffith (PI) 4/15/12-3/31/17
Estrogen, ovarian aging and calcium channel modulation
This project is to use an ovarian aging rat model system to test the non-genomic estrogenic mechanisms that control Ca++ and synaptic functions.
Role: Co-I

RO1 AA024659 Rajesh C. Miranda (PI) 3/10/16-2/28/21
Prenatal MicroRNA Neuro-Therapeutics for Fetal Alcohol Exposure
This project is to investigate the role of microRNA in its neuro-therapeutic potential/property in subjects prenatally exposed to alcohol.
Role: Co-I

Completed (in the last 5 years):

RO1 AA13440 Rajesh C. Miranda (PI) 7/1/08-6/30/13
Fetal Alcohol Exposure and Neurodevelopment
This project is to identify the role of miRNA in mediating the toxicity associated with alcohol exposure during development.
Role: Co-I

U0 AA017120-1 Timothy Cudd (PI) 9/1/07-8/31/12
Translational Studies of FASD Using a Sheep Model
This project is to test the potential therapeutic actions of choline on developmental alcohol mediated effects using a sheep model system.
Role: Co-I

5RO1 AA010940 Timothy Cudd (PI) 8/1/08-7/31/13
Ovine Model System for Alcohol Related Birth Defects
This project employs a sheep model to investigate the underlying mechanisms responsible for mediating the wide spectrum of alcohol-induced teratogenic effects.
Role: Co-I
BIOGRAPHICAL SKETCH
Provide the following information for the key personnel and other significant contributors in the order listed on Form Page 2.
Follow this format for each person. DO NOT EXCEED FOUR PAGES.

NAME
Choe, Yoonsuck

POSITION TITLE
Professor

eRA COMMONS USER NAME
YOONCHOE

EDUCATION/TRAINING (Begin with baccalaureate or other initial professional education, such as nursing, and include postdoctoral training.)

<table>
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<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
<th>YEAR(s)</th>
<th>FIELD OF STUDY</th>
</tr>
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<tbody>
<tr>
<td>Yonsei University (Seoul, Korea)</td>
<td>B.S.</td>
<td>1993</td>
<td>Computer Science</td>
</tr>
<tr>
<td>The University of Texas at Austin</td>
<td>M.A.</td>
<td>1995</td>
<td>Computer Sciences</td>
</tr>
<tr>
<td>The University of Texas at Austin</td>
<td>Ph.D.</td>
<td>2001</td>
<td>Computer Sciences</td>
</tr>
</tbody>
</table>

A Personal Statement
I am leading an active research group working on computational neuroscience, imaging, and neuroinformatics, generously funded by National Institutes of Health and National Science Foundation, with one active grant at the moment. I have also advised many undergraduate and graduate students who went on to earn advanced degrees and secure a position in the academia. I am confident that my continuing efforts will contribute greatly to the proposed “Bridges” program.

B Positions and Honors

Positions and Employment
2014– Professor, Department of Computer Science and Engineering, Texas A&M University
2007–2014 Associate Professor, Department of Computer Science and Engineering, Texas A&M University
2005– Director, Brain Networks Laboratory (Texas A&M CS)
2001–2007 Assistant Professor, Department of Computer Science, Texas A&M University
1999 Intern, HNC Software Inc., San Diego, CA (DARPA CVIM Project)

Honors
2013 30th Anniversary Distinguished Alumni Award (one of 6 recipients), Department of Computer Science, Yonsei University, Seoul, Korea
2013 Charles H. Barclay, Jr. 45 Fellow (College of Engineering Faculty Fellow), Texas A&M University
2012 Graduate Faculty Teaching Excellence Award, Department of Computer Science, Texas A&M University
2010 President, Korean Computer Scientists and Engineers Association in America (KOCSEA)
2009 Co-author on best student paper award, IEEE CIMSVP (C. Park and Y. H. Bai)
2008 Best scientific paper award, International Conference on Pattern Recognition (with Y. H. Bai and C. Park)
2006 Big 12 Faculty Fellowship Award
2004 Graduate Faculty Teaching Excellence Award, Department of Computer Science, Texas A&M University
1999 Schlumberger graduate fellowship

C Selected Peer-Reviewed Publications (in chronological order)
Selected from over 100 peer-reviewed publications

C.1 Most Relevant Publications
The “*” mark indicates students supervised by Yoonsuck Choe.


C.2 Other relevant publications

The “∗” mark indicates students supervised by Yoonsuck Choe.


D Research Support

All projects are peer reviewed.


   The goal of this project is to design and develop an enhanced version of the Knife-Edge Scanning Microscope (KESM) for increased robustness and higher-resolution imaging and fluorescence imaging capabilities. Choe is the PI of the project, overseeing all aspects of the project.

2. **NSF: Completed** Principal investigator for the project *CRCNS: Data Sharing: Open Web Atlas for High-Resolution 3D Mouse Brain Data*, #1208174, 9/1/2012–8/31/2014.

   The goal of this project is to extend the web-based Knife-Edge Scanning Microscope (KESM) Brain Atlas developed in prior project (below). Transition from a closed-source base to an open-source API, use of vector graphics, and Web-GL-based in-browser volume viewer are major improvements. The project also includes mapping to external atlases such as the Allen Brain Atlas. Choe is the PI of the project, overseeing all aspects of the project.


   The goal of this project was to develop a web-based, light-weight brain atlas to serve the data from the Knife-Edge Scanning Microscope. We successfully deployed the atlas online at http://kesm.org, serving three whole-brain-scale mouse brain data sets (~1 um resolution).
NAME: W. Les Dees

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

POSITION TITLE: Senior 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.)

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<th>INSTITUTION AND LOCATION</th>
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<tr>
<td>Texas A&amp;M University, College Station, TX</td>
<td>BS</td>
<td>1971</td>
<td>Animal Science</td>
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<tr>
<td>Texas A&amp;M University, College Station, TX</td>
<td>BS</td>
<td>1972</td>
<td>Biomedical Science</td>
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<tr>
<td>Texas A&amp;M University, College Station, TX</td>
<td>MS</td>
<td>1979</td>
<td>Reproductive Physiol</td>
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<tr>
<td>Texas A&amp;M University, College Station, TX</td>
<td>PhD</td>
<td>1982</td>
<td>Veterinary Anatomy</td>
</tr>
<tr>
<td>U. of TX Southwestern Med. Ctr, Dallas, TX</td>
<td>Postdoc</td>
<td>1984</td>
<td>Neuroendocrinology</td>
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</table>

A. Personal Statement
The main focus of my research has been to study the effects of substances that control or alter the onset of female puberty. I have been funded as PI by the NIH for over 30 years. My research focuses on identifying the role(s) of specific genes, peptides, growth factors and excitatory amino acids on the hypothalamic control of the timing of puberty. Other studies assess the effects of exposure to toxins including manganese, lead and alcohol on the actions and interactions of the above mentioned puberty-related substances. Anatomical, physiological and molecular approaches are used to assess hypothalamic-pituitary-ovarian function using prepubertal rodents and rhesus monkeys as animal models.

B. Positions and Honors
1985 Asst. Instructor, Physiology, Univ. of TX Southwestern Med. Center, Dallas
1985-1986 Research Asst. Professor, Physiology (Division of Neuropeptides), as above
1986-1991 Assistant Professor, Veterinary Integrative Biosciences, Texas A&M University
1989-present Member, Faculty of Toxicology, as above
1990-present Member, Faculty of Neuroscience, as above
2000-present Member, Faculty of Reproductive Biology, as above
1992-1997 Associate Professor, as above
1998-2016 Professor, as above
2016-present Senior Professor

NIH Research Scientist Development Award, 1987-92 and 1993-98
Carrington Laboratories Research Award, 1989
Pfizer Research Award, 1996
TAMU Faculty Distinguished Achievement in Research, 1996
TAMU, College of Veterinary Medicine Research Leadership Award, 2013
C. Contribution to Science.

My early research interest was in general reproductive neuroendocrinology, but soon began to focus more specifically on assessing the neuroendocrine events and factors that control or alter the onset of puberty. Research from my laboratory has allowed us to make contributions in basic puberty research, as well as assessing the effects of alcohol (ALC), manganese and other toxic substances on puberty-related events. Below I have chosen four areas demonstrating significant contributions to science. The first three represent some of the basic and ALC research contributions throughout the long-term progression of a puberty research project funded by NIH/NIAAA. The fourth shows contributions from another research project funded by NIH/NIEHS.

1. Effects of ALC on Female Puberty:

Interest turned from studying effects of ALC on pituitary hormone secretion in mature females to studying immature females as a result of the delayed puberty noted in the younger animals in a preliminary study. Early work by others indicated that ALC could delay puberty but very little was known of the pituitary hormones affected and nothing was known as to whether the ALC effect was due to a hypothalamic or a pituitary insult. Our initial studies, in prepubertal animals, defined the ALC-induced changes and differential responses noted in secretion of key pituitary hormones, then later demonstrated these changes correlated with differential responses of specific hypothalamic releasing and inhibiting peptides. Importantly, our studies were the first to show that ALC acts at the hypothalamic level to suppress luteinizing hormone-releasing hormone (LHRH) secretion causing delayed vaginal opening in rats, and delayed development of a normal menstrual cycle in rhesus monkeys. This research was significant in our attaining a better understanding of the adverse effects of ALC at puberty and instrumental for further advancement of the field (also see below). Selected representative articles below demonstrate these contributions. I served as the principal investigator/project director for each of these studies.

Dees, WL and CW Skelley. The effects of ethanol during the onset of female puberty. Neuroendocrinology 51:64-69, 1990. PMID2106089


Dissen, GA, RK Dearth, HM Scott, SR Ojeda, and WL Dees. Alcohol ingestion inhibits the increased secretion of puberty-related hormones in the developing female rhesus monkey. Endocrinology 141:1325-1331, 2000. PMID10746635

2. Role of IGF-1 at Puberty and the Effects of ALC on IGF-1:

The onset of puberty is complex and it became apparent early on that more research was needed to identify critical basic factors capable of stimulating prepubertal LHRH release in order to help advance the puberty field and, as a result, advance our knowledge with regard to the effects of ALC on this important stage of development. Insulin-like growth factor-1 (IGF-1) seemed to be a prime candidate for such a role, but no information was available in this regard. Our basic research was the first to demonstrate IGF-1 induces LHRH release, and later showed that IGF-1 administration to prepubertal rats can accelerate the onset of female puberty, and that estradiol plays an important role. We also have published articles among which demonstrated that ALC alters the peripheral synthesis of the peptide, its hypothalamic effect to block IGF-1 stimulated prepubertal LHRH release, and its ability to interfere with glial-neuronal interactions contributing to the LHRH secretion. The basic science IGF-1 research stated above was a very important contribution to the puberty field and has resulted in the expansion of research assessing the hypothalamic actions of IGF-1 at puberty, and after maturity, by other laboratories. This research on prepubertal ALC and IGF-1, including glial/neuronal actions and interactions, contributed to the overall understanding of how ALC effects puberty, and continues to provide important information in this regard. Selected representative articles below demonstrate these contributions. I served as the principal investigator/project director for each of these studies.


3. The effects of IGF-1 and ALC on KiSS-1 Influences at Puberty:

Kisspeptin, a protein product of the KiSS-1 gene, is important for LHRH secretion at puberty in all species, including humans. Because IGF-1 is considered one of the components contributing to early signaling processes controlling LHRH release and puberty, we assessed its ability to regulate the KiSS-1 gene. We were the first to show that IGF-1 is capable of inducing KiSS-1 in immature animals. Subsequently, we demonstrated the pathway of this action and then the ability of ALC to block this effect. We also demonstrated that chronic ALC administration causes suppressed KiSS-1 gene expression in prepubertal females and is associated with delayed puberty. These demonstrations that IGF-1 is an upstream stimulator of the KiSS-1 gene, and that ALC can suppress this gene were both novel, and provided the initial insights into the regulation and pathways controlling a gene that is critical for the onset of puberty. This research has resulted in expanding basic and ALC related IGF-1/KiSS-1 studies in this and now other laboratories. Additionally, this area of research applies directly to adolescent health with regard to critical substances controlling the timing of puberty and for their potential use in interventions. Selected representative articles below demonstrate these contributions. I served as the principal investigator/project director for each of these studies.
Manganese and Precocious Pubertal Development:

Manganese (Mn) is an essential element that is beneficial and needed for normal development, but exposure to higher levels can be toxic. Because Mn depletion has been associated with reproductive dysfunction we wanted to discern if low levels would affect pubertal development. Our research demonstrated that prepubertal rats exposed to Mn exhibited precocious pubertal development and this was associated with elevated serum levels of gonadotropins and estradiol. Importantly, we showed that Mn acts at the hypothalamic level to stimulate prepubertal LHRH secretion in order to drive the pubertal process. Subsequently, we have determined that Mn is capable of up-regulating both IGF-1 and KiSS-1, two critical regulators in the reproductive hypothalamus associated with the initiation of puberty. This is novel and indicates an environmental substance that can initiate precocious puberty by acting through normal neuroendocrine events, just too early. Interestingly, the same genes and hormones that are suppressed by ALC are induced by Mn. These results indicate that Mn may be involved in normal puberty, but also suggest that it may contribute to precocious puberty if an individual is exposed to moderately elevated levels too early in life. This research has health related potential and gives researchers another tool with which assess activation of pubertal processes, and to study precocious puberty, a serious endocrine problem for which the cause is unknown in over 95% of the cases in females. Selected representative articles below demonstrate these contributions. I served as the principal investigator/project director for each of these studies.


Srivastava VK, JK Hiney and WL Dees. Manganese stimulated kisspeptin is mediated by the IGF-1/Akt/mTOR pathway. Endocrinology, 157:3233-3241, 2016. PMID27309941
D. Research Support.

Ongoing projects:

NIH/NIEHS
“Actions of Manganese on Neuroendocrine Development”

The overall objective is to assess central mechanisms of action of low level manganese exposure to induce precocious female pubertal development.

R01-AA07216  Dees (PI)  June. 1, 2015-May 31, 2020
NIH/NIAAA
“Neuroendocrine Effects of Alcohol on Puberty”

The overall goals are to assess the involvement of specific puberty-related genes, growth factors and peptides on female puberty, and to determine actions and interactions of alcohol on the molecular and physiological roles of these substances as they relate to the pubertal process.
NAME: Sharon DeMorrow

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

POSITION TITLE: Associate 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.)

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<td>Bsc (Hon I)</td>
<td>11/94</td>
<td>Biochemistry</td>
</tr>
<tr>
<td>University of Queensland, Brisbane, Australia</td>
<td>PhD</td>
<td>06/99</td>
<td>Biochemistry</td>
</tr>
<tr>
<td>Max Planck Institute of Psychiatry, Munich Germany</td>
<td>Postdoctoral fellow</td>
<td>10/02</td>
<td>Neuroscience</td>
</tr>
<tr>
<td>Johannes Gutenberg University, Mainz, Germany</td>
<td>Postdoctoral fellow</td>
<td>12/04</td>
<td>Neuroscience</td>
</tr>
<tr>
<td>Scott and White Memorial Hospital, Temple, TX</td>
<td>Postdoctoral fellow</td>
<td>06/07</td>
<td>Digestive Diseases</td>
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A. Personal Statement

The long-term goal of my research program is to identify signaling molecules that are released during acute and chronic liver diseases that may contribute to the non-hepatic consequences of liver failure, such as hepatic encephalopathy. To this end, we have demonstrated that transforming growth factor β (TGFβ) is expressed in hepatocytes and released into the blood stream in a rodent model of acute liver failure and contributes to aspects of hepatic encephalopathy, including permeability of the blood brain barrier, and cognitive impairment. This proposal investigates the downstream consequences of aberrant TGFβ signaling in drug-induced liver injury, specifically the effects of liver-derived TGFβ on peripheral and central IGF1 signaling. We have preliminary data indicating that the TGFβ-mediated decrease in circulating IGF1 levels observed during acute liver failure is not due to a decrease in IGF1 expression, but rather is a result of increased IGF1 degradation. In contrast, TGFβ directly down-regulated IGF1 expression in neurons via a mechanism involving microRNA Let-7f during hepatic encephalopathy. Our working hypothesis is that during acute liver failure, aberrant hepatic TGFβ signaling suppresses both the circulating and central bioavailability of IGF1 via differential mechanisms, and that strategies to restore IGF1 levels will prove to be both hepatoprotective and neuroprotective.

I have the experience, skills and experimental tools available to investigate this important area of hepatotoxicity and neuropathophysiology associated with liver damage. I have a strong background in the neuroscience field and the current proposal perfectly merges my prior neuroscience-related experience and skills with more recent experience in the pathophysiology of liver diseases. Dissecting the consequences of aberrant TGFβ signaling in acute liver failure and its subsequent involvement in hepatic encephalopathy may lead to an enhanced understanding of the pathological processes and consequences of this particular type of liver injury. This knowledge may play a paramount role in the development of novel treatment paradigms for managing the consequences of acute drug-induced liver injury.

B. Positions and Honors

Positions and Employment

1994-1999  Graduate Research Assistant, Australian Postgraduate Research Award (APRA) recipient (Graduate scholarship)

Jan 2000-Aug 2002  Postdoctoral position in the Independent Research Group of Neurodegeneration at the Max Planck Institute of Psychiatry, Munich Germany (Group leader: Dr. Christian Behl)

Sep 2002-Dec 2004  Postdoctoral position at the Department of Pathobiochemistry, Johannes Gutenberg University, Mainz, Germany (Group Leader: Prof Christian Behl)
May 2005-June 2007 Postdoctoral fellow at the Division of Research and Education, Scott & White Memorial Hospital, Temple TX, USA (Group Leader: Prof. Gianfranco Alpini)

June 2007-Aug 2008 Assistant Professor, (non-tenure track) Department of Internal Medicine, Division of Research and Education, Texas A&M Health Science Center and Scott & White Hospital, Temple TX.

Aug 2008 – Aug 2013 Assistant Professor (tenure track), Department of Internal Medicine, Texas A&M Health Science Center and Scott & White Hospital, Temple TX.

Jan 2009 – present Faculty member, Scott & White Digestive Disease Research Center, Scott & White Hospital, Temple Texas

Aug 2013 – present Associate Professor (with tenure), Department of Internal Medicine, Texas A&M Health Science Center and Scott & White Hospital, Temple TX.

Other Experience and Professional Memberships

Membership:
Current Member – American Association for the Study of Liver Disease
Current Member – American Association for Cancer Research
Current Member – American Society of Cell Biology
Current Member for the Society for Neuroscience.
Current Member of the International Society for Neurochemistry
Current Member of the American Society for Biochemistry and Molecular Biology
Current Member of the American Physiological Society
Current Member of the American Society of Integrative Pathology
Member of the American Physiological Society’s sub committee for Animal care and experimentation. January 2012 – December 2014

Editorial board memberships
Assistant Editor  World journal of Gastrointestinal pathophysiology (Jan 2010 – present)
Associate Editor  BMC Research Notes (May 2010 – present)
Editorial board  Research Journal of Medical Sciences (January 2007-present)
Editorial board  World Journal of Gastroenterology (February 2007 – present)
Editorial board  Digestive and Liver diseases (January 2010-present)
Faculty Member for 'Liver Biology & Pathobiology' Section, Gastroenterology and Hepatology Faculty of the FACULTY of 1000 MEDICINE (January 2010 - present)

Honors
1994-1999 Australian Postgraduate Research Award (APRA) recipient (PhD scholarship)
1997 Scholarship for participation in the Third Advanced School of Neurochemistry on "Frontier Approaches to Brain Function and Disease"
2000-2002 Max Planck Society Stipendium
2017 Recipient, Texas A&M HSC College of Medicine, Faculty development award

C. Contribution to Science
1. Excitotoxic cell death in hippocampal neurons. My early publications directly assessed the molecular mechanisms associated with excitotoxic cell death in the hippocampus. These publications involved identifying and assessing potential neuroprotective pathways in the kainic acid model of temporal lobe epilepsy. Specifically, we focused on the protective effects of estrogen and the endocannabinoid system and demonstrated that firstly, estrogen protects against the oxidative cell death associated with excitotoxicity via the phosphorylation and inactivation of glycogen synthase kinase 3β. Secondly, we demonstrated the novel finding that the endocannabinoid receptor Cb1 is essential as a dampening mechanism against excessive excitotoxic stimuli. Mice lacking the Cb1 receptor had excessive seizure activity in response to low doses of kainic acid compared to wild type mice, thereby identifying Cb1 as integral to the on-demand protection against acute excitotoxicity in the central nervous system – an observation that was published in Science.


2. Progranulin regulation of hyperplastic and neoplastic cholangiocyte proliferation. Progranulin (PGRN) mediates cell cycle progression, cell motility, and inflammatory processes in a number of cell types. Structurally, it belongs to none of the well-established growth factor families. To date, no unique receptor for PGRN has been identified. My lab demonstrated that the expression and secretion of PGRN is increased in cholangiocarcinoma cell lines and human biopsy samples compared to their non-malignant counterparts. In parallel, increased PGRN levels could be detected in the serum (but not bile) from patients with cholangiocarcinoma compared to non-malignant controls. We demonstrated that the increased PGRN expression in cholangiocarcinoma cells is driven by the IL-6-mediated activation of the ERK1/2/RSK1/C/EBPβ pathway. Increased PGRN secretion exerts subsequent growth-promoting effects on cholangiocarcinoma cells via the activation of Akt and subsequent phosphorylation and nuclear extrusion of Forkhead box protein O1 (FOXO1). These data suggest that the upregulation of PGRN may be a key feature associated with the progression of cholangiocarcinoma; inhibiting PGRN expression or function may be a viable target for the development of an effective adjunct therapy to treat this deadly disease.

Interestingly, we have also demonstrated that PGRN expression and secretion are increased during hyperplastic cholangiocyte proliferation in a rodent model of extrahepatic biliary obstruction. Furthermore, PGRN exerted growth-promoting effects on cholangiocytes via the nuclear extrusion and inhibition of FOXO1, though the mechanism by which this occurred is different. Specifically, in cholangiocarcinoma, FOXO1 was extruded after phosphorylation via a mechanism involving Akt signaling pathway, whereas in hyperplastic cholangiocyte proliferation, PGRN inhibited the expression of the deacetylase Sirt1 and subsequently increased acetylation of FOXO1, which also results in nuclear extrusion. This discrepancy may be due to the increased reliance on Akt signaling by cholangiocarcinoma compared to their non-malignant counterparts.

• G. Frampton, P. Invernizzi, F. Bernuzzi, H Pae, D Horvat, C. Galindo, M. Quinn, L. Huang, M. McMillin, L. Rimassa, S DeMorrow*. (2012) Interleukin-6- driven progranulin expression increases cholangiocarcinoma growth by an Akt dependent mechanism. Gut. 61(2):268-77. PMID: 22068162


3. Autocrine factors regulating cholangiocarcinoma growth. Cholangiocarcinoma is a devastating cancer that exhibits poor prognosis and surgical resection is virtually the only measure for a curative treatment. Other attempts, including radiotherapy and photodynamic therapy, to relieve biliary obstruction due to non-resectable tumors have been demonstrated successfully as an adjuvant therapy following surgery or as palliative therapy. I believe that by dissecting the autocrine and paracrine pathways that regulate cellular growth and migration, we will identify possible target genes for therapies designed to slow the rate of cholangiocarcinoma growth. Specifically, cholangiocarcinoma cells produce increased levels of these biogenic amines, which can be detected in the bile of cholangiocarcinoma patients. This increased production is due to the increased expression of the synthesis enzymes and a marked suppression of the degradation enzyme monoamine oxidase A (MAO-A). We have shown that the expression of MAO-A correlates with tumor progression and overall survival, with patients exhibiting lower levels of MAO-A exhibiting a higher incidence of metastasis and poorer prognosis. In addition, we have recently shown that cholangiocarcinoma express high levels of the novel growth factor progranulin as a result of aberrant interleukin-6 signaling, which has subsequent growth-promoting effects.
4. Neurological changes associated with acute and chronic liver failure. We have shown that during cholestatic liver disease, the hypothalamic-pituitary-adrenal axis is suppressed leading to decreased circulating glucocorticoid levels. We believe that the suppression of the HPA axis is probably as a result of increased circulating bile acids observed during liver damage. These bile acids cause a leakiness to the blood-brain-barrier and then enter the brain to bring about the neurological changes observed. This has opened up a very novel field of research in which bile acids should be viewed as neuroendocrine hormones rather than mere detergents that aid in fat digestion. Furthermore, in models of acute liver failure we have demonstrated a role for inflammatory molecules TGFβ1 and CCL2 in the neurological decline associated with hepatic encephalopathy.

- M. McMillin, G. Frampton, M Quinn, A Divan, S, Grant, N Patel, K Newell-Rogers, S. DeMorrow (2015). Suppression of the HPA axis during cholestasis can be attributed to hypothalamic bile acid signaling. Mol. Endocrinology 29(12); 1720-39. PMID: 26341088

Complete List of Published Work in MyBibliography:

D. Research Support
Ongoing:
Title: Dysregulated Hypothalamic Neuropeptides During Biliary Hyperplasia
Funding agency: NIH-NIDDK (5R01DK082435-02)
Dates of grant period: 04/01/2010-07/31/2021
Role on grant: Principal Investigator

The goal of this grant is to evaluate the effects of extrahepatic biliary obstruction on the activity of the hypothalamic neuropeptides and to determine the mechanism by which this occurs. We propose the novel central hypothesis that early release of bile acids into the serum as a result of cholestasis results in the suppression of ghrelin expression from the stomach and increased production of leptin from adipose tissue. This imbalance results in the subsequent suppression of NPY and increase in amelanocyte stimulating hormone (αMSH) expression in the hypothalamus, both of which are exacerbated by hypothalamic bile acid signaling. These peripheral and central changes in neuropeptide expression co-ordinately regulate cholangiocyte proliferation and biliary fibrosis.

Title: The role of hypothalamic neuropeptides on biliary function during cholestasis
Funding agency: VA Merit review
Dates of grant period: 10/1/2014- 9/30/2018
Role on grant: Principal investigator
The objective of this grant is to investigate mechanisms by which extrahepatic biliary obstruction regulates the expression of the hypothalamic peptide galanin and the subsequent effects on cholangiocyte proliferation. Our working hypothesis is that liver damage results in early release of bile acids into the serum leading to increased bile acid content in the brain, thereby allowing bile acids to alter the expression of galanin in the hypothalamus. The activation of central and peripheral galanin-mediated events during cholestasis may be partly responsible for the stimulation of cholangiocyte proliferation.

Title: Analysis of gene expression changes in the brain of mouse models of acute liver failure.
Funding agency: Temple Health and Biosciences District
Dates of grant period: 12/01/2016-11/30/2017
Role on grant: Principal investigator
The overall objective of this proposal is to compare and contrast the changes in gene expression in 3 key brain regions during drug-induced liver injury using 2 mouse models of acute liver failure, one that develops overt hepatic encephalopathy and the other that does not. We will focus on 2 main areas of interest 1) Bile acid signaling and downstream consequences to include neurological cholesterol homeostasis and cerebral energy metabolism, 2) TGFβ1-signaling and TGFβ1-mediated inflammatory pathways.

Title: Pathogenic role of thrombospondin-1 in acute and chronic liver failure
Funding agency: Department of Veteran Affairs Career Development Award-2
Dates of grant period: 10/01/16-09/30/21
Role on grant: Mentor (Matthew McMillin, PI)
The goal of this research proposal is to determine the consequences of thrombospondin-1 on TGFβ1 activation and the subsequent effects during acute and chronic liver failure.

Completed:
Title: Biogenic amines regulate cholangiocarcinoma cell growth
Funding agency: American Cancer Society (RSC118760)
Dates of grant period: 7/01/2010 to 12/30/2014
Role on grant: Principal investigator
We have previously shown that cholangiocarcinoma cells produce increased amounts of serotonin and dopamine. The goal of this grant is to evaluate the mechanisms by which this occurs and to further evaluate the consequences of serotonin and dopamine on cholangiocarcinoma growth and progression.

Title: The role of hedgehog signaling in hepatic encephalopathy
Funding agency: Intramural funding – Scott & White Research grants program
Dates of grant period: (08/01/2012--07/31/2014)
Role on grant: Principal Investigator
The goal of this grant is to evaluate the interplay between the hedgehog ligands (sonic and indian hedgehog) and TGFβ that are released from the liver during acute liver failure, and may exert various effects on the brain during hepatic encephalopathy.
Scott V. Dindot

Position Title
Associate Professor

EDUCATION/TRAINING

<table>
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<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
<th>YEAR(s)</th>
<th>FIELD OF STUDY</th>
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<tr>
<td>Texas A&amp;M University, College Station, TX</td>
<td>B.S.</td>
<td>1999</td>
<td>Molecular and Cell Biology</td>
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<tr>
<td>Texas A&amp;M University, College Station, TX</td>
<td>Ph.D.</td>
<td>2003</td>
<td>Genetics</td>
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<tr>
<td>Baylor College of Medicine, Houston, TX</td>
<td>Postdoctorate</td>
<td>2008</td>
<td>Molecular and Human Genetics</td>
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A. PERSONAL STATEMENT

The overarching themes of research in my laboratory are to understand the genetic and epigenetic mechanisms underlying disease, with a particular interest in understanding how epigenetic modifications affect the penetrance and expressivity of pathological states. My lab is also exploring ways to manipulate epigenetic modifications as a therapeutic approach. My role in this project will be to facilitate the experimental analysis of the key epigenetic modifications associated with different transcriptional states.

B. POSITIONS AND HONORS

Positions
2003 - 2008 Postdoctoral Research Fellow, Department of Molecular and Human Genetics, Baylor College Medicine, Houston, TX.
2003 - 2007 Adjunct Assistant Professor, Department of Biological Sciences, Lone Star College, Kingwood, TX.
2008 - 2014 Assistant Professor (Tenure Track), Department of Pathobiology, College of Veterinary Medicine & Biomedical Sciences, Texas A&M University, College Station, TX.
2010 - 2012 Applications Researcher, Institute for Applied Mathematics and Computational Science, Texas A&M University, College Station, TX.
2011 Assistant Professor (Joint appointment), Department of Molecular and Cellular Medicine, College of Medicine, Texas A&M Health Science Center, College Station, TX.
2014 - Associate Professor with Tenure, Department of Pathobiology, College of Veterinary Medicine & Biomedical Sciences, Texas A&M University, College Station, TX.

Other Experience, Committees and Professional Membership
Scientific Advisory Committee, Texas A&M Institute for Genomic Medicine, 2009
Texas Genetics Society, 2009 - present
CVM Grant Review, College of Veterinary Medicine & Biomedical Sciences, Texas A&M University, 2010 - 2011
Faculty of Genetics, Texas A&M University, 2009 - present
Faculty of Neuroscience, Texas A&M University, 2010 - present
Graduate Advisory Committee, Veterinary Pathobiology, 2010 - present
Morris Animal Foundation/Cat Health Network Study Section, 2010
Faculty of Reproductive Biology, Texas A&M University, 2011 - present
Reviewer, Foundation for Angelman Syndrome Therapeutics Seed Grants, 2013 - present
Study Section, National Institute of Environmental Health Sciences, Children’s Environmental Health and Disease Prevention Research Center Study Section, 2012
Study Section, U.S. Department of Agriculture-Agriculture and Food Research Initiative Panel Member: Animal Breeding, Genetics and Genomics
Reviewer, Cancer Research Council, Texas A&M University, 2011
Century Scholars Mentor, Texas A&M University, 2013 - present
Texas A&M University Neuroscience Institute Seminar Series Committee, 2012
Study Section, National Institute of Environmental Health Sciences, Environmental Health Sciences Review Committee Core Centers Program, 2013
Advisory Board, Texas A&M University, Rodent Behavioral Core, 2013 - present
Dup15q Alliance Professional Advisory Board, 2013 – present
SFARI Investigator, 2013 - present
Study Section, National Institute of Environmental Health Sciences, Environmental Health Sciences Review Committee Core Centers Program, 2015

Honors and Awards
NICHDD NRSA (T32).  Department of Molecular and Human Genetics, Baylor College of Medicine, Houston, TX, 2003
NIH Pediatric Loan Repayment Grant, 2004
NINDS NRSA (T32).  Cain Pediatric Neurology Research Foundation Laboratories, Baylor College of Medicine, Houston, Texas. (Declined), 2004
NICHDD NRSA (F32). “Analysis of genomic imprinting at the Ube3a locus”, 2004 - 2006
Outstanding Scientific Achievement Award. College of Veterinary Medicine & Biomedical Sciences, Texas A&M University, 2013

C. CONTRIBUTIONS TO SCIENCE

1. Genomic imprinting is an epigenetic phenomenon that results in the differential allelic expression of certain genes in a parent-of-origin dependent manner. Imprinted gene expression is governed by different epigenetic modifications that are imparted onto the parental chromosomes during gametogenesis and stably maintained during development. This paper was one of the first genome-wide studies (ChIP-chip) to examine the epigenetic properties of imprinted genes. This study also lead to the identification of novel regulatory elements associated with imprinted gene expression.


2. The ubiquitin-protein E3A ligase (UBE3A) gene is imprinted with maternal-specific expression in neurons and biallelically expressed in all other cell types. Loss-of-function mutations of the maternally inherited UBE3A allele cause Angelman syndrome, a severe neurodevelopmental disorder that is associated with intellectual disability, ataxia, absent speech, epilepsy, and an atypical 'happy' disposition. This paper describes the development of a mouse model engineered with a fluorescent reporter (Ube3aYFP mouse model) that was used to examine the allelic expression patterns of Ube3a in the brain, providing for the first time the ability to visualize the allelic expression pattern of a gene in a single cell. This mouse model has also been widely used
by other academic and pharmaceutical laboratories to investigate the Ube3a gene and identify new therapies to treat Angelman syndrome. Furthermore, this paper was the first to show that loss of Ube3a leads to specific abnormalities in synaptic development, which is one of the only known morphological defects observed in Angelman syndrome.


3. The generation of live-born animals through somatic cell nuclear transfer was seen as one of the most significant technological advancements in the field of reproductive biology in the 20th century; however, it was soon realized that animals generated through SCNT were often born with developmental defects consistent with several imprinted disorders (e.g., Beckwith-Wiedemann syndrome). These papers were among the first studies to demonstrate that SCNT leads abnormal epigenetic programming of imprinted and non-imprinted genes.


4. Adenoviral gene therapy is a promising therapeutic strategy to treat many conditions, particularly neurological disorders; however, a major issue impeding the use of gene therapy in the brain is the route of administration, as the blood brain barrier largely limits the transduction of the neuronal cells (e.g., neurons and glia). Injection of viral vectors through the cerebrospinal fluid is thought to be a viable alternative for several neurological conditions (e.g., lysosomal storage diseases). This paper is the first to show that helper dependent adenoviral vectors delivered by a single intrathecal injection result in transduction and long-term transgene expression from neuroependymal and neuronal cells in the mouse brain.


*Corresponding author

My Bibliography at NCBI
https://www.ncbi.nlm.nih.gov/sites/myncbi/1jgffQiSgO_Ai/bibliography/42093496/public/?sort=date&direction=ascending

D. RESEARCH SUPPORT
Current support relevant to this application

<table>
<thead>
<tr>
<th>Dindot (PI)</th>
<th>11/01/2014-02/30/2017</th>
</tr>
</thead>
</table>

Foundation for Angelman Syndrome Therapeutics
Development and characterization of a pig model of Angelman syndrome
Synopsis: The goal of this study is to generate and characterize a pig model of Angelman syndrome using the CRISPR-cas and somatic cell nuclear transfer technologies.
Synopsis: The goal of this study is to examine the epigenetic regulation of the $Ube3a$ gene in the brain and investigate therapeutic strategies in mouse models of Angelman syndrome.

Role: PI

Past support relevant to this application

- **1R01AG042189** Sohrabji (PI) 09/1/2011-08/30/2016
  NIH-NIA/NINDS
  Epigenetics of the Aging Astrocyte: Implications for Stroke
  Synopsis: The goal of this study is to examine age and sex specific epigenomic changes in astrocytes obtained from the ischemic cortex. Our laboratory is characterizing sex and age associated changes to the astrocyte epigenome before and after ischemic stroke.
  Role: Co-I

- **Explorer Award** Dindot (PI) 05/01/2013-04/30/2016
  Simons Foundation Autism Research Initiative
  Development and characterization of transgenic mouse models of 15q Duplication syndrome
  Synopsis: The goal of this study is to generate and characterize transgenic mouse models of 15q Duplication syndrome.
  Role: PI

- **Research Grant** Dindot (PI) 01/01/2012-04/30/2014
  Angelman Syndrome Foundation
  Examining rescue of neurological deficits in Angelman syndrome mice by expression of the E6-AP isoforms
  Synopsis: The goal of this study is to examine the ability of the human $UBE3A$ isoforms to rescue neurological deficits in a mouse model of Angelman syndrome.
  Role: PI

- **Research Grant** Reiter (PI)
  15q Duplication Alliance
  Construction of Tet-responsive $Ube3a$ over-expression mice
  Synopsis: The goal of this study is to develop an inducible mouse model of 15q Duplication syndrome.
  Role: Co-PI

- **Research Grant** Dindot (PI)
  Angelman Syndrome Foundation
  Determining the role of the E6-AP isoforms in synaptic maturation
  Synopsis: The goal of this study is to examine rescue of dendritic spine defects in a mouse model of Angelman syndrome.
  Role: PI
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: Dustin W. DuBois, PhD
POSITION TITLE: Assistant Professor
eRA COMMONS USER NAME (credential, e.g., agency login): DWDUBOIS

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.)

<table>
<thead>
<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
<th>Completion Date MM/YYYY</th>
<th>FIELD OF STUDY</th>
</tr>
</thead>
<tbody>
<tr>
<td>University of Texas, Austin, Texas</td>
<td>B.A.</td>
<td>05/1998</td>
<td>Biology</td>
</tr>
<tr>
<td>Texas A&amp;M University Health Science Center</td>
<td>Ph.D.</td>
<td>08/2004</td>
<td>Pharmacology &amp; Biomedical Sciences</td>
</tr>
<tr>
<td>Wake Forest University</td>
<td>Postdoc</td>
<td>09/2006</td>
<td>Pharmacology &amp; Physiology</td>
</tr>
<tr>
<td>University of Wisconsin</td>
<td>Postdoc</td>
<td>12/2008</td>
<td>Pharmacology, Anesthesiology, Physiology</td>
</tr>
</tbody>
</table>

A. Personal Statement:

As an Assistant Professor in the Department of Neuroscience and Experimental Therapeutics at the Texas A&M University Health Science Center, my research interests have focused on understanding the cellular and molecular mechanisms governing the interaction between a wide array of neuropsychopharmacological agents and their respective receptors. For 12 years, I have investigated the effects of ethanol exposure to neurons focusing on its impact to GABAergic and Glutamatergic neurotransmission. Two years were spent examining the chronic effects of ethanol on the Glutamatergic and GABAergic neurotransmitter systems in brain areas regulating anxiety, and 10 years were devoted to understanding the consequences of perinatal ethanol exposure on the developing GABAergic system in the basal forebrain. Unfortunately during my early postdoctoral career, I suffered a devastating illness due to an insidious genetic blood clotting disorder (Factor V Leiden) that slowed my career progress and impacted my scientific productivity. However, during the past 6 years my health and scientific productivity have recovered. Most recently, I have spent the last four years investigating the effects of cognitive aging on basal forebrain neurons in collaboration with Drs. William Griffith and David Murchison. These projects have included the examination of the effects of estrogen on basal forebrain neurons during reproductive aging, and our current project expands upon these interests of understanding excitatory and inhibitory synaptic transmission by examining the impact of aging on excitatory and inhibitory synaptic transmission in the basal forebrain using optogenetically engineered transgenic mouse lines. My contributions as a co-investigator have been to conduct and design the electrophysiological experiments of this important project as well as aid in implementing important cognitive behavioral tasks. I have brought 16 years of electrophysiological expertise in various preparations such as dissociated neurons and acute brain slice to this project. In addition, I have experience with multiple animal models including those of both rats and mice. Throughout my research, I have also used molecular biological approaches such as western blotting and single-cell RT-PCR to understand the contributions of gene and protein expression. Since returning to Texas A&M, I have worked closely with my collaborators, Drs. Griffith and Murchison, and I believe that this exciting proposal significantly advances our understanding of the impact of aging on synaptic transmission in the basal forebrain and aid in identifying new targets for potential therapies. I look forward to achieving the goals of this exciting project.


B. Positions and Honors

2004-2006  Postdoctoral Fellow, Dept. of Physiology and Pharmacology, Wake Forest University School of Medicine, Winston-Salem, N.C.
2006-2008  Postdoctoral Research Scientist, Dept. of Anesthesiology, Univ. of Wisconsin, School of Medicine, Madison, WI
2009-2010  Research Scientist, Dept. of Neuroscience and Experimental Therapeutics, College of Medicine, Texas A&M Univ. Health Science Center, College Station, TX
2010-present  Assistant Professor of Research, research track, Dept. of Neuroscience and Experimental Therapeutics, College of Medicine, Texas A&M University Health Science Center, College Station, TX

C. Contributions to Science (underlined authors are current co-investigators)

1. GABAA receptors as a target of early postnatal ethanol exposure. Much of my research career has focused on examining the GABAA receptor as a target of ethanol. Ethanol is often viewed as a ‘dirty drug’ that impacts many targets and developmental processes. Since GABAA receptors also serve as an excitatory, neurotrophic role during brain development in addition to their normal role as an inhibitory neurotransmitter receptor in adulthood, they are of particular interest. Understanding the role that GABAA receptors play in fetal alcohol spectrum disorders is critical to the development of potential therapies for this devastating disorder.


2. The basolateral amygdala as a target of chronic ethanol exposure. The amygdala is an important target of ethanol exposure as it controls anxiety-like behavior. Ethanol “chemically conditions” the neurotransmitter systems of the amygdala to create enhanced anxiety upon withdrawal from ethanol. Understanding the role the amygdala plays in adult alcoholism is crucial to the development of novel therapies.

3. The use of varenicline as a potential therapy for fetal alcohol spectrum disorder. We initiated experiments in this area to test the hypothesis that the nicotinic partial agonist, varenicline, may serve as a novel therapy for FASD. To our surprise, we have found that varenicline can reverse certain ethanol-induced perturbations. Current studies are underway to examine even more characteristics related to ethanol-induced changes in behavior and GABA synaptic transmission. These data are important for determining whether varenicline will prove to be a viable therapy for FASD.


Complete List of published works in MyBibliography:
http://www.ncbi.nlm.nih.gov/sites/myncbi/1TEwhfrLM00Aq/bibliography/49312456/public/?sort=date&direction=ascending

D. Research Support

Ongoing Research Support
Currently funded under:
The Interaction of Varenicline, Ethanol, and CNS Development. The overall goal of this project is to examine sex differences associated with perinatal ethanol exposure and the use varenicline as a potentially novel therapy for FASD. No overlap with Estrogen or optogenetic projects.

1R01 AG047652-01 Griffith (PI) 6/15/2014-2/28/2019
Optogenetic approaches to study complex neuronal circuits during cognitive aging. The goal of this project is to establish and examine novel optogenetic mouse lines as a means to study cognitive aging.
Role: Co-Investigator

Estrogens, ovarian aging, and calcium channel modulation. The goal of this project is to examine the impact of estrogens on cognitive aging in female specimens.
Role: Co-Investigator

Research Support Completed
2R01 AA12386-06-09 Gerald Frye (PI) 08/01/08-07/31/12
CNS development, GABAARs and Vulnerability to Ethanol
The goal of this study is to characterize ethanol-induced damage to GABA synaptic function in medial septum / diagonal band neurons in brain slices and validate a septal neuron cell culture model as a tool to understand the mechanisms involved. A primary focus includes testing the hypothesis that ethanol-induced formation of endogenous neurosteroids distorts GABA synaptic development and adversely impacts spatial learning and memory performance in Morris water maze.
Role: Co-Investigator

Postdoctoral Fellowship Training Grant Award 08/01/04-08/01/06
Alcohol Training Program, Wake Forest University School of Medicine
Role: Postdoctoral Investigator
Individual Postdoctoral Fellowship
NIH

Project Goal: Ethanol’s Effect on Basolateral Amygdala GABA Receptors (in Dr. McCool’s laboratory), but declined due to health problems and transitioning to postdoctoral position in Dr. Pearce’s laboratory.
NAME: David J. Earnest

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

POSITION TITLE: 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.)

<table>
<thead>
<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
<th>Completion Date MM/YYYY</th>
<th>FIELD OF STUDY</th>
</tr>
</thead>
<tbody>
<tr>
<td>University of Michigan, Ann Arbor, MI</td>
<td>B.S.</td>
<td>1976</td>
<td>Zoology</td>
</tr>
<tr>
<td>Northwestern University, Evanston, IL</td>
<td>M.S.</td>
<td>1979</td>
<td>Neurobiology</td>
</tr>
<tr>
<td>Northwestern University, Evanston, IL</td>
<td>Ph.D.</td>
<td>1984</td>
<td>Neurobiology</td>
</tr>
<tr>
<td>University of Rochester School of Medicine, Rochester, NY</td>
<td>Post-Doc</td>
<td>1984-87</td>
<td>Neurobiology</td>
</tr>
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</table>

A. Personal Statement

I have 30 years of experience in the application of multidisciplinary approaches to study the cellular and molecular neurobiology of cell-autonomous circadian clocks in the suprachiasmatic nucleus (SCN) and in peripheral tissues throughout the body with specific expertise in the analysis of how environmental cues and endogenous signaling molecules mediate temporal coordination of these clocks. Our current studies use animal and in vitro models to study: 1) the role of microRNAs (miRNAs) and other signaling molecules such as estrogen in the local temporal coordination of cell- and tissue-specific circadian clocks; 2) mutual interactions between the circadian clock mechanism, inflammatory signaling and metabolism; and 3) the mechanisms linking circadian rhythm disruption with metabolic disorders such as obesity and diabetes, and with pathological changes in neuroprotective responses to stroke. My previous grant funding has provided ample experience with coordinating interdisciplinary research projects involving multiple investigators. I also serve on the executive committee for the Center for Biological Clocks Research (CBCR), which was established in 2003 to coordinate training, research and outreach activities in circadian biology at Texas A&M. This research proposal is an extension of my collaborative interactions with Dr. Farida Sohrabji over the past three years. Through this collaboration, we have been successful in establishing her model to study sex and age differences in the neuroprotective responses to ischemic stroke and its application to study effects of shift work-related circadian rhythm disruption on ischemic stroke outcomes. Our collaborative experiments have yielded key findings that provide the conceptual basis for this proposal and have been recently published in *Endocrinology*.

B. Positions and Honors

<table>
<thead>
<tr>
<th>Year</th>
<th>Position and Institution</th>
</tr>
</thead>
<tbody>
<tr>
<td>1984-87</td>
<td>NIMH Postdoctoral Fellow (MH 09129), Department of Neurobiology and Anatomy, University of Rochester School of Medicine</td>
</tr>
<tr>
<td>1987-91</td>
<td>Faculty Scientist, Department of Neurobiology and Anatomy, University of Rochester School of Medicine</td>
</tr>
<tr>
<td>1991-94</td>
<td>Assistant Professor, Department of Neurobiology and Anatomy, University of Rochester School of Medicine</td>
</tr>
<tr>
<td>1991-94</td>
<td>Assistant Professor, Department of Human Anatomy and Medical Neurobiology, Texas A&amp;M University Health Science Center, College of Medicine</td>
</tr>
<tr>
<td>2001-2005</td>
<td>Associate Professor, Department of Human Anatomy and Medical Neurobiology, Texas A&amp;M University Health Science Center, College of Medicine</td>
</tr>
</tbody>
</table>
2005-present  Professor, Department of Neuroscience and Experimental Therapeutics
            Texas A&M University Health Science Center, College of Medicine

Other positions:
2003-Present  Executive Member, Center for Biological Clocks Research, Texas A&M University
2006- present  Joint Faculty Appointment, Department of Biology, TAMU
1995- present  Faculty of Neuroscience/Texas A&M Institute of Neuroscience/Faculty of Reproductive Biology

Honors and Professional Service:
1978-Present  Member, Society for Neuroscience
1986-Present  Member, Society for Research on Biological Rhythms
1997  NIH Special Emphasis Panel on the Molecular Biology of Sleep
1999  NIH Special Emphasis Panel on the Phenotypic Characterization of Sleep in Mice
2002  NIH Special Emphasis Panel ZRG1 IFCN-3 Biological Rhythms and Sleep Mechanisms
2005  NIH: ZMH1 ERB-L Conti Grant Review Panel
2005  NIAAA: AA-1 Review Group
2005  NIAA: ZRG1 F02A Behavioral Neuroscience Special Emphasis Panel
2010  NIH: ZRG1 IFCN-L (02) Special Emphasis Panel/Scientific Review Group: Biological Rhythms and Sleep
2012  NIAAA: RFA-AA-13-001, Specialized Alcohol Research Center (P50) Review
2013  NIAAA: RFA-AA-13-001, Specialized Alcohol Research Center (P50) Review

C. Contribution to Science

1. Role of neurotrophins in the photic regulation of SCN circadian function: Circadian photoentrainment is governed by the rhythmic sensitivity of the SCN pacemaker to the phase-shifting effects of photic signals. Our findings were the first to implicate brain-derived neurotrophic factor (BDNF) as a critical signal in the circadian regulation of SCN pacemaker sensitivity to light. Research studies in this area demonstrate that: 1) BDNF is expressed and regulated in a circadian manner in the SCN; 2) TrkB receptors are expressed on retinohypothalamic tract fibers innervating BDNF-expressing SCN neurons; and 3) decreased BDNF expression or blockade of the TrkB receptor inhibit phase-shifting responses to light.


2. Development of immortalized cell lines for studying SCN circadian pacemaker function: Considering some of the limitations of early in vitro models for exploring SCN circadian function, we developed immortalized lines of SCN cells in 1992. These cell lines have provided a unique tool for studying the cellular and molecular neurobiology of cell-autonomous circadian clocks in the SCN. In addition to its extensive applications in my lab, the SCN2.2 cell line has been successfully exploited for the last 20 years by over 60 labs throughout the world and by 4 pharmaceutical companies. Subsequent applications of the SCN2.2 and other cell lines were extended through our application of co-culture techniques to identify diffusible factors from SCN cells that mediate the communication of circadian outputs to other cell types and the coupling between individual SCN clock cells.
3. Mutual interactions between circadian clocks, inflammatory signaling pathways and fatty acid metabolism: Our recent work has unveiled the mechanism by which circadian clock dysregulation contributes to diet-induced tissue inflammation that leads to the development of systemic insulin resistance and metabolic phenotypes associated with obesity and diabetes. Results of these studies indicate that high-fat diet and saturated fatty acids (SFA) such as palmitate modulate the core clock mechanism in macrophages, which in turn induces the proinflammatory activation of macrophages, leading to exacerbate diet-induced adipose tissue inflammation and systemic insulin resistance. Recent studies have continued to yield noteworthy results demonstrating that SFA (but not polyunsaturated fatty acids) induce cell-specific modulation of peripheral circadian clocks in a time-dependent manner and that SFA-mediated inflammation through AMPK and the NF-κB signaling pathway is responsible for this feedback dysregulation of circadian timekeeping. Our findings have important implications for the effective application of chronotherapeutic drug and/or even omega-3 fatty acid treatment strategies in the management/prevention of systemic metabolic disorders, and other inflammation-related pathologies (e.g., cardiovascular disease, stroke).

4. Permanent effects of alcohol exposure during early brain development on the SCN and the regulation of circadian rhythms: Alcohol exposure during the period of rapid brain development produces structural damage in different brain regions, often leading to long-term or permanent neurobehavioral disturbances. Using a rat model, we discovered that binge-like exposure alcohol exposure during the early postnatal period (i.e., third trimester equivalent of human brain development): 1) decreases BDNF levels and neuropeptide-containing neurons in the SCN; 2) disrupts SCN clock gene oscillations; and 3) permanently alters key properties of circadian rhythms including their free-running period, entrainment and the rate of re-entrainment to light-dark cycles and phase-shifting responses to light. These long-term alterations in circadian behavior, along with the developmental alcohol-induced changes in SCN neurotrophins (e.g., BDNF) and neuropeptides (e.g., VIP), may have important implications in clinical sleep-wake disturbances reported in neonates, children and adults exposed to alcohol in utero.

<table>
<thead>
<tr>
<th>Reference</th>
<th>Author(s)</th>
<th>Year</th>
<th>Title</th>
<th>Journal/Conference</th>
<th>Page(s)</th>
<th>PMCID</th>
<th>PMID</th>
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<tr>
<td>b.</td>
<td>Kim, S.-M., Neuendorff, N., Chapkin, R.S. and Earnest, D.J.</td>
<td>2016</td>
<td>Role of Inflammatory Signaling in the Differential Effects of Saturated and Poly-Unsaturated Fatty Acids on Peripheral Circadian Clocks.</td>
<td>EBiomedicine</td>
<td>7: 100-111. (PMID: 27322464)</td>
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5. Immediate-early gene function in circadian photoentrainment: Early research investigations in my lab contributed to the initial identification of immediate-early genes as key components of the pathway for circadian photoentrainment. Prior to our studies and similar investigations in several other labs, the anatomical substrates communicating entraining light signals to the SCN circadian clock had been defined but little information was available on the molecular mechanism underlying circadian photoentrainment. In conjunction with published observations from two other labs, our studies demonstrate that the immediate-early gene, c-fos, plays a key role in the molecular mechanism by which light signals reset the SCN clock and entrain circadian rhythms. Furthermore, we showed that the photic induction of c-fos expression occurred primarily in retinorecipient SCN neurons containing gastrin-releasing peptide.


List of Published Work in MyBibliography (from 57 peer-reviewed publications):

D. Research Support

Ongoing:

None

Pending:

R21 NS098298-01 (PI: D. Earnest) 4/1/17 – 3/31/19
NINDS/NIH
Circadian Clock Disruption and Ischemic Stroke Outcomes: Age and Sex Differences

Synopsis: The main objectives of the proposed research are to examine the effect of circadian rhythm disruption during adulthood on the severity of ischemic strokes that occur later in life and its implications in promoting a chronic proinflammatory condition that contributes to the severity of stroke outcomes. Due to significant overlap, the present AHA application will be withdrawn if this NIH proposal is funded.
**Completed (in the last 5 years):**

**#14GRNT18370013**  
Role: PI  
1/1/14-12/31/15  
AHA  
Circadian Clocks and Neuroprotection in Response to Stroke during Reproductive Aging  

Synopsis: The main objective of the proposed research is to identify mid-life changes in peripheral circadian clocks in reproductive senescent females and determine whether alterations in their timekeeping function are related to the loss of ovarian hormones. No overlap with present proposal.

**P01 NS39546 (PI: V. Cassone)**  
Role: Project 3 Leader (10% effort)  
07/01/2006 – 06/30/2012  
NINDS/NIH  
Coordination of Circadian Physiology of Diverse Species; Project 3: Intercellular Integration of SCN Output Signals  

Synopsis: The overall objective of this project was to identify the diffusible outputs from SCN cells that coordinate circadian clocks in other cell types and restore circadian timekeeping in mutant or SCN-lesioned arrhythmic rodents in vivo. This application is completed.
# Biographical Sketch

Provide the following information for each individual included in the Research & Related Senior/Key Person Profile (Expanded) Form.

<table>
<thead>
<tr>
<th>NAME</th>
<th>POSITION TITLE</th>
<th>ASSOCIATE PROFESSOR</th>
</tr>
</thead>
<tbody>
<tr>
<td>SHOSHANA EITAN</td>
<td></td>
<td>ASSOCIATE PROFESSOR</td>
</tr>
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**EDUCATION/TRAINING** (Begin with baccalaureate or other initial professional education, such as nursing, and include postdoctoral training).

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<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (IF APPLICABLE)</th>
<th>YEAR(S)</th>
<th>FIELD OF STUDY</th>
</tr>
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<tbody>
<tr>
<td>Open University, Tel-Aviv, Israel</td>
<td>BA</td>
<td>1987-90</td>
<td>Biology</td>
</tr>
<tr>
<td>Weizmann Institute of Science, Rehovot, Israel</td>
<td>MsC</td>
<td>1990-92</td>
<td>Neurobiology</td>
</tr>
<tr>
<td>Weizmann Institute of Science, Rehovot, Israel</td>
<td>PhD</td>
<td>1992–97</td>
<td>Neurobiology</td>
</tr>
<tr>
<td>Norman Cousin Center of Psychoneuroimmunology, UCLA, Los Angeles, CA</td>
<td>Trainee</td>
<td>1997-00</td>
<td>Neuropharmacology</td>
</tr>
<tr>
<td>Neuropsychiatric Institute, UCLA, Los Angeles, CA</td>
<td>Post-Doctorate</td>
<td>2000-02</td>
<td>Neuropharmacology</td>
</tr>
<tr>
<td>Neuropsychiatric Institute, UCLA, Los Angeles, CA</td>
<td>Assistant Researcher</td>
<td>2002-05</td>
<td>Neuropharmacology</td>
</tr>
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</table>

**RESEARCH AND PROFESSIONAL EXPERIENCE:**

**Positions and Employment (in chronological order):**

1997-2000 Trainee, Norman Cousin Center of Psychoneuroimmunology, UCLA, Los Angeles, CA
2000-2002 Post-Doctoral Fellow, Neuropsychiatric Institute, UCLA, Los Angeles, CA
2002-2005 Assistant Researcher, Neuropsychiatric Institute, UCLA, Los Angeles, CA
2005-2013 Assistant Professor, Behavioral and Cellular Neuroscience, Department Of Psychology, Texas A&M University, College Station, TX
2008-present Faculty Member, Texas A&M Institute for Neuroscience (TAMIN), College Station, TX
2013-present Associate Professor, Behavioral and Cellular Neuroscience, Department Of Psychology, Texas A&M University, College Station, TX

**Honors:**

1992 Wolf Prize Award for Master Students.
1997 Rothschild Foundation Scholar.
2012 One-time extraordinary merit award (CLLA).
2014 One-time merit award (CLLA).

**Memberships:**

- Society of Neuroscience
- Society of Neuroscience, Texas A&M chapter
- Faculty of Neuroscience, Texas A&M Institute for Neuroscience

**Editorial Board membership:** Pain Studies and Treatment (PST)
Dr. Eitan had an extensive training at UCLA supervising preclinical research studies on opioid use and abuse. She is an Associate Professor at Texas A&M University, where she has successfully created a cohesive, independent, and productive research program. Her research (as demonstrated by her publications) involved extensive behavioral and molecular analyses.

List of publications (in chronological order):
Please note that in neuroscience, the authorship convention for research articles is the senior person on the project (the principal investigator) is placed as last author. As stated in the publication guidelines established by the Society for Neuroscience (the major neuroscience professional society, with 35,000 members), “it is usual in neuroscience and allied fields for authors to be listed in descending order of their contribution to the paper, with the exception that the senior author is often listed last”.

*Students and research assistants under the PI’s mentorship are underlined


**Ongoing Research Support**

Program to Enhance Scholarly and Creative Activities (PESCA)
5/14-10/15
Targeting oxytocin systems for treating opioid abuse in adolescents
Role: PI

Texas A&M Genomics Seed Grant
5/14-8/15
Effects of various opioids and social environment on gene expression
Role: PI
**Completed Research Support**

NIH (NIDA)
9/06-8/08
Functionality of the opioid system during adolescent development across genders
Role: PI

Hogg Foundation for Mental Health
6/10-6/11
Mood disorders co-morbidities and nonmedical opioid use
Role: PI

College of liberal Arts (CLLA)
4/13-11/14
Opioid use in pediatric pain management
Role: PI
**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: Jonathan A. Friedman, M.D.

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

POSITION TITLE: 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.)*

<table>
<thead>
<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
<th>Completion Date MM/YYYY</th>
<th>FIELD OF STUDY</th>
</tr>
</thead>
<tbody>
<tr>
<td>University of California, San Francisco. S.F., CA</td>
<td>M.D.</td>
<td>1993-1997</td>
<td>Medicine</td>
</tr>
<tr>
<td>University of California, San Francisco. S.F., CA</td>
<td>Internship</td>
<td>1997-1998</td>
<td>General Surgery</td>
</tr>
</tbody>
</table>

A. Personal Statement

I'm a neurosurgeon specializing in complex intracranial surgery. After training at the Mayo Clinic in Rochester, MN, I joined the faculty at Dartmouth Medical School, and specialized in cerebrovascular and skull base surgery and co-directed the stroke program. Currently I direct the Texas Brain and Spine Institute, a multidisciplinary Center of Excellence in Neuroscience. The mission of the TBSI is to provide cutting edge patient care in the neurosciences and develop translational research programs and teaching in collaboration with the College of Medicine. My primary basic science research interest is in neural regeneration, with particular regard to surgical paradigms to facilitate axonal regeneration following spinal cord injury. My main focus in clinical research has been subarachnoid hemorrhage and cerebrovascular disease. For this application, I will provide expertise on neuroinflammatory disease especially and assist with project resources such as human brain samples.

The majority of my research focuses on clinical and translational studies related to cerebrovascular disease. I have specifically collaborated with coinvestigators in the current proposal on prior translational studies in cerebrovascular disease. As my clinical practice involves all aspects of neurosurgery with a special focus in cerebrovascular surgery, I will be able to support the current proposal with brain tissue for the studies as outlined.


B. Positions and Honors

Positions
1989, 1990 Research Fellow, Department of Rheumatology, Northwestern University, Chicago, IL
1993 Research Fellow, Department of Neurology, University of California, San Francisco
1994 Research Fellow, Department of Neurosurgery, University of Heidelberg, Germany
2001-2002 Post-Doctoral Fellow, Department of Molecular Neuroscience, Mayo Clinic, Rochester, MN
2003-2005 Assistant Professor, Section of Neurosurgery, Dartmouth Medical School, Lebanon, NH
2003-2005 Staff Neurosurgeon, Dartmouth Hitchcock Medical Center, Lebanon, NH
2005-2008 Assistant Professor, Departments of Surgery, Neurosciences, and Experimental Therapeutics, Texas A&M Health Science Center, College of Medicine, College Station, TX
2008-2014 Associate Professor, Departments of Surgery, Neurosciences, and Experimental Therapeutics, Texas A&M Health Sciences Center, College of Medicine, College Station, TX
2005-Present Regional Associate Chair of Surgery-Bryan/College Station Campus, Texas A&M Health Science Center, College of Medicine, College Station, TX
2005-Present Director, The Texas Brain & Spine Institute, Bryan/College Station, TX
2005-Present Staff Neurosurgeon, St. Joseph Hospital, Bryan, TX
2007-2010 Associate Dean, Bryan – College Station Campus, Texas A&M Health Science Center College of Medicine, College Station, TX
2014-Present Professor, Department of Surgery, Neurosciences, and Experimental Therapeutics, Texas A&M Health Science Center, College of Medicine, College Station, TX

Honors
1988 Illinois State Scholar
1988-1992 Chancellor’s Scholar, University of California, Berkeley
1991 Phi Beta Kappa
1992 Graduation with High Honors, University of California, Berkeley
1993, 1994 Dean’s Research Fellowship, University of California, San Francisco
2002 Mayo Brothers Distinguished Fellowship Award
2002 Scholarly Clinician Award, Mayo Foundation
2003 Synthes Award for Resident Research in Spinal Cord Injury
2008 Health Policy Scholarship, American College of Surgeons, American Association of Neurological Surgeons
2009 Champion of Care, St. Joseph Foundation, Bryan, TX
2014-2015 Preceptor of the Year, O.C. Cooper Preceptorship Program, Texas A&M Health Science Center College of Medicine, Family & Community Medicine
2016 Always Honor, CHI St. Joseph Health

Clinical Licensure and Specialty Board Certification
Board Certified American Board of Neurological Surgery 2006 to Present

Medical Licensure
Texas 2005 to Present
New Hampshire 2003-2005
Arizona 2002-2004
Wisconsin 2000-2003
Minnesota 1998-2003

Memberships in Professional Societies
Neurosurgical Society of America
Texas Association of Neurological Surgeons
American College of Surgeons
Neurocritical Care Society
Brazos Valley Physicians Organization
Section on Cerebrovascular Surgery, American Association of Neurological Surgeons
American Association of Neurological Surgeons
Congress of Neurological Surgeons
Service
Director, The Texas Brain and Spine Institute, Bryan – College Station, TX
Editorial Board, Neurocritical Care
Ad hoc Referee:
  Clinical Neurology and Neurosurgery
    Journal of Neurology, Neurosurgery, and Psychiatry
    Brain Research
    Clinical Anatomy
  Neurosurgical Focus
    Practical Reviews in Neurosurgery

C. Contribution to Science

1. Axonal regeneration after Spinal cord injury

2. Novel spinal cord repair

3. Treatment of cerebrovascular disease

4. Imaging in vascular disease
D. Research Support

Ongoing:
1R01AG042189 (F. Sohrabji) Role: Co-I 9/15/11-5/31/17
NIH (NIA/NINDS/ORWH)
Epigenetics of the Aging Astrocyte: Implications for Stroke
Major goals: The overall goal of this application is to identify aging- and stroke-related epigenomic changes in astrocytes (in response to RFA ES 10-002). No overlap with present proposal.
Curriculum vitae

Investigator: Luis Rene Garcia

Undergraduate Institution: University of Texas at Austin
Major: Microbiology
Degree & Year: BS with Special Honors, 1990

Graduate Institution: University of Texas at Austin
Major: Microbiology
Degree & Year: Ph.D., 1996

Postdoctoral Institution: California Institute of Technology
Area: Behavioral and Developmental Genetics

Positions and Employment
1. 1990: Research Intern. NIH, Bethesda, Maryland. Supervisor: Dr. Rose Mage.
2. 1990-1996: Graduate Student. University of Texas at Austin, Dept of Microbioloy. Supervisor: Dr. Ian J. Molineux
4. 2000-2002: Howard Hughes Postdoctoral Scholar. California Institute of Technology, Division of Biology and Associate, Howard Hughes Medical Institute. Supervisor: Dr. Paul W. Sternberg
5. 2002-2008 Assistant Professor, Department of Biology; Texas A&M University
6. 2008- present Associate Professor, Howard Hughes Medical Institute Investigator, Home Institute, Department of Biology; Texas A&M University

Honors
1. The Texas Achievement Award (5 year undergraduate scholarship)
2. NSF minority pre-doctoral fellowship (accepted)
3. Ford Foundation pre-doctoral fellowship (declined in order to accept the NSF Award)
4. University of Texas Ex Students' Association Ethel and Robert L. Terry Memorial Scholarship.
5. National Research Service Award Postdoctoral fellowship.
6. Searle scholars Award
7. Presidential Early Career Award for Scientists and Engineers
8. Howard Hughes Medical Institute investigator

Appointments:
September 2008: Howard Hughes Medical Institute
September 2008: Associate professor Department of Biology, Texas A&M University
September 2002: Assistant professor Department of Biology, Texas A&M University

Publications


Guo, X and García, LR. 2014. SIR-2.1 integrates metabolic homeostasis with the reproductive neuromuscular excitability in aging male *C. elegans*. *eLife*:3:e01730


BIOGRAPHICAL SKETCH

Provide the following information for the Senior/key personnel and other significant contributors in the order listed on Form Page 2. Follow this format for each person. DO NOT EXCEED FIVE PAGES.

<table>
<thead>
<tr>
<th>NAME</th>
<th>POSITION TITLE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Garcia, Tanya Pamela</td>
<td>Assistant Professor of Biostatistics</td>
</tr>
</tbody>
</table>

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

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

<table>
<thead>
<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE</th>
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<th>FIELD OF STUDY</th>
</tr>
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<tbody>
<tr>
<td>University of California, Irvine</td>
<td>B.S.</td>
<td>06/2003</td>
<td>Mathematics</td>
</tr>
<tr>
<td>University of California, Berkeley</td>
<td>M.S.</td>
<td>05/2005</td>
<td>Industrial Engineering &amp; Operations Research</td>
</tr>
<tr>
<td>University of Western Ontario, London, ON, Canada</td>
<td>M.S.</td>
<td>08/2006</td>
<td>Statistics</td>
</tr>
<tr>
<td>Texas A&amp;M University, College Station</td>
<td>Ph.D.</td>
<td>08/2011</td>
<td>Statistics</td>
</tr>
<tr>
<td>Texas A&amp;M University, College Station</td>
<td>Postdoctoral</td>
<td>12/2012</td>
<td>Statistics</td>
</tr>
</tbody>
</table>

A. Personal Statement

As a trained statistician, I am proposing in this K01 to acquire rigorous training of Huntington’s disease (HD) in terms of disease-related background and statistical training in methods beyond my current expertise so that I may produce targeted methods designed to maximally use information from the rich data of HD studies. I have a long-standing interest in interdisciplinary collaborations, beginning as a graduate student and continuing through my current collaborations where I develop practical methods that address challenges in neurodegenerative disease research and advance the statistical theory underlying those methods. My established record of success includes the achievement of fellowships and awards from 2008-2015 totaling over $350,000, including recognition as the 2011 American Statistical Association Gertrude M. Cox recipient (awarded annually to two promising, predoctoral female statisticians). I am a 2013-2015 Huntington’s Disease Society of America Human Biology Project Fellow where I have worked with Drs. Karen Marder (a primary mentor) and Yuanjia Wang (co-mentor) to predict when HD motor-diagnosis occurs using only family history information from first-degree relatives, or independent baseline information. Despite my contributions to HD research, predictions of HD motor-diagnosis can be significantly improved by exploiting the longitudinal effects of clinical performance measures and rich neuroimaging data without compromising their structures for simplicity. Currently, I do not have the expertise to handle these complexities, but my extensive training in health-driven statistical methodology and strong programming skills (Matlab, R, Fortran 90, C++) provides me the unique expertise to carry out the proposed research. In addition, the world-class experts in my mentoring and collaboration team will provide me access to the necessary expertise and guidance to ensure success of the proposed project.

B. Positions and Honors

Employment

<table>
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<tr>
<th>DATE</th>
<th>POSITION</th>
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<tbody>
<tr>
<td>09/2002 - 06/2003</td>
<td>Research Assistant, Department of Mathematics, University of California, Irvine</td>
</tr>
<tr>
<td>06/2004 - 08/2004</td>
<td>Research Assistant, RWTH Institute of Operations Research and Logistics, Aachen, Germany</td>
</tr>
<tr>
<td>06/2006 - 08/2006</td>
<td>Research Assistant, Department of Statistics, University of Western Ontario, London, Canada</td>
</tr>
<tr>
<td>10/2006 - 07/2008</td>
<td>Graduate Assistant, Department of Statistics, University of Neuchâtel, Switzerland</td>
</tr>
<tr>
<td>07/2008 - 08/2008</td>
<td>Research Assistant, The University of Texas M.D. Anderson Cancer Center, Houston, TX</td>
</tr>
<tr>
<td>06/2010 - 08/2010</td>
<td>Research Assistant, Oak Ridge National Lab, Oak Ridge, TN</td>
</tr>
<tr>
<td>11/2012</td>
<td>Visiting Scholar, School of Mathematics and Statistics, The University of Sydney, Australia</td>
</tr>
</tbody>
</table>
Honors

2000  National Hispanic Scholar Finalist
2000 - 2003  The National Dean's List
2000 - 2002  Leadership Scholarship
2002 - 2003  Ronald E. McNair Scholar, University of California, Irvine
2003  Phi Beta Kappa Member, University of California, Irvine Chapter
2003  Graduated *Summa Cum Laude*, School of Physical Sciences, University of California, Irvine
2003  Outstanding Senior in Mathematics, University of California, Irvine
2003  Mathematical Departmental Service/Undergraduate Service Award, University of California, Irvine
2003 - 2005  Chancellor’s Opportunity Predoctoral Fellowship, University of California, Berkeley ($67,000)
2006  Teaching Assistant of the Year in the Sciences, University of Western Ontario
2010  William S. Connor Award, Texas A&M University
2010  Ford Foundation Predoctoral Fellowship Honorable Mention
2011  Phi Kappa Phi Member, Texas A&M University Chapter
2011  American Statistical Association, Gertrude M. Cox Award

Professional Societies

2008 - Present  Member, American Statistical Association
2008 - Present  Member, Institute of Mathematical Statistics
2008 - Present  Member, Eastern North American Region/International Biometric Society
2011 - Present  Member, International Chinese Statistical Association

C. Contribution to Science

1. Knowledge of biological factors linked to neurodegenerative diseases is essential for understanding disease-development and determining preventive treatments. I have contributed two new statistical methods. First, I have developed a new statistical framework for predicting the genetic risk of a neurodegenerative disease using family history information from first-degree relatives. The method has proved useful in better understanding rare genetic mutations such as with Huntington's, Parkinson's and Alzheimer's diseases. Second, I have developed a strategically simple method that unbiasedly evaluates the effects of potential biomarkers on the distribution of ages when different events occurred that most impact a subject's normal life (e.g., cognitive and motor impairment). The methods have aided to prioritize certain variables/features in clinical trial planning.


2. With the emergence of new technologies, biomedical studies often collect large amounts of data on a small subset of subjects so as to not miss any informative variables/features. An ensuing statistical challenge is
parsing through the data to identify truly informative variables/features and to make inference about their effects on a scientific response of interest. I have contributed an unprecedented feature selection procedure with two key novelties. First, variables/features are selected using data-driven weights that are shown to improve the stability and accuracy of the variables/features selected. Second, variables/features classified in a tree-structure (e.g., microbes classified at different taxonomy levels) are selected such that our method identifies the important clusters at each level of the tree.


3. In a field in which the vast majority of models require accommodating missing data, I have addressed the challenging task of jointly modeling the mean and covariance matrix for incomplete longitudinal data and time-series data by introducing a novel, data-based and graphical method which is simple to implement and handles missing values well. The method allows researchers to easily visualize their data and formulate appropriate mean-covariance models without resorting to standard choices in software packages.


**Complete List of Published Work in MyBibliography:**

**C. Research Support**

**Ongoing Research Support**

<table>
<thead>
<tr>
<th>Institution</th>
<th>Principal Investigator</th>
<th>Start Date</th>
<th>End Date</th>
</tr>
</thead>
<tbody>
<tr>
<td>School of Public Health Research Enhancement and Development Initiative (REDI)</td>
<td>Garcia (PI)</td>
<td>06/01/2015-05/31/2016</td>
<td></td>
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<tr>
<td>Huntington’s Disease Society of America</td>
<td>Garcia (PI)</td>
<td>11/15/2013-11/15/2015*</td>
<td></td>
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<tr>
<td>Texas A&amp;M Health Science Center, Start-Up Funds</td>
<td>None</td>
<td>01/2013-12/2016</td>
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</table>

Develop a new statistical method to identify and estimate the effects of baseline neuroimaging measures in relation to age at onset for Huntington’s disease. Develop flexible and advanced statistical methods that objectively define HD motor-onset and improve its predictions through models integrating a patient’s baseline and family history information. (*I have a no-cost extension until 06/30/2016.*)

These funds are supporting my ongoing research of estimating the cumulative risk of neurodegenerative diseases for kin-cohort studies, developing feature-selection techniques and building models for multivariate data with missingness.
Postdoctoral Training Grant from the National Cancer Institute, R25T-CA090301
Carroll (PI) 07/2011-12/2012
This traineeship supported my collaboration with biologists to develop advanced statistical tools for identifying variables/features in the gut microbiota that affect obesity.

National Consortium for Graduate Degrees for Minorities in Engineering and Science (GEM) Ph.D. Fellowship
Garcia (PI) 09/2010-06/2011
This award supported my research of developing nonparametric estimators to analyze survival rates for Huntington's Disease when disease status is unknown.

Philanthropic Educational Organization (P.E.O.) Scholar Award
Garcia (PI) 09/2010-06/2011
This award supported my research of developing flexible, semiparametric methods to analyze general regression models with measurement error, and improve a general class of survival models for comparing treatments.

NSF Texas A&M University System Louis Stokes Alliance for Minority Participation Bridge to the Doctorate Fellowship
Garcia (PI) 09/2008-06/2010
This award supported my research of developing graphical methods for jointly modeling the mean and covariance of univariate longitudinal data with missingness.
NAME: Geraci, Lisa

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

POSITION TITLE: Associate Professor of Psychology

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.)

<table>
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<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
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<th>FIELD OF STUDY</th>
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<tr>
<td>Macalester College, St. Paul, MN</td>
<td>BA</td>
<td>05/1996</td>
<td>Psychology</td>
</tr>
<tr>
<td>Stony Brook University, Stony Brook, NY</td>
<td>PhD</td>
<td>05/2001</td>
<td>Cognitive Psychology</td>
</tr>
<tr>
<td>Washington University in St. Louis, St. Louis, MO</td>
<td>Post-doc</td>
<td>05/2005</td>
<td>Memory and Aging</td>
</tr>
</tbody>
</table>

A. Personal Statement

My expertise is in cognitive functioning in older adults. I have 20 years of experience conducting research using a variety of cognitive and neuropsychological tests with younger and older adults. I began my research with older adults as a post-doctoral fellow, funded by a grant from the National Institute on Aging, at Washington University in St. Louis in 2001. I have continued my research on aging and cognition as an assistant and, now, associate professor, at Texas A&M University. I have published my research in top-tier journals and obtained both NIH and NSF funding to support this research.

B. Positions and Honors

Positions and Employment

2005-2011 Assistant Professor, Department of Psychology, Texas A&M University
2011- Associate Professor, Department of Psychology, Texas A&M University

Other Experience and Professional Memberships

2005-pres Member, Association of Psychological Science
2006-pres Member, American Psychological Association
2014-pres Editorial Board, Experimental Aging Research
2014-pres Editorial Board, Journal of Memory and Language
2016-pres Editorial Board, Memory & Cognition
2014-pres NSF, Perception, Action, and Cognition Program, Board of Reviewers

Honors

2015 Fellow of the Association for Psychological Science
2013 Fellow of the Psychonomic Society
2004 National Institutes of Health Clinical Research Loan Repayment Recipient
2002 National Institute on Aging Summer Institute on Aging Research Scholar
2001 American Psychological Association Dissertation Research Award Recipient

C. Contribution to Science

My early work on aging delineated the types of memory that are affected by aging. My work showed that implicit (unconscious, unaware) memory is largely intact in healthy aging, whereas explicit (conscious,
aware) memory is impaired. Further, I found that whereas older adults have impairments in veridical memory, they have high levels of false memories.


Recent work examines the underlying reasons for age-related changes in memory and examine ways to improve memory performance for older adults. For example, I examine ways to counter older adults’ negative performance expectations to improve subsequent memory performance.


In addition to examining cognition in healthy aging, I have also examined how cognition is affected by Alzheimer’s disease.


D. Additional Information: Research Support and/or Scholastic Performance

**Ongoing Research Support**

<table>
<thead>
<tr>
<th>BCS1348944</th>
<th>Geraci (co-PI)</th>
<th>3/01/2014-2/28/2017</th>
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<tbody>
<tr>
<td><em>Unity and diversity in self-regulation and executive functioning</em></td>
<td></td>
<td></td>
</tr>
<tr>
<td>The goal of this project was to assess the common underlying cognitive processes involved in self-regulation and executive functioning tasks.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Role: co-PI (with Schmeichel, co-PI)</td>
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**Completed Research Support**

<table>
<thead>
<tr>
<th>R01AG039502</th>
<th>Geraci (PI)</th>
<th>10/01/2011-9/31/2015</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Investigating how prior task success improves memory performance in older adults</em></td>
<td></td>
<td></td>
</tr>
<tr>
<td>The goal of this project was to use prior task success to improve memory performance in older adults and to examine the mechanisms and the boundary conditions for this effect.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Role: PI</td>
<td></td>
<td></td>
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</tbody>
</table>
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: James W. Grau

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

POSITION TITLE: Mary Tucker Currie Professor, Psychology and Neuroscience

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.)

<table>
<thead>
<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
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<th>FIELD OF STUDY</th>
</tr>
</thead>
<tbody>
<tr>
<td>University of Colorado, Boulder, CO</td>
<td>B.A.</td>
<td>05/1981</td>
<td>Molecular Bio./Psychology</td>
</tr>
<tr>
<td>University of Pennsylvania, Philadelphia, PA</td>
<td>M.A.</td>
<td>05/1982</td>
<td>Experimental Psychology</td>
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</tbody>
</table>

NOTE: The Biographical Sketch may not exceed five pages. Follow instructions below.

A. Personal Statement

I have studied factors that affect spinal cord function for over 25 years. The strength of our research program has been recognized by Texas A&M University (TAMU), which has provided generous laboratory space within the newly constructed Interdisciplinary Life Sciences Building (http://vpr.tamu.edu/resources/ilsb) and the funds needed for core equipment (for PCR, western imaging, confocal microscopy, and animal housing). More recently, I have been working to build expertise in spinal cord injury (SCI) at TAMU and the Texas A&M Health Science Center (TAMHSC) through a proposal to hire 4 new assistant professors in this area, supported in part through a gift from the TIRR Foundation.

I have published approximately 64 peer-reviewed papers, and an edited volume (Patterson and Grau, 2001), on spinal function. In addition, my laboratory regularly presents 6-8 abstracts at the Society for Neuroscience meeting. I believe that our approach has been successful because we bring a rigorous experimental methodology, and strong behavioral techniques, to the study of spinal cord function. We couple this work with robust pharmacological and cellular tools. This in turn is complemented by an eye towards clinical relevance and application, with the aim of translating our work to clinically relevant behavioral paradigms to examine both recovery after a contusion injury and the development of neuropathic pain.

Our research team (Grau and Miranda) has worked together for over 16 years. My background in behavioral/surgical/pharmacological methods is complemented by Dr. Miranda’s expertise in cellular assays and histopathology. At a conceptual level, I bring a strong background in learning/plasticity/pain and Dr. Miranda brings expertise in cell death and signal pathways.

Duties: My job description involves a focus on research (60% academic year; 100% summer months). My remaining time is spent on teaching (30% academic year) and service (10% academic year).

The following articles review our work on spinal cord plasticity:


B. Positions and Honors

**Professional Experience**

- Research Assistant, Biopsychology, Univ. of Colorado, 1978-1981, under Dr. S. F. Maier.
- Graduate Student, Psychology, Univ. of Pennsylvania, 1981-1985, under Dr. R. A. Rescorla.
- Visiting Assistant Professor, Psychology, Univ. of North Carolina at Chapel Hill, 1985-1987.
- Assistant Professor, Psychology, Texas A & M University, 1987-1992
- Associate Professor, Psychology and Faculty of Neuroscience, Texas A&M University, 1992-1998
- Professor, Psychology and Faculty of Neuroscience, Texas A&M University, 1998-present
- Chair, Texas A&M Institute for Neuroscience, 2007-2011

**Honors and Awards**

- Phi Beta Kappa
- American Psychological Association Fellow (Divisions 3, 6 and 28)
- University Faculty Fellow Award, Texas A&M University, 2000-2005 ($100,000)
- University Research Award, 2001
- Elected President of Division 6 (Behavioral Neuroscience and Comparative, Am. Psy. Assn.), 2003
- Mary Tucker Currie Professor of Psychology, Fall, 2005 (and continuing)
- American Psychological Society Fellow
- Jerry Johnston Andrew Spinal Research Award (2014, $10,000)

C. Contribution to Science

**Early Training: Learning and Pain**

As an undergraduate at the University of Colorado (Boulder) I worked with Dr. Steve Maier, who is internationally known for his work on learned helplessness and pain modulation. At that time, the endogenous opioids and their receptors had just been discovered. We hypothesized that an opioid mediated inhibition of pain (nociceptive) fibers (an antinociception) could contribute to the motivational deficit induced by uncontrollable stimulation. We found evidence for this and were able to show that exposure to uncontrollable stimulation induces a lasting increase in opioid reactivity (Grau et al., *Science*). As a graduate student, I worked with Dr. Robert Rescorla (a NAS member) at the University of Pennsylvania, strengthening my background in the areas of learning, experimental design, and through courses at the medical school, pharmacology. For my dissertation, I outlined how alternative forms of antinociception may be linked to learning/memory and proposed a model of these phenomena (Grau, 1987). My subsequent work continues to respect the importance of experimental design and the complexities of behavioral assessment. That perspective has influenced graduate students, such as Dr. Tamara King (now an assoc. prof. at the University of Maine) who developed a popular method to assess chronic pain in animals (based on context conditioning; King et al., 2009, *Nature Neuroscience*).


**Evidence for Spinally Mediated Instrumental Learning and the Role of BDNF**

We began to explore whether spinal systems are sensitive to environmental relations in the late 1980’s. Building on prior work by Thompson and his colleagues, we showed that spinal antinociceptive systems are sensitive to stimulus-stimulus (Pavlovian) relations (Grau et al., 1990). We were the first to show that this
system exhibits a number of complex Pavlovian phenomena (e.g., blocking, overshadowing; Illich et al., 1994) and unravel the functional mechanism that underlies the learning (Joynes et al., 1996). Recognizing that learning about response-outcome (instrumental) relations is potentially more relevant to physical training, we then began to explore whether spinal systems could support this form of learning. While prior work had shown that instituting a relation between hindleg position and shock could bring about a change in leg position, the results were open to alternative interpretation (Church, 1989) and for this reason, ignored. Thus, as of 1998, those working within the field of learning could claim that instrumental learning required brain systems. We systematically evaluated the alternative interpretations of prior work, refined the definitions of instrumental and operant learning (Grau, 2000), and developed new methods to show, beyond a doubt, that spinal systems are sensitive to response-outcome relations (Grau et al., 1998). We also discovered that exposure to noxious shock independent of leg position (uncontrollable stimulation) induces a lasting inhibition of instrumental learning, a phenomenon reminiscent of learned helplessness. Interestingly, this adverse effect can be prevented and reversed by exposure to controllable stimulation (Crown & Grau, 2001), an effect we have subsequently shown depends upon an up-regulation of brain derived neurotrophic factor (BDNF; Huie et al., 2012). Our work on spinal learning is reviewed in Grau, 2014.


Uncontrollable Stimulation Induces a Form of Metaplasitcity that involves Glia, TNF, and a Shift in GABA

Prior work had shown that peripheral application of an irritant (capsaicin) that engages pain (C) fibers induces a diffuse over-excitation of nociceptive neurons within the spinal cord (central sensitization) that enhances reactivity to mechanical stimulation. This sensitization has a lasting (memory-like) effect and depends upon a form of NMDA receptor (NMDAR) mediated plasticity. We posited that exposure to uncontrollable shock impairs spinal learning because it induces a similar state, diffusely saturating NMDAR-mediated plasticity and blocking the development of selective response modifications. Supporting this, Ferguson et al. (2006) showed that exposure to intermittent tail shock induces enhanced mechanical reactivity (EMR) and that pretreatment with an NMDAR antagonist blocks the development of the learning impairment. Further, treatment with a peripheral irritant (that induces both a robust EMR and central sensitization) impairs spinal learning. Subsequent work showed that the adverse effect of uncontrollable stimulation depend upon non-neuronal cells (astrocytes and/or microglia; Vichaya et al., 2009) within the spinal cord and involve the cytokine tumor necrosis factor (TNF; Huie et al., 2012). More recently, we have related these effects to an alteration in GABA function that increases neural excitability within the spinal cord (Huang et al., 2016).


Nociceptive Stimulation Impairs Recovery After Spinal Cord Injury (SCI)

The observation that uncontrollable stimulation impairs adaptive plasticity within the spinal cord led us to posit that this treatment would adversely affect recovery after a contusion injury. We showed that just 6 min of intermittent shock a day after injury impairs recovery and that this effect is evident 6 weeks after treatment (Grau et al., 2004). Uncontrollable stimulation also increases weight loss, slows the recovery of bladder function, and increases tissue loss at the site of injury. Dr. S. Garraway, now an assistant professor at Emory
University, related the adverse effect of nociceptive stimulation to a down-regulation of BDNF signaling (Garraway et al., 2011). She also showed that noxious stimulation enhances behavioral signs of chronic pain and that these effects are accompanied by increased expression of the cytokine TNF (Garraway et al., 2014). Recently, we showed that the adverse effect of nociceptive stimulation on cellular function and recovery are blocked by lidocaine administered by means of a lumbar puncture (Turtle et al., 2016).


**Other Contributions to the Spinal Cord Injury Literature**

In the course of studying SCI, we have addressed a number of important methodological issues and made some new discoveries. Early on, we recognized that our ability to evaluate alternative treatment regimes would depend upon the evaluation window. At issue is the time period over which recovery should be observed. Dr. Michelle Hook (now an assistant professor at TAMHSC) showed how an appropriate window of observation could be empirically derived (to maximize both statistical power and efficiency; Hook et al., 2004). We also examined the properties of a common behavioral measure of locomotor recovery (the BBB score) and showed how a simple transformation could improve its metric properties (Ferguson et al., 2004), making the data more amendable to parametric analyses (and thereby increasing statistical power). The graduate student who led that study (Adam Ferguson) is now an assistant professor at UCSF and is well known for his work using advanced statistical analyses to uncover the inter-relation between alternative treatments and behavioral measures (e.g., Ferguson et al., 2014, Nat Neurosci). Other work examined whether the adverse effect of nociceptive stimulation on recovery could be attenuated by treatment with a pre-emptive analgesic (morphine). Contrary to our hypothesis, we found that the adverse effect of shock treatment on recovery is unaffected by morphine treatment (Hook et al., 2009). More worrisome, we discovered that morphine treatment per se adversely affects behavioral recovery and increases mortality after SCI. These adverse effects of morphine treatment have been related to increased expression of interleukin-1 beta (IL-1ß; Hook et al., 2011).


D. Research Support

Completed

**R01 NS069537** (PI: Ferguson; coI: Grau) 04/01/10-03/31/14 NIH/NINDS

Title: Metaplasticity and recovery after spinal cord injury: cellular mechanisms

Goals: The project examined the cellular mechanisms that underlie the behavioral deficit observed after uncontrollable stimulation in spinally transected rats, with a focus on tumor necrosis factor.

Role: Co-I

Overlap: None

**R01 DA031197-01** (PI: Hook; coI: Grau) 4/1/11-3/31/16 NIH/NIDA

Title: Morphine undermines recovery of function after SCI: Neurobiological mechanisms

Goals: Prior work has shown that morphine treatment can have an adverse effect on recovery after SCI. This project examined the molecular changes that underlie this effect and how it can be prevented.

Role: co-I

Overlap: None

**R21 NS081606** (PI: Garraway, coI: Grau) 7/1/2013-6/30/2016 NIH/NINDS

Title: Cellular mechanisms underlying pain following spinal cord injury

Goals: The experiments outlined within this grant explored the mechanisms that underlie nociception induced sensitization of pain circuits after a spinal contusion injury, with a focus on tumor necrosis factor (TNF).

Role: Co-I

Overlap: None

Current Grants

**Neilsen Foundation** (PI: Grau; coI: Hook, Miranda) 12/1/14-1/31/17 Craig H. Neilsen Foundation

Title: How and when does peripheral input affect recovery after SCI

Goals: Recognizing that the environmental conditions that induce maladaptive plasticity in spinally transected rats fail to predict how nociceptive stimulation affects recovery after a contusion injury, the experiments outlined within this proposal sought to clarify the circumstances under which electrical stimulation impacts recovery and expands the region of secondary injury. We also proposed to test whether blocking electrically induced neural activity (using epidural lidocaine) has a protective effect. The cellular assays associated with this proposal yielded the serendipitous finding that nociceptive stimulation induces hemorrhage.

Role: PI

Overlap: None

**R21 NS091723** (PI: Grau; coI: Miranda) 2/1/16-1/31/18 NIH/NIDA

Title: Effect of inflammation on recovery and pain after spinal cord injury

Goals: The project uses a peripheral irritant (capsaicin) to examine how engaging pain fibers days to weeks after injury affects spinal function, how treatment induces cell death, and whether blocking the initiation of pyroptosis with BBG and/or probenecid has a therapeutic effect. Cellular assays associated with this project revealed that capsaicin treatment also induces hemorrhage.

Role: PI

Overlap: None
NAME: William H. Griffith, Ph.D.

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

POSITION TITLE: Professor & Chair

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.)

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<thead>
<tr>
<th>INSTITUTION AND LOCATION</th>
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<tr>
<td>Lamar University, Beaumont, TX</td>
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<tr>
<td>Lamar University, Beaumont, TX</td>
<td>MS</td>
<td>08/1975</td>
<td>Biology</td>
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<td>University of Texas Medical Branch, Galveston, TX</td>
<td>Ph.D.</td>
<td>12/1980</td>
<td>Pharmacology/Neurosci</td>
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<tr>
<td>School of Pharmacy, University of London</td>
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<td>12/80-10/82</td>
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<tr>
<td>Baylor College of Medicine, Houston, TX</td>
<td>Postdoctoral</td>
<td>11/82-12/83</td>
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A. Personal Statement

My development as a scientist results directly from my early training in pharmacology, electrophysiology and neuroscience from outstanding mentors. My graduate training in pharmacology was with Joel Gallagher in the Pharmacology department at UTMB in Galveston. I then worked as a postdoctoral fellow with David Brown in London in the Department of Pharmacology, School of Pharmacy. It was at this time that knowledge of M-current and other ion channel electrophysiology was just beginning in the brain, and this proved to be a very exciting time in neuroscience. Finally, I was fortunate to learn hippocampal synaptic physiology while working with Daniel Johnston at the Baylor College of Medicine. I was well prepared for my first faculty position in the Department of Medical Pharmacology & Toxicology in the College of Medicine at Texas A&M and I have remained here for over thirty years. I am currently Professor and Chair of the Department of Neuroscience and Experimental Therapeutics. My lab is in an ideal position to help conduct the proposed research.

My research program over the past many years has described age-related changes in ligand-gated channels, voltage-gated calcium channels and calcium homeostasis in basal forebrain neurons across aging and behavioral state. Our long-term goal is to identify the cellular and molecular mechanisms responsible for these age-related changes. We utilize a rodent model of aging coupled with a variety of techniques including, patch-clamp electrophysiology, measurements of intracellular calcium concentration ([Ca2+]i), laser scanning confocal fluorescent microscopy, single-cell reverse transcription/polymerase chain reaction (scRT-PCR) and behavioral characterization using the water maze. Recently, we have incorporated optogenetic stimulation techniques to our electrophysiological repertoire. The present proposal will be a natural extension of our previous work and will allow us to investigate the synaptic consequences of some of the age-related changes we have observed. Below are references demonstrating age-related changes in calcium homeostasis.


B. Positions and Honors

12/1980-10/1982 Research Associate, Department of Pharmacology, School of Pharmacy, University of London, Professor David A. Brown, Advisor
11/1982-12/1983 Postdoctoral Fellow, Department of Neurology, Section Neurophysiology, Baylor College of Medicine, Dr. Daniel Johnston, Advisor
01/1984-08/1989 Assistant Professor, Department of Medical Pharmacology & Toxicology College of Medicine, Texas A&M University
09/1089-09/1994 Associate Professor, Department of Medical Pharmacology & Toxicology College of Medicine, Texas A&M University
09/94-12/2005 Professor, Department of Medical Pharmacology and Toxicology, College of Medicine, Texas A&M University Health Science Center
01/2006- present Professor, Department of Neuroscience and Experimental Therapeutics College of Medicine, Texas A&M University Health Science Center
06/2006- present Chair, Department of Neuroscience and Experimental Therapeutics College of Medicine, Texas A&M University Health Science Center

Honors and Professional Service

1980 Pharmacology Research Award, National Student Research Forum
1980 Overall Excellence of Research Award, National Student Research Forum
1980 James E. Beall, II Memorial Award for Research in the Neurosciences
1981 Academic Excellence Award, Graduate School Biomedical Sciences at Galveston
1984-87 Councilor for the Texas A&M Chapter of the Society for Neuroscience
1988 President, Texas A&M Chapter of the Society for Neuroscience
1990-present ad hoc reviewer, National Science Foundation
1991-2004 member, Oklahoma Center for the Advancement of Science and Technology (OCAST)
1993-present ad hoc reviewer, National Institutes of Health
1994, 1998 NIH Intramural Review, Lab of Molecular and Cellular Neurobiology, NIAAA
1997 Editorial Board, American Journal of Physiology, Heart and Circulatory Physiology
1999 Chairman of Study Section Panels, OCAST
1999-2002 Editorial Board, British Journal of Pharmacology
2013-2014 Councilor (elected), Association of Medical School Neuroscience Department Chairpersons
2014 Distinguished Alumnus Award, University of Texas Medical Branch, Graduate School of Biomedical Sciences
2016-2018 President (elected) of the Association of Medical School Neuroscience Department Chairpersons

C. Contributions to Science

1. Development of a basal forebrain model to study central cholinergic neurons during aging

My early research program focused on development of an ex vivo brain slice preparation of the basal forebrain, an area of the brain thought to be involved in Alzheimer’s disease (AD) and dementia. In the mid 1980’s, the “cholinergic hypothesis” of AD was the primary working hypothesis as to the cause of AD because of numerous studies demonstrating extensive cholinergic cell death during the disease and existing clinical treatments consisted of only cholinergic therapies. What was unknown at the time, was why do cholinergic cells die and can this be prevented. We were successful in developing a brain slice preparation to study the voltage-gated currents and electrophysiological properties of cholinergic cells in the hope of identifying potential targets for improved therapeutic treatments. We were the first to record form AChE- positive neurons in the brain (1986). Even as newer theories of AD were developed, these early studies provided a foundation for many others working in the basal forebrain.
2. Mechanisms for compensatory changes in calcium homeostasis during aging

Our main emphasis over the years has been to investigate ligand-gated channels, voltage-gated calcium channels and calcium homeostasis in basal forebrain neurons across aging. Our long-term goal has been to identify the cellular and molecular mechanisms responsible for changes in age-related function and to develop targeted therapies to reverse age-related deficits. We were the first to identify a particularly intriguing modification during aging, namely, an increase in rapid intracellular calcium buffering in identified cholinergic neurons. We first reported this phenomenon in 1998 and then extended these findings to show that the increase in calcium buffering was prevented or reversed by dietary caloric restriction (2007). More importantly, we established that only cognitively impaired subjects (as assessed by water maze testing) demonstrated increased intracellular calcium buffering, while unimpaired subjects maintained buffering values similar to young (2009). Finally, only age impaired subject’s demonstrated reduced synaptic inhibition in the basal forebrain during aging (2014). Our results support a model in which aged cognitively impaired subjects demonstrate physiological modifications that disrupt calcium homeostasis during aging, while cognitively unimpaired subjects make compensatory changes to offset these age-related changes in basal forebrain neurons. We are investigating these compensatory changes that promote healthy aging.


3. Optogenetic models to study age-related change in synaptic function

We are currently extending our recent findings of decreased synaptic inhibition during cognitive aging to include investigations of specific neurotransmitter systems in animal models of age-related cognitive impairment. Our ultimate objective is to improve the quality of life in cognitively-impaired aged individuals by restoring “youthful synapses” through the use of better research tools and rational drug design. We have developed an aging colony of transgenic optogenetic mice that should prove a significant contribution to the aging field in the near future.

Complete List of Published Work in MyBibliography: https://www-ncbi-nlm-nih-gov.ezproxy.library.tamu.edu/pubmed/?term=griffith+wh
D. Research Support

**Ongoing:**

- **R01-AG041360**  
  **Griffith (PI)**  
  4/15/2011-3/31/2017 (no cost extension)  
  Estrogens, ovarian aging and calcium channel modulation.  
  $1,526,348 total costs.  
  Role: PI  
  Co-I: Dustin DuBois  
  Co-I: David Murchison

- **R01 AG047652**,  
  **Griffith (PI)**  
  6/15/14-2/28/2019  
  Optogenetic approaches to study complex neuronal circuits during cognitive aging  
  Role: PI  
  Co-I DW DuBois  
  Co-I D Murchison

- **R56AA021844**,  
  **Role Co-I**  
  6/1/2015 – 5/31/2017  
  “The Interaction of Varenicline, Ethanol, and CNS Development”;  
  Dustin DuBois (PI), WH Griffith (10% effort)

**Completed in the last five years**

- **R01-AG029421**  
  **Role Co-I**  
  8/1/07 – 7/31/12,  
  Basal Forebrain and cognitive aging: novel experimental and therapeutic avenues”,  
  J.L. Bizon (PI),  
  W.H. Griffith, Co-I

- **R01-AG007805 (years 12-17)**,  
  **Griffith (PI)**  
  9/2003-8/2010  
  Physiology of cholinergic basal forebrain neurons.  
  Role PI
BIOGRAPHICAL SKETCH

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

NAME
Ayman K. Hamouda

POSITION TITLE
Tenure-Track Assistant Professor of Pharmaceutical Sciences / Assistant Professor of Neuroscience and Experimental Therapeutics

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

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

<table>
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<tr>
<td>Al-Azhar University-Gaza, Gaza Strip</td>
<td>BPharm</td>
<td>1998</td>
<td>Pharmacy</td>
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<tr>
<td>Texas Tech University Health Science Center, Lubbock TX.</td>
<td>PhD</td>
<td>2007</td>
<td>Pharmacology and Neuroscience</td>
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<tr>
<td>Harvard Medical School, Boston, MA</td>
<td>Postdoctoral Instructor</td>
<td>2007-2009</td>
<td>Neurobiology</td>
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<tr>
<td>Harvard Medical School, Boston, MA</td>
<td></td>
<td>2009-2013</td>
<td>Neurobiology</td>
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Since 2003, I have been extensively involved in research projects in the general area of structure and pharmacology of pentameric ligand-gated ion channels especially the nicotinic acetylcholine receptors (nAChRs). Brain nAChRs are known for their role in the development of nicotine addiction (cigarette smoking). In addition, they are important for cognition, learning and neuronal survival and they are implicated in many neuropathologies including Alzheimer’s disease, epilepsy, schizophrenia, anxiety and depression. As such, selective targeting of nAChRs holds a leading position among drug strategies to treat these conditions.

As a graduate student in Dr. Michael P. Blanton’s lab, I established conditions for affinity purification of heterologously expressed neuronal nAChRs. The ability to isolate milligrams of neuronal α4β2, α4β4 and α3β4 nAChRs in lipid membranes allowed us to use, for the first time, photoaffinity labeling to study their structure and to biochemically identify the specific sites of drug interaction with these important brain receptors. As a postdoc in Dr. Jonathan B. Cohen’s lab, I established a purification scheme similar to the one I developed previously and continued to characterize drug interactions with nAChRs. Using [3H]Epibatidine, we provided the first direct identification of the mode of binding of an agonist to a neuronal nAChR.

As an independent investigator, my long-term goal is to develop nAChR subtype-selective ligands suitable for therapeutic applications. My current research primarily focuses on the pharmacology of nAChR positive allosteric modulators (PAMs) as a novel therapeutic class that provides modulation of a subpopulation of nAChRs without sustained activation and non-physiological alteration in cholinergic transmission seen with agonists. I study the pharmacology of several available nAChR PAMs in vitro using biophysical and electrophysiology methods to identify their mode of interaction with nAChR and define PAM/nAChR structural moieties that confer subtype selectivity. Recently, I am moving toward examining the effect of lead nAChR PAMs in vivo using animal models for Alzheimer’s Disease and for inflammatory pain.

A. Positions and Honors.

Positions
1998-2002 Research/Teaching Assistant, Faculty of Pharmacy, Al-Azhar University-Gaza
2003-2007 Graduate Student Research Assistant, Dept. of Pharmacology and Neuroscience, TTUHSC
2007-2009 Postdoctoral Research Associate in Neurobiology, Harvard Medical School, Boston, MA
2009-2013 Instructor in Neurobiology, Harvard Medical School, Boston, MA
2013-present Assistant Professor, Dept. of Pharmaceutical Sciences, Texas A&M HSC
2014-present Assistant Professor, Dept. of Neuroscience and Experimental Therapeutics, Texas A&M HSC

Texas A&M Institute for Neuroscience

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Honors and Awards

2003-2004 Fulbright Foreign Student Scholarship, Department of State, USA.
2005-2006 2nd Place (2005) and 1st Place (2006), Annual Research Poster Competition, TTUHSC
2006 Society for Neuroscience Graduate Student Travel Award
2006 Chancellor’s Scholarship, Texas Tech University
2007 Alexander D. Kenny Outstanding Graduate Student Award, TTUHSC
2007 GSBS Outstanding Graduate Student Award, Graduate School of Biomedical Sciences, TTUHSC
2007 Convocation speaker, Graduate School of Biomedical Science–TTUHSC
2009 Seminar, Department of Neurobiology, Harvard Medical School, Boston, MA
2010 Seminar, Department of Neurobiology, Harvard Medical School, Boston, MA
2011 Invited speaker, Department of Cell Physiology and Molecular Biophysics, TTUHSC.
2011 Invited speaker, Workshop at the Center for Membrane Protein Research, TTUHSC
2011 Seminar, Department of Neurobiology, Harvard Medical School, Boston, MA
2014- Member, Texas A&M Institute of Neuroscience (TAMIN), Texas A&M University.
2014 Invited speaker, International Brain Research Organization (IBRO)-MENA Neuroscience Conference, Doha, Qatar.
2015 Invited speaker, Department of Biology Nona Symposium, Texas A&M University.
2015 Invited speaker, Research Initiative for Scientific Enhancement (RISE) program, University of Puerto Rico.
2016- Grant reviewer: University Research Board Fund-the American University of Beirut.
2016- Grant reviewer: New Investigator Award-American Association of Colleges of Pharmacy (AACP)
2017 Research and Education Abstract Reviewer: AACP Annual Meeting
2017- Grant reviewer: American Heart Association Cell Transport BSC 2 Committee member.

B. Publications

Research Papers: (* = corresponding Author; ** = Equal Contributions)


**Book Chapters:**


**Abstracts/ Poster Presentations:**


Hamouda, A. K. Wang Z.J, Mohamed T.S., Alaskari A. (2016) "3-(2-chlorophenyl)-5-(5-methyl-1-(piperidin-4-yl)-1H-pyrazol-4-yl)isoxazole (CMPI) is a selective positive allosteric modulator of low-sensitivity (α4)3(β2)2 nicotinic acetylcholine receptor". *Society for Neuroscience, 667.15*


**C. Research Support**

4/07-10/2013: National Institute of Health  P01 GM58448 (PIs KW Miller and JB Cohen; Role, Investigator) Locating general anesthetic sites in acetylcholine receptors and GABA-A receptors.

10/2013 -10/2016: Texas A&M Health Sciences Center Faculty Development Fund ($200,000; Role, PI). Nicotinic acetylcholine receptors structure and pharmacology

7/2015 - 6/2017: American Heart Association-15GRNT25890003 ($139,802; Role, PI; Funded, relinquished to accept NIH R15 award). Identification of Positive Allosteric Modulator Binding Sites in α4β2 Nicotinic Acetylcholine Receptors.

7/2015 - 6/2018: National Institute of Health-1R15 NS093590-01 ($435,781; Role, PI; Funded). Neuronal nicotinic acetylcholine receptors (nAChRs)
NAME: Han, Arum

eRA COMMONS USER NAME (credential, e.g., agency login): ARUMHAN1

POSITION TITLE: Professor

EDUCATION/TRAINING

<table>
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<td>Seoul National University, Seoul, Korea</td>
<td>B.S.</td>
<td>1997</td>
<td>Electrical Engineering</td>
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<tr>
<td>University of Cincinnati, OH</td>
<td>M.S.</td>
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<tr>
<td>Georgia Institute of Technology, GA</td>
<td>Ph.D.</td>
<td>2005</td>
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A. Personal Statement

Dr. Han is a Professor in the Department of Electrical and Computer Engineering and the Department of Biomedical Engineering at Texas A&M University. He is also a graduate faculty at Texas A&M Health Science Center and Faculty of the Texas A&M Institute for Neuroscience. His research focuses on development of microfluidic, lab-on-a-chip, and organ-on-a-chip systems that enable unique biological experiments at high throughput and high accuracy that can then be readily adopted by the broad bio/medical science community. One of his main research areas is in the development of organ-on-a-chip systems (i.e. microphysiological systems (MPSs)) that mimic in vivo physiological responses in vitro, especially in the area of neurobiology. He has developed an in vitro central nervous system (CNS) myelination model through microfabricated co-culture and 3D organoid culture systems approach that for the first time demonstrated in vitro CNS myelination on microchip, which is currently being used for drug screening applications. He has also been developing in vitro multi-channel blood-brain barrier (BBB) MPS using a co-culture approach. Another focus area is in developing high-throughput screening microfluidic lab-on-a-chip systems for therapeutics development and toxicity testing. He has developed and utilized robust fully automated microfluidic cell-culture systems for multi-day drug and toxin screening assays. In addition, he has pioneered the development of high-throughput microfluidic lab-on-a-chip systems for microorganism research and biotechnology applications, such as systems for developing highly efficient microbial strains for biofuel production as well as systems for therapeutic development against pathogenic microorganisms causing infectious diseases. In addition, he has also developed several high-throughput single-cell assay microfluidic systems for applications in cancer metastasis diagnosis and prognosis using acoustophoresis and impedance spectroscopy principles. His main contribution to this proposal is his expertise and experiences in developing microfabricated lab-on-a-chip and organ-on-a-chip systems, especially their usage in high-throughput drug/toxin screening assays. He has authored more than 100 peer-reviewed publications and has received funding from the Bill and Melinda Gates Foundation, NIH, NSF, USDA, US Army Corp of Engineers, Defense Threat Reduction Agency (DTRA), Qatar National Research Foundation (QNRF), and several other international sponsors and private companies in the past five years. He is also the co-PI of the recent NIH/NCATS-funded Tissue Chip Testing Center (TCTC, PI: Dr. Ivan Rusyn), which was established to provide tissue chip developers the means to test and validate various tissue chip platforms independently, where he is providing his expertise in conducting tissue chip-based drug assays. Most of his research relies heavily on multidisciplinary collaboration with researchers from various fields of life sciences, including neurobiologists, microbiologists, biochemists, biomedical engineers, toxicologists, medical doctors, as well as engineers. He is experienced in leading multidisciplinary projects, including a $2M project supported by the NSF (Emerging Frontiers in Research and Development grant), demonstrating multidisciplinary team leadership.

B. Positions and Employment

Positions and Employment
1998 - 2000 Research Assistant, Department of Electrical & Computer Engineering and Computer Science, University of Cincinnati, Cincinnati, OH
C. Contributions to Science Most Relevant to This Proposal

1. **Experience in developing microfluidic organ-on-a-chip systems:** There is a growing need for developing *in vitro* systems that can better mimic the physiological responses of *in vivo* systems, with the ultimate goal of replacing or supplementing many cell/tissue or animal models used in mechanistic studies of diseases, as well as high-throughput screening for therapeutics development and toxicity testing. Within this area of organ-on-a-chip, we have pioneered the development of central nervous system (CNS) models of developing brain. We have for the first time demonstrated *in vitro* myelination of CNS neurons in a microchip format using a neuron-glia co-culture platform and a neural cell aggregate (i.e., organoid) platform utilizing primary cells, which are now being used for screening candidate drug molecules. We have also developed a multi-channel blood-brain barrier (BBB)-on-a-chip system that allows us to conduct 16 independent experiments to be conducted in parallel and also monitor the condition of all 16 BBB compartments using integrated impedance sensing electrode arrays (manuscript under preparation). We are also experienced in various co-culture platforms involving not only mammalian cell co-culture, but also bacterial cell-mammalian cell co-culture systems that are being used in gut intestinal (GI) tract model. These experiences will contribute to developing the proposed salivary gland MPSs.

2. Experience in developing high-throughput microfluidic live-cell array screening systems: Microfluidic live-cell array systems have arrays of pico-liter microchambers connected through arrays of fluidic delivery channels and control channels. Our main contribution is in developing highly robust and simple-to-use microfluidic screening systems that can be used for fully automated multi-day assays. We have demonstrated the use of an axon guiding cell culture platform for drug screening applications. We have also demonstrated the use of such robust systems for understanding the effect of environmental toxins such as polycyclic aromatic hydrocarbon (PAH) and drugs through multi-day fully automated assays. We have also pioneered the development of microfluidic systems for microorganism studies, especially those involved in bioenergy production. These multi-layer microfluidic systems we developed allowed multi-week assay of microorganisms, demonstrating the robustness of the microfluidic systems we develop. These experiences of both developing and utilizing microfluidic systems will contribute to validating the proposed MPSs as well as utilizing those systems for medium-throughput pilot-scale drug screening.


3. Experience in single-cell manipulation, cell patterning, and 3D cell culture of mammalian cells and bacterial cells in microchip format: We have extensive experience in utilizing diverse ranges of cells, both mammalian and bacterial cells, and have experiences in manipulating, patterning, and culturing these cells in various formats on microfluidic chips (e.g., single-cell manipulation, co-culture, cell aggregate formation/culture, suspension vs adherent cell culture, primary vs. cell line culture). These experiences will contribute to being able to handle the various cell types needed in the proposed MPS, as well as handling diverse ranges of biomolecules and toxicants for the required screening assays on MPSs.


4. Experience in developing microfabrication methods that enable low-cost microchips: Finally, developing such microfluidic lab-on-a-chip/organ-on-a-chip systems require new microfabrication methods development. Our main contribution is in developing methods that allow low-cost production of microchips beyond the prototype stage. This new method allowed us to fabricate microfluidic cell/tissue-chips (specifically, the brain-on-a-chip device) routinely in the hundreds of devices, a manufacturing throughput
needed during extensive microchip performance testing. This experience will contribute to the understanding of challenges faced with MPS fabrication, understand the device-to-device variability, and ultimately contributing to developing better designs and fabrication methods for tissue-chip systems.


D. Additional Information: Research Support and/or Scholastic Performance

**Ongoing Research Support**

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<td>Rusyn (PI)</td>
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<td>National Institute of Health (NIH)/National Center for Advancing Translational Sciences (NCATS) TEX-VAL: Texas A&amp;M Tissue Chip Validation Center</td>
<td></td>
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</tr>
<tr>
<td>This project will establish a facility to test and validate tissue chips to replace animal testing in chemical toxicology studies and drug testing. The new center will test reference compounds in up to 12 tissue chips to evaluate chips’ functionality, reproducibility, robustness and reliability; manage data to assure chips’ quality and develop a database for storage and sharing; and work with U.S. and European agencies to make tissue chip testing useful for regulatory decisions.</td>
<td></td>
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</tr>
<tr>
<td>Role: Co-PI</td>
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</tbody>
</table>

| NPRP9-001-2-001   | Sultan (PI)           | 12/1/2016 – 11/31/2019 |
| Qatar National Research Foundation (QNRF) |
| This project is to develop and use a droplet microfluidics-based high-throughput screening system for antimicrobial drug discovery. |
| Role: Co-PI       |

| NPRP9-052-2-020   | Han (PI)              | 6/1/2016 – 5/31/2019 |
| Qatar National Research Foundation (QNRF) |
| Air Pollution Cleanup Based Water Energy Food Nexus Technology |
| This project is to convert air-polluting gases into potential chemical seed solutions that are able to trigger development of integrated technology for production of energy and cultivation of agricultural plants or crops through desalination of saline water. |
| Role: Co-PI       |

| EFRI 1615202      | Han (PI)              | 3/01/2016 – 07/31/2017 |
| National Science Foundation (NSF) |
| Emerging Frontiers in Research and Innovation (EFRI) Research Experience and Mentoring Grant #3 |
| This is an educational and outreach grant providing multidisciplinary exposures to high school and undergraduate students, STEM teachers, as well as providing mentoring opportunities for graduate students and postdoctoral researchers. Third year supplement. |
| Role: PI          |

| 1R21EB021005-01   | Han (PI)              | 9/15/2015 – 6/30/2017 |
| National Institutes of Health (NIH) / National Institute of Biomedical Imaging and Bioengineering (NIBIB) |
| A High-Throughput Microfluidic in vitro CNS Myelination Model towards Drug Screening |
| This project is to develop and utilize a microfluidic brain-on-a-chip platform for high-throughput screening of drug candidates against neurodegenerative diseases. |
| Role: PI          |

| DBI 1532188       | Yakovlev (PI)        | 8/15/2015 – 8/14/2018 |
| National Science Foundation |
| Texas A&M Institute for Neuroscience |
MRI: Development of a Microfluidic Flow Cytometer for High-Throughput Noninvasive Single-cell Physio-Chemical Analysis

This project is to develop an integrated microfluidic flow cytometer system with integrated Raman spectroscopy for analyzing single-cell biophysical and chemical properties.
Role: Co-PI

EFRI 1519008    Han (PI)    5/01/2015 – 4/30/2017
National Science Foundation (NSF)
Emerging Frontiers in Research and Innovation (EFRI) Research Experience and Mentoring Grant #2
This is an educational and outreach grant providing multidisciplinary exposures to high school and undergraduate students, STEM teachers, as well as providing mentoring opportunities for graduate students and postdoctoral researchers. Second year supplement.
Role: PI

NPRP7-1634-2-604    Han (PI)    11/1/2014 – 10/31/2017
Qatar National Research Foundation (QNRF)
Self-sustainable and Highly Efficient Desalination System based on Microbe-Nanostructure Hybrids
This project is to develop a microbial desalination cell based on highly efficient and low-cost nanomaterial-anodes/cathodes for seawater desalination.
Role: PI

DBI 1353759    Han (PI)    5/15/2014 – 4/30/2017 (no-cost extension)
National Science Foundation (NSF)
IDBR: TYPE A – Microfluidic Fungal Transformation System for Ultra High-Throughput Functional Genomics
This project is to develop a microfluidic high throughput fungal transformation system that will allow high-throughput functional genomics.
Role: PI

EFRI 1240478    Han (PI)    8/15/2012 – 7/31/2017 (no-cost extension)
National Science Foundation (NSF)
EFRI-PSBR: Microalgae Lab-on-Chip Photobioreactor Platform for Genetic Screening and Metabolic Analysis Leading to Scalable Biofuel Production
This multi-disciplinary project is to develop microfluidic lab-on-chip devices with capabilities to precisely assay and manipulate parallel samples at single-cell resolution and utilize it to analyze and optimize the growth and hydrocarbon production potential of an engineered recombinant photosynthetic microalgae.
Role: PI

Completed Research Support from Past Three Years

NPRP 5-671-2-278    Sadr (PI)    10/15/2012 – 10/15/2015
Qatar National Research Foundation (QNRF)
Microfluidic Platforms for High-Throughput Screening of Microbes Utilizing Wastewater
This project is to develop a high throughput microbial electrolysis cell array for screening microbes and conditions that maximizes hydrogen production from wastewater.
Role: PI

ECCS 1232251    Kim (PI)    8/1/2012 – 7/30/2015
National Science Foundation (NSF)
Multi-Frequency Multi-Parametric Acoustophoretic Microfluidic System for Particle and Cell Separation
Role: Co-PI

OPP1058695    de Figueiredo (PI)    5/1/2012 – 10/31/2014
Bill and Melinda Gates Foundation
Defeating Antibiotic Resistance Before It Emerges
Role: Co-PI
NAME: Hardin, Paul Eric

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

POSITION TITLE: Distinguished 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.)

<table>
<thead>
<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
<th>Completion Date MM/YYYY</th>
<th>FIELD OF STUDY</th>
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<tbody>
<tr>
<td>Southern Methodist University</td>
<td>B.S.</td>
<td>05/1982</td>
<td>Biology</td>
</tr>
<tr>
<td>Indiana University</td>
<td>Ph.D.</td>
<td>05/1987</td>
<td>Genetics</td>
</tr>
<tr>
<td>Brandeis University</td>
<td>Postdoctoral</td>
<td>08/1991</td>
<td>Neurogenetics</td>
</tr>
</tbody>
</table>

A. Personal Statement

I have >25 years experience using *Drosophila* as a model system for molecular clocks research. As a post-doctoral fellow and head of my own lab I discovered that the *Drosophila* circadian timekeeping mechanism is based on cell-autonomous transcriptional feedback loops. These feedback loops are highly conserved, and now serve as the molecular basis for circadian timekeeping in all animals including humans. I have successfully administered multiple research projects, set up successful collaborations, and produced numerous peer reviewed research publications and invited reviews. My track record of research accomplishments has led to several honors including the Aschoff-Honma Prize for contributions to the field of biological clocks in 2002, the John W. Lyons Jr. ’59 Endowed Chair in Biology at Texas A&M University in 2005 and a Distinguished Professorship in Biology at Texas A&M University in 2008.

B. Positions and Honors

**Positions and Employment**

1991-1995  Assistant Professor, Department of Biology and the Center for Advanced Invertebrate Molecular Sciences, Texas A&M University.
1995-2000  Associate Professor, Department of Biology and Biochemistry, University of Houston.
2000-2005  Professor, Department of Biology and Biochemistry, University of Houston.
2004-2005  John and Rebecca Moores Professor, Department of Biology and Biochemistry, University of Houston.
1996-2005  Adjunct Professor, Department of Biology, Texas A&M University.
2005-2008  John W. Lyons ’59 Chair and Professor, Department of Biology, Texas A&M University.
2005-2008  Adjunct Professor, Department of Biology and Biochemistry, University of Houston.
2006-2009  Member, Faculty of Genetics, Texas A&M University.
2006-2009  Member, Texas A&M Institute of Neuroscience, Texas A&M University.
2008-2008  John W. Lyons Jr. ’59 Chair and Distinguished Professor, Department of Biology, Texas A&M University.

**Other Experience and Professional Memberships**

Texas A&M Institute for Neuroscience 347
C. Contribution to Science

1. **The discovery of transcriptional feedback loops.** My first contribution to the circadian clocks field occurred as a postdoctoral fellow studying the molecular basis of circadian timekeeping in the laboratory of Michael Rosbash. At the time we knew that mutations in the *period (per)* gene from *Drosophila* altered the period of or eliminated rhythms in activity, but studies characterizing *per* developmental and spatial expression and the PER protein sequence didn’t provide insight into how this gene contributed to circadian timekeeping. I tested the hypothesis that *per* mRNA was rhythmically expressed over the course of a day, and found that this was indeed the case. Importantly, the period of *per* mRNA rhythms were shorter or longer in the corresponding *per* mutants, indicating that PER protein feeds back to control the pace of *per* mRNA cycling. I then discovered that PER-dependent feedback controls *per* mRNA cycling at the transcriptional level, and revealed conserved E-box regulatory elements that drive rhythmic transcription. These studies revealed the transcriptional feedback loop concept to the field as a mechanism for circadian timekeeping. Similar transcriptional feedback loops have been found in fungi, plants and other animals, and understanding how these feedback loops keep circadian time, entrain to daily environmental cues, and control overt rhythms in physiology, metabolism and behavior remains a major focus of molecular clock research. I spearheaded the experimental design and conducted all experiments for the first two papers, and was the corresponding author for the other papers.


2. **The discovery of interlocked feedback loops.** The transcriptional feedback loop controls *per* and other genes whose transcripts peak near dusk during a daily cycle. However, the identification of *per* activators *Clock (Clk)* and *cycle (cyc)* revealed that *Clk* mRNA cycles with a peak near dawn in antiphase to *per*. While investigating *Clk* mRNA cycling, we discovered that the *Drosophila* circadian oscillator is comprised...
or two interlocked feedback loops: the previously identified core loop in which CLK and CYC activate and PER and its partner TIMELESS (TIM) repress rhythmic RNA expression that peaks near dusk, and an interlocked loop in which CLK and CYC repress and PER and TIM activate rhythmic RNA expression that peaks near dawn. Subsequent work in my lab and in collaboration with others showed that this interlocked loop is directly regulated by the transcriptional repressor VRILLE (VRI) and activated by PAR DOMAIN PROTEIN 1ε/δ (PDP1ε/δ) and an uncharacterized constitutive activator. Interlocked feedback loops are a well-conserved feature of animal circadian clocks, and their regulation and impact on circadian timekeeping is an important focus in the field. I served as corresponding author on all but the last paper listed below, which was a collaboration.


3. The discovery of rhythms in chemosensory physiology. Circadian oscillators are present in many Drosophila tissues, but which outputs they control and how they control them is a mystery. The antenna is the fly’s primary olfactory organ, and also contains robust molecular circadian oscillators. My lab collaborated with that of Stuart Dryer to show that these oscillators control robust rhythms in the amplitude of electrophysiological responses to different classes of odorants. This was the first demonstration that clocks in peripheral tissues are necessary and sufficient to control a physiological activity. Subsequent analysis in my lab defined key elements of the pathway the clock uses to control olfactory sensitivity. My lab expanded these studies to show that molecular clocks in the proboscis control rhythms in gustatory physiology through the same pathway used by the olfactory system. Future studies in this area promise to characterize the first complete output pathway from molecular oscillator to physiological rhythm. I served as the primary investigator in all but the first paper, where I was a co-investigator.


4. The discovery of PER-dependent rhythms in CLOCK phosphorylation, DNA binding and chromatin modifications. The pace of circadian rhythms is largely controlled via post-transcriptional regulation of clock proteins that drive the core transcriptional feedback loop. While studying CLK-dependent activation within the core feedback loop, we discovered that CLK protein phosphorylation cycles in abundance with a peak during transcriptional repression. Subsequent studies in my lab showed that PER repression complex binding promotes CLK phosphorylation via DBT kinase, releases CLK activation complexes from E-boxes, and decreases activating chromatin modifications at the per and tim loci to repress transcription. Recent collaborative work showed that CLK phosphorylation functions to lengthen circadian period, but is not required to repress transcription. The regulation and function of rhythms in CLK phosphorylation are now being studied in multiple species from Drosophila to mice. I served as the primary investigator in all but the last paper, where I was a co-investigator.


Complete List of Published Work in MyBibliography:
http://www.ncbi.nlm.nih.gov/sites/myncbi/1FAlAibQn705h/bibliography/47404002/public/?sort=date&direction=ascending

D. Research Support

Ongoing Research Support

5R21NS094807-01 Hardin (PI) 08/15/15 - 07/31/17
Circadian clock activation and tissue specificity in *Drosophila*
The goals of this study are to determine how CLOCK-CYCLE (CLK-CYC) heterodimers initiate circadian oscillator function in normal and ectopic cells and tissues in Drosophila.
Role: PI

Completed Research Support (last three years)

5R01 NS052854-09 Hardin (PI) 08/16/10 - 07/31/16
Regulation of Circadian Transcription
The goals of this project are to determine how PER-TIM-DBT complexes repress CLK-CYC transcription and how CLK-CYC switches from the repressed to the activated state by identifying and characterizing kinases and phosphatases that regulate CLK phosphorylation.
Role: PI
NAME: Harlow, Mark Lee

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

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.)

<table>
<thead>
<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
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<th>FIELD OF STUDY</th>
</tr>
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<tbody>
<tr>
<td>Texas A&amp;M University, College Station, TX</td>
<td>Dual B.S.</td>
<td>1990-1994</td>
<td>Biochemistry, Genetics</td>
</tr>
<tr>
<td>Stanford University, Stanford, CA</td>
<td>Ph.D.</td>
<td>1994-2001</td>
<td>Neuroscience</td>
</tr>
<tr>
<td>Stanford University, Stanford, CA</td>
<td>Post-doc</td>
<td>2001-2007</td>
<td>Neuroscience</td>
</tr>
</tbody>
</table>

A. PERSONAL STATEMENT

The aim of this proposal is to investigate the role of inflammation in Sarcopenia and Duchenne Muscular Dystrophy (DMD). In particular, we propose to study how the classically activated innate immune system (M1) alters the morphology of the neuromuscular junction. Terminal Schwann cells (TSCs) are non-myelinating glia that cap axon terminals at the NMJ. Work largely from the Thompson lab has demonstrated that TSCs play important roles in synapse regeneration following injury in the young adult, and has recently suggested that TSCs actively participate in neuromuscular synapse elimination and maturation in the neonate. Here I propose that increased proliferation and activation of TSCs, promoted by the activation of the M1 inflammation pathway, may underlie the morphological changes in both aged and dystrophic NMJs. Throughout my career, I have used the NMJ as model synapse to study basic mechanisms of neurotransmitter release and the structural organization of macromolecules at the active zone and within synaptic vesicles. I have made important and valued contributions to the field. Specifically I have helped develop the field of electron tomography, and contributed to the understanding of the organization of macromolecules at the neuromuscular junction. My contributions to electron tomography include the development and testing of algorithms for the denoising and signal analysis of electron tomographic tissue volumes. The development and testing of alignment algorithms for electron tomographic volume reconstruction, as well as the development and testing of tools used in the analysis of electron tomographic reconstructions. Using these tools and approaches I have made significant contributions toward understanding how macromolecules such as ion channels are organized, and synaptic vesicles are docked, at the active zone release site. Furthermore I have demonstrated that synaptic vesicles themselves possess an organized macromolecular structure that, in the process of docking with the macromolecules at the active zone, must become oriented properly. My work as an independent investigator has dealt with the macromolecular description of synaptic vesicles and synaptic vesicle contents – using single vesicle imaging techniques at both the light and EM level, as well classic biochemical techniques. In the past year I have begun a collaboration with the Thompson lab looking at how NMJs age. We believe our findings are not only relevant to Sarcopenia, but also to muscular dystrophies, such as DMD, and we look forward to extending our preliminary findings on the innate immune pathways that induce damage on muscle fibers and NMJs.
B. POSITIONS AND HONORS

Positions and Employment
2007    Co-Instructor, Biol 170; instructor BIOL 170L (Undergraduate Neurobiology), UC Merced.
2007-09  Staff Scientist, Department of Neurobiology, Stanford University School of Med., Stanford, CA.
2009-    Assistant Professor of Biology, Texas A&M University, College Station, TX.

Other Experience and Professional Memberships
1995-    Member, Society for Neuroscience
2009-    Member, Biophysical Society
2009-    Member, Training Faculty, Texas A&M Institute for Neuroscience (TAMIN)
2014    Ad hoc reviewer, NSF Career Awards

Honors
2006    1st Place Winner (Postdoc Category) Poster Presentation at the 2006 Neuroscience Institute at Stanford Research Conference at Asilomar.

C. Contribution to Science

1. I have focused much of my career on understanding synaptic transmission at the cholinergic Neuromuscular junction. My work as an independent investigator has dealt with the macromolecular description of synaptic vesicles and synaptic vesicle contents – using single vesicle imaging techniques at both the light and EM level, as well classic biochemical techniques. My lab’s recent research is the investigation of a novel form of communication between neurons that could be an important mechanism at the synapse to control local protein synthesis. The organism used in these studies has been the electric ray, Torpedo californica. Our published findings indicate that synaptic vesicles, isolated and purified from both the PNS and CNS, contain two classes of sRNAs: microRNAs (miRNAs) known to function in RNA silencing and post-transcriptional regulation of gene expression, and transfer-RNA fragments, shown to be important for cellular stress response as well as post-transcriptional regulation of gene expression in a non-miRNA dependent pathway. I have assembled a research team complementary to my own that includes three co-collaborators (Drs. Aramayo, Sachs, and Thompson) who provide essential expertise in bioinformatics, and post-transcriptional regulation, and synapse physiology. I am currently finishing a transcriptome study of the electric lobe of Torpedo californica with my colleagues Aramayo and Sachs that was delayed due to the death of a graduate student in my laboratory. This study provides a strong set of proteomic tools for the in silico analysis of proteins associated with the SV sRNAs at the electric organ.


2. Throughout my career, I have studied the basic mechanisms of neurotransmitter release and the structural organization of macromolecules at the active zone and within synaptic vesicles. I have made important and valued contributions to the field of electron tomography, and contributed to the understanding of the organization of macromolecules at the neuromuscular junction. Furthermore I have demonstrated that synaptic vesicles themselves possess an organized macromolecular structure that, in the process of docking with the macromolecules at the active zone, must become oriented properly. This specific contribution, published in 2013, was research that I continued after starting my own independent research at Texas A&M University.


3. My contributions to electron tomography include the development and testing of algorithms for the denoising and signal analysis of electron tomographic tissue volumes. The development and testing of alignment algorithms for electron tomographic volume reconstruction, as well as the development and testing of tools used in the analysis of electron tomographic reconstructions. Using these tools and approaches I have made significant contributions toward understanding how macromolecules such as ion channels are organized, and synaptic vesicles are docked, at the active zone release site.


Complete List of Published Work in MyBibliography:

D. RESEARCH SUPPORT

Ongoing Research Support:
Departmental Startup Funds, Texas A&M University Harlow (PI) 2/1/2009-
Research Initiation Funds
The purpose of these funds is to set up the PI's laboratory and fund preliminary studies needed to be competitive for extramural research support.
Role: PI

Research Support Completed during the Last Three Years
None
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: MICHELLE A. HOOK

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

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.)

<table>
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<td>University of New England, Armidale, Australia</td>
<td>Ph.D.</td>
<td>1998</td>
<td>Physiology</td>
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<td>University of Memphis, Memphis, TN</td>
<td>Post-doctoral</td>
<td>1997-1999</td>
<td>Psychology</td>
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<tr>
<td>UTMDACC, Bastrop, Texas</td>
<td>Post-doctoral</td>
<td>1999-2000</td>
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</tr>
<tr>
<td>Texas A&amp;M University</td>
<td>Post-doctoral</td>
<td>2002-2004</td>
<td>Psychology</td>
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</table>

Please refer to the Biographical Sketch sample in order to complete sections A, B, C, and D of the Biographical Sketch.

A. PERSONAL STATEMENT

I have been studying behavior in laboratory animals for more than 20 years, and specifically studying neurotrauma (the spinal contusion model of injury) for 15 years. I have a book chapter, 32 peer-reviewed papers, and 13 funded (4 current, 9 completed) grants on this topic. The primary focus of my research has been on recovery of function after injury. As part of this research, I have conducted pioneering studies of addiction after injury, which stemmed further interest in co-morbid conditions such as depression and pain. Most of my training has been in psychology departments, and I am constantly reminded of the salience of psychological well-being and its potential role in recovery. As a result, I have developed a behavioral ethogram for the assessment of depression in the rodent contusion model. This model is unique as we behaviorally phenotype depressed subjects based on a cluster of symptoms (rather than simply comparing performances across treatment groups), an approach that is akin to diagnoses in the clinical population. Using this approach we are able to identify depressed subjects, independent of experimental treatment groups, and assess physiological and molecular changes associated with depression per se. I have collaborated with Dr. Sohrabji and her laboratory, for example, to assess serum, brain and spinal expression levels of pro-inflammatory cytokines in depressed and not depressed SCI rats. We have shown that a depressive-phenotype is associated with increased serum levels of pro-inflammatory cytokines, increased heart rates and decreased heart rate variability; mirroring the molecular and physiological changes associated with depression in humans. We will use this powerful model system to assess depression following stroke, in the submitted proposal. My laboratory is very experienced at performing the behavioral and molecular assays outlined. My experience is highlighted in the following publications.


B. POSITIONS AND HONORS

Professional Experience

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<th>Year</th>
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<tr>
<td>1999</td>
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<tr>
<td>2001</td>
<td>Part-time Assistant Professor</td>
<td>Southwestern University</td>
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<td>2001-2002</td>
<td>Project Coordinator</td>
<td>Georgia State University</td>
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<td>2004-2008</td>
<td>Assistant Research Scientist</td>
<td>Texas A&amp;M University</td>
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<tr>
<td>2008-2011</td>
<td>Assistant Research Professor</td>
<td>Texas A&amp;M University</td>
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<td>2011-2013</td>
<td>Associate Research Professor</td>
<td>Texas A&amp;M University</td>
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<td>2014-present</td>
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<td>Texas A&amp;M University</td>
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Honors and Awards

American Society of Primatologists General Small Grant (1999)
Del Duca Foundation Scholarship (France) for Postdoctoral Research (not taken). (1997)
Academic Women's Association Student Travel Scholarship (1997)
University of New England Research Scholarship (for graduate study) (1994)

Society Memberships:


C. CONTRIBUTION TO SCIENCE

1. SCI increases symptoms of depression in a rodent model. The incidence of depression is significantly increased in spinally injured patients, relative to the general population. We have now shown that depression-like symptoms are also increased in rodents with SCI, relative to intact controls. We developed and validated a method for characterizing depression in SCI rats, and have shown in preliminary studies that depression-like behavior is associated with non-subjective measures of physiological function (heart-rate and heart-rate variability) as well as elevated pro-inflammatory cytokine levels (Brakel et al. in prep). These innovative studies lay the foundation for a new area of research in neurotrauma models, and address a consequence of injury that significantly impacts quality of life and likely impacts successful rehabilitation. Understanding the molecular mechanisms underlying decreased psychological well-being will be important not only for SCI, but for a range of inflammatory based conditions including traumatic brain injury, stroke and multiple sclerosis.


2. Morphine undermines functional recovery after SCI. Morphine is one of the most frequently prescribed analgesics for the treatment of pain. We have shown, however, that irrespective of the route of administration morphine administered in the acute phase of a spinal cord injury significantly undermines recovery of locomotor function, decreases general health and produces symptoms of paradoxical pain. The adverse effects of morphine on locomotor recovery may be mediated, at least in part, by increased neuronal death with drug administration. We have begun to elucidate the molecular mechanisms underlying these adverse effects. Our data suggest that kappa-opioid receptors mediate the morphine-induced attenuation of recovery, and that these receptors may be located on glial cells following injury. We have shown that the adverse effects of morphine can be blocked by 1) an IL1 receptor antagonist, 2) a kappa-opioid receptor
antagonist and 3) Minocycline (Aceves et al. in prep). These data have significant clinical implications for pain management after SCI.


3. Addictive potential of morphine following spinal cord injury. Clinicians have significant concerns about addiction when prescribing drugs of abuse, such as opioids, as analgesics following injury. Despite these concerns, the potential for addiction had not been examined in animal models of SCI. We conducted pioneering studies on addiction, using the established self-administration and place preference paradigms, in the rodent contusion model. We showed that the potential for addiction appears to be attenuated, but not negated, in the acute phase of SCI. In the chronic stage of SCI, however, SCI subjects administer high amounts of morphine commensurate with their sham counterparts. The 20-30 mg of morphine, which is administered in less than 6 hours, far exceeds that needed for analgesia and is suggestive of addiction. Interestingly, morphine administration in the chronic phase of SCI (14+ days) does not undermine functional recovery. Comparisons of the windows of vulnerability (acute and chronic SCI) to the effects of morphine may provide information on critical molecular mediators of both addiction and the morphine-induced attenuation of locomotor recovery.


Complete List of Published Work in MyBibliography:

D. RESEARCH SUPPORT

COMPLETED

R21 NS081606 Role: Col (PI: Garraway) Dates funded: 7/1/2013-6/30/2015

Cellular mechanisms underlying pain following spinal cord injury

The experiments outlined within this grant explore the mechanisms that underlie nociception induced sensitization of pain circuits after a spinal contusion injury. The experiments assess the immediate and long-term effects of noxious input (peripheral inflammation) on the induction and maintenance of pain behaviors following SCI. At a molecular level, the studies identify the role TNFα signaling pathway plays in mediating nociceptive input-induced pain hypersensitivity following SCI

Overlap: None

RO1 DA031197 Role: PI Dates funded: 4/01/2011-3/31/2016 (1 year no cost extension)

Morphine undermines recovery of function after SCI: Neurobiological mechanisms

To improve the safety and analgesic efficacy of opioids used after SCI, the proposed experiments will 1) identify critical molecular changes that underlie morphine’s effects, 2) use pharmacological manipulations to block adverse effects (reduced recovery) at a spinal level, and potentiate morphine’s beneficial (analgesic) effects, and 3) identify cellular changes produced though activation of classic and/or non-classic opioid receptors.

Overlap: None
Learning Within the Spinal Cord: Clinical Implications

Using a spinal transection paradigm, we have previously shown that exposure to controllable shock fosters spinal cord plasticity. This grant examined the possibility that training with controllable stimulation enables learning because it causes an up-regulation in the synthesis and release of the neurotrophin BDNF. Experiments proposed used pharmacological techniques to assess both the necessity and sufficiency of BDNF. Cellular assays examined the impact of training on BDNF mRNA expression. We also examined whether instrumental training enhances recovery after spinal cord injury and if this effect was related to BDNF release.

Overlap: None.

Influence of environmental stimulation on learning and recovery after injury

The major goals of this project were to examine the types of nociceptive stimuli that influence recovery after a spinal cord injury and the role of brain systems.

Overlap: None.

Neurotrophin delivery using injectable hydrogels for increased plasticity after SCI

In collaboration with bioengineers (Z. Khaing and C. Schmidt) we were exploring a hydrogel based delivery system for the application of BDNF and other ligands after a contusion injury.

Overlap: None

CURRENT GRANTS

Department of Defense (CDMRP) Role: PI Dates funded: 6/1/2017-5/31/2020

Derivation of the Mechanisms Mediating the Adverse Effects of Morphine in a Rodent Model of SCI: Functional Recovery and Neuron Loss

These studies aim to identify the molecular mechanism underlying morphine’s effects on the immune response and cell death after SCI. We will compare the temporal expression and the functional activity (cytokine expression, ex vivo phagocytosis) of activated microglia in morphine and vehicle-treated SCI subjects. We will then test the efficacy of targeting microglia as a future therapeutic intervention, using hM4Di DREADDs.

Overlap: None

Gillson-Longenbaugh Foundation Role: PI Dates funded: 8/1/2015-7/31/2017

Psychological Wellbeing in a Rodent Model of Spinal Cord Injury

This study focuses on the role of inflammation in the development of depression after spinal cord injury (SCI). Specifically, we are testing the effectiveness of anti-inflammatory medications and evaluating changes in immune function at behavioral, physiological and molecular (spinal, peripheral and supraspinal) levels. These data, from a comparable model of neurotrauma, will inform the experiments and methodology applied in the current proposal.

Overlap: None

Mission Connect Role: co-PI Dates funded: 4/1/2016-3/31/2017

Functional Outcome Correlates in Acute Spinal Cord Injury related to Opioid Use

Using data collected from patients admitted to The Institute for Rehabilitation and Research (TIRR) Memorial Hermann that contains follow up information at 1 year after traumatic SCI, we will examine the relationship between opioid use in humans during acute SCI and functional outcomes, specifically with motor recovery, pain, and depression.

Overlap: None

Craig H. Neilson Foundation Role: Co (PI: Grau) Dates funded: 12/31/2014-12/30/2017

How and when does peripheral input affect recovery after SCI
This proposal examines how peripheral nociceptive stimulation (uncontrollable electrical stimulation) affects recovery of function and cell death in the rodent spinal contusion model.

Overlap: None.
BIOGRAPHICAL SKETCH

Provide the following information for the Senior/key personnel and other significant contributors in the order listed on Form Page 2. Follow this format for each person. **DO NOT EXCEED FOUR PAGES.**

<table>
<thead>
<tr>
<th>NAME</th>
<th>POSITION TITLE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Roozbeh Jafari</td>
<td>Associate Professor</td>
</tr>
</tbody>
</table>

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

rjafari

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

<table>
<thead>
<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
<th>MM/YY</th>
<th>FIELD OF STUDY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sharif University of Technology, Tehran, Iran</td>
<td>BS</td>
<td>9/95-1/00</td>
<td>Electrical Engineering</td>
</tr>
<tr>
<td>State University of New York, Buffalo, NY</td>
<td>MS</td>
<td>8/00-1/02</td>
<td>Electrical Engineering</td>
</tr>
<tr>
<td>University of California, Los Angeles</td>
<td>MS</td>
<td>1/02-8/04</td>
<td>Computer Science</td>
</tr>
<tr>
<td>University of California, Los Angeles</td>
<td>PhD</td>
<td>1/02-8/06</td>
<td>Computer Science</td>
</tr>
<tr>
<td>University of California, Berkeley</td>
<td>Post-doc</td>
<td>8/06-7/07</td>
<td>Electrical Eng. &amp; CS</td>
</tr>
</tbody>
</table>

**A. Personal Statement**

My research is primarily in the area of wearable computing with emphasis on medical/biological applications, their signal processing and algorithm design. I have worked on a variety of projects building wearable computers for motor function monitoring, Fall prevention and physiological monitoring using novel sensing and signal processing paradigms. Having in depth engineering knowledge and understanding the requirements of the clinical applications have been instrumental in the success of my research. I have been publishing extensively which illustrates the success of my earlier projects and have been supervising a large number of graduate and undergraduate students.

**B. Positions and Honors**

**Professional Position**

- 5/01-8/01 Engineering Intern IBM Corporation, Endicott, NY
- 6/07-8/13 Assistant Professor University of Texas at Dallas
- 9/13-8/15 Associate Professor University of Texas at Dallas
- 9/15-present Associate Professor Texas A&M University

**Honors**

- 2002 UCLA Graduate Division (Full) Fellowship for 2002-2003
- 2006 Best Teaching Assistant Award for 2005-2006, Computer Science Dept., UCLA
- 2011 Best Paper Award at Real-time Embedded Technology and Applications Symposium
- 2012 Junior Faculty Research Award 2012, School of Engineering and Computer Science, UTD
- 2012 NSF Faculty Early Career Development (CAREER) Award 2012
- 2014 Andrew P. Sage Best Transactions Paper Award from IEEE Systems, Man and Cybernetics Society

**C. Peer-Reviewed Publications (selected from over 100 articles available at [http://jafari.tamu.edu](http://jafari.tamu.edu)):**


D. Research Support

Ongoing Research Support

National Science Foundation (NSF), Role: PI
CAREER: Ultra Low Power Architectures for Wearable Computing Jafari (PI) 2012-2017

DARPA and SRC, Role: site-PI
The TerraSwarm Research Center (TSRC) Lee (PI) 2013-2017

TXMRC, Role: PI

Texas Instruments, $25,000, 9/14-9/15, Role: PI
Gesture Recognition using Wrist-worn EMG and Motion Sensors Jafari (PI) 2014-2015

Semiconductor Research Corporation (SRC), Role: PI
Non-Contact & Dry-Contact Reconfigurable Electroencephalography Jafari (PI) 2012-2015

National Institute of Health (NIH), Role: PI
Sway and Biofeedback for Fall Prevention Jafari (PI) 2011-2015

National Science Foundation (NSF), Role: co-PI
NetSE: Large: Collaborative Research: Exploiting Multi-modality for Tele-Immersion Jafari (PI) 2010-2015

Texas A&M Institute for Neuroscience 362
NAME: Sharon C. Kerwin

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

POSITION TITLE: Professor and Associate Department Head

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.)

<table>
<thead>
<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
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<th>FIELD OF STUDY</th>
</tr>
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<tbody>
<tr>
<td>Texas A&amp;M University</td>
<td>B.S.</td>
<td>05/1986</td>
<td>Veterinary Science</td>
</tr>
<tr>
<td>Texas A&amp;M University</td>
<td>D.V.M.</td>
<td>05/1988</td>
<td>Veterinary Medicine</td>
</tr>
<tr>
<td>Louisiana State University</td>
<td>Internship</td>
<td>07/1989</td>
<td>Small Animal Vet Med</td>
</tr>
<tr>
<td>Louisiana State University</td>
<td>Residency</td>
<td>07/1992</td>
<td>Small Animal Surgery</td>
</tr>
<tr>
<td>Texas A&amp;M University</td>
<td>Residency</td>
<td>07/2016</td>
<td>Veterinary Neurology</td>
</tr>
</tbody>
</table>

A. Personal Statement
I have been a practicing veterinary orthopedic and spine surgeon since 1992, with expertise in arthroscopy, trauma of the long bones and spine, angular limb deformity, total hip replacement and joint stabilization. From the research perspective, my experience has included biomechanics, bone grafting spinal cord injury and gait analysis. More recently, my board certification in neurology has allowed a more in-depth perspective on gait analysis, spinal biomechanics, and the considerable crossover that occurs between orthopedics and neurologic disease.

B. Positions, Services, and Honors:
Tom and Joan Read Chair in Veterinary Surgery, 2014-present
Professor and Associate Department Head, Department of Veterinary Small Animal Clinical Sciences, 2014-present
Interim Department Head, VSCS, 2014-2015

Clinical Licensure:
Texas, 1988-present

Board Certifications:
Diplomate American College of Veterinary Surgeons, 1994
Diplomate American College of Veterinary Internal Medicine (Neurology) 2016

Professional Memberships
1988 Member, American Veterinary Medical Association
1989 Member, Veterinary Orthopedic Society
2003 Member, AO Vet
2011 Founding Member, Veterinary Neurosurgical Society

Top 5 Publications:


**Current and Pending Support:**

Treatment of non-union long bone fractures with canine mesenchymal stem cells: a pilot study. Co-Principal Investigators: Gregory CA, Saunders WB. Co-Investigators: Krause U, Eichelberger B, Kerwin S, Dejardin L. Funded by departmental resource funds, Department of Cellular and Molecular Medicine, Texas A&M University Health Science Center, Temple, TX. Dates involved in project: 2013-present.


**Pending Support:**

NAME: Khosravian, Homa

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

POSITION TITLE: Research 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.)

<table>
<thead>
<tr>
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</tr>
</thead>
<tbody>
<tr>
<td>Sharif University of Technology, Tehran, Iran</td>
<td>B.S</td>
<td>07/2005</td>
<td>Chemical Engineering</td>
</tr>
<tr>
<td>Sharif University of Technology, Tehran, Iran</td>
<td>M.S</td>
<td>08/2007</td>
<td>Chemical Engineering</td>
</tr>
<tr>
<td>University of Illinois at Chicago, Chicago</td>
<td>PhD</td>
<td>08/2013</td>
<td>Chemical Engineering</td>
</tr>
<tr>
<td>University of Manchester, Manchester, UK</td>
<td>Postdoctoral</td>
<td>08//2014</td>
<td>Chemical Engineering</td>
</tr>
</tbody>
</table>

A. Personal Statement

I have a PhD in Chemical Engineering with an extensive training in nanoscience and nanoengineering, which is necessary to conduct the proposed research project. I am very interested in bioengineering research and my long-term goal is to use my nanotechnology background to develop new materials and methodologies that can be used in treatment (and/or early diagnosis) of CNS disorders (e.g. stroke, Alzheimer Disease) and thus reduce the burden associated with these diseases on aged population. I have the drive, passion, leadership skills, training, and commitment necessary to achieve this goal. My current research includes synthesis of nanomaterials, environmental health and safety of nanomaterials and heterogeneous catalysis. Currently, through collaboration with Professor Farida Sohrabji, I am working on developing new therapeutic molecule for treatment of ischemic stroke in its delayed phase by using the molecular building block of nanotechnology, diamndoids and their adamanty ligands.

B. Positions and Honors

Positions and Employment

2008-2013 Research/Teaching Assistant, Department of Chemical Engineering, University of Illinois at Chicago, Chicago, IL
2013-2014 Postdoctoral Research Associate, Department of Chemical Engineering and Analytical Science, The University of Manchester, Manchester, UK
2014-2015 Adjunct Professor, Kankakee Community College, Kankakee, IL
2015- Research Assistant Professor, Department of Chemical Engineering, Texas A&M University, College Station, TX
2016- Faculty, Texas A&M Institute for Neuroscience, Texas A&M University, College Station, TX

Other Experience and Professional Memberships

2008- Member of North American Catalysis Society
2008-2015 Member-American Institute of Chemical Engineers
2009- Member of American Vacuum Society
2016 Symposium Chair: Engineering and Health Science Symposium Series.
2016- Senior member-American Institute of Chemical Engineers
2017 Session co-chair, Science and Engineering of Catalyst Preparation, 2017 AIChE annual meeting.

Honors and Professional Service

Ad-hoc Reviewer for American Chemical Society (2016)
National Science Foundation CMMI panel reviewer (2016)
C. Contribution to Science

1. Controlled synthesis of nanomaterials on metal oxide surfaces:
Nanomaterials are vital in vast number of processes in chemical industry. Their applications have significant impact on our daily lives which includes: Energy processing, food processing, pharmaceuticals, bulk chemicals, fine chemicals, and environment. Formation of supported nanoclusters in the order of 10 atoms represents an exciting opportunity for new forms of applications (e.g., heterogeneous catalysts). My publications directly address new methods to prepare such nanoparticles with controlled size distribution. This research background assists me in developing novel nanomaterials for the proposed research.


2. Surface chemistry studies of nanoclusters assembly on graphene, a 2D material:
Since 2004, graphene has attracted widespread attention in numerous research areas when it was shown to be surprisingly stable with only one layer thickness even at room temperatur. This was substantiated in 2010, when Geim and Novoselov were awarded the Nobel Prize in Physics “for groundbreaking experiments regarding the two-dimensional material graphene”. The special structural, thermal, mechanical, chemical and electronic properties of graphene grabbed extensive attention for future numerous applications (i.e. electronic devices). Graphene is a fascinating supporting material in catalysis and model catalysis studies because of its high chemical and thermal stability. One of the most important issues in graphene preparation is the quality and the thickness of the film. Continuous, thinner and larger graphene sheets are considered to have higher quality than smaller, thicker graphene island with discontinuity. Therefore, graphene preparation methods are a great challenge for researchers. Epitaxial growth of graphene on a metal substrate by employing surface segregation of carbon atoms or, hydrocarbon dissociation at elevated temperature is one of the promising approaches in graphene preparation. In this research nanomaterials were prepared and deposited on graphene support in an effort to understand the underlying factors that have effect on structure-reactivity of catalyst, which eventually helps us for rational design of catalysts.

- Khosravian H, Formation of Rhodium Nanoclusters Fabricated from Rh(CO)2(acac) on Graphene/Cu(111)”, AIChe Annual Meeting, San Francisco, CA (2016).

3. Enhanced oil recovery:
On average, only 40% of crude oil can be recovered from an oil field with current oil production infrastructure. This, unfortunately, results in leaving behind almost 60% of the oil. To increase the efficiency of oil recovery, it is crucial to have a clear understanding of sub surface characteristics and how transport of oil in soil can be more efficient. With Enhanced Oil Recovery methods, which includes using nanomaterials, surfactants etc that enhance the interaction between oil and the reservoir, the amount of recovered oil is increased to 60%. In this...
respect, the effect of flow history on residual oil saturation during two-phase flow in porous media was
investigated. This research is important for many environmental and engineering applications, such as
secondary oil recovery or designing efficient remediation schemes for the contaminated sites by petroleum-
based products.

- Khosravian H, Joekar-Niasar V, Shokri N, “Effects of Flow History on Oil Entrapment in Porous Media:
  An Experimental Study”, Transport Phenomena and Fluid Mechanics, AIChE Journal, Vol. 61(4),

4. **Platinum group metals (PGMs) recovery and environmental remediation:**
Access to clean, affordable and reliable energy is the key to a sustainable growth. The process of harvesting
non-renewable energy sources results in a degraded environment (air, water, and soil pollution). One way to
prevent the negative environmental impacts of using non-renewable energy is to promote the use of renewable
energy. Unfortunately, the current capacity for producing energy from renewable sources is extremely limited
and more than 80% of today’s energy needs are addressed through non-renewable energy sources. Therefore,
the non-renewable energy technologies should incorporate environmentally friendly production methods and
renewable energy technologies should provide higher capacity to address our energy needs. One aspect of my
research focuses on platinum recovery from spent catalysts. In short, my research is about kinetic study of
platinum extraction from spent reforming catalysts as recycling of the precious metals bearing catalysts is
crucially important due to: a) high value of the precious metals and b) the environmental considerations.

- Baghalha M, Khosravian H, Mortaheb H, “Kinetics of platinum extraction from spent reforming

**D. Additional Information: Research Support and/or Scholastic Performance**

**Ongoing Research Support**

Start-up fund, Texas A&M University 08/31/15-present
Role: PI
W. R. Klemm

Current Position: Senior Professor of Neuroscience, Texas A&M University
Education: D.V.M., Auburn University; Ph.D. (“Distinguished alumnus”); Univ. of Notre Dame

Experience Directly Relevant to Proposed Project

Dr. Klemm is an experienced scientist, with over 550 publications, over 230 of which were in peer-reviewed scholarly journals. He was received numerous awards (see below), selected to the Editorial Boards of seven research journals as well as six science-education journals, and has conducted official peer reviews on ~800 manuscripts for 38 scholarly journals.

He now specializes in science writing. His writing and speaking credentials can be seen at http://thankyoubrain.com. His works include 20 books, 54 book chapters, and hundreds of non-technical articles and blog posts (which have over two million reader views).

Research Interests Statement

Education research, “neuro-education:” brain-based education, educational technology, on-line learning, collaborative learning, constructivism, learning and memory strategies and tactics. He leads a neuro-education discussion on Linked-In.

Neuroscience: human cognition (EEG coherence, free will, dreaming), animal behavior (brainstem mechanisms, alcohol and substance abuse, biological water). His recent books include The Learning Skills Cycle (Rowman & Littlefield), Making a Scientific Case for Conscious Agency and Free Will (Academic Press), Mental Biology (Prometheus/Random House). Memory Power 101 (Skyhorse) Atoms of Mind (Springer), and Core Ideas in Neuroscience (Benecton).

Selected Awards and Recognitions

2. “Distinguished Member” award, Sigma Xi.
3. “Distinguished Lecturer,” Sigma Xi.
4. “Distinguished Alumnus,” Auburn University College of Veterinary Medicine
5. Board of Directors (three terms), Assoc. Director, Research & Doctoral Univ., Director, Southwest Region, Sigma Xi.
6. President, AAAS, Southwest and Rocky Mountain Division
   Ad Hoc Reviewer: 38 journals, 8 book publishers, NSF, NIH, USDA.
8. Biographical Listings: 18, including Marquis’ Who’s Who in America, Who’s Who in the World
Ongoing Research Support: none

Completed Research Support (educational outreach)


**Science Promotion in rural Public Schools**, NIH/SEPA, Co-PI and Project Manager. Project Dates: 9/01/07 to 8/31/2012; Total Funded Amount: $1,352,000. Personnel: Larry Johnson, PI, W. R. Klemm, Co-PI and Program Manager

Selected Publications (neuroscience)


**Selected Publications (education)**


Klemm, W. R. 2013. Teaching Beginning College Students with Adapted Published Research Reports. J. Effective Teaching. 13 (2), 6-20.


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:** Ko, Gladys Yi-Ping

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

**POSITION TITLE:** Associate Professor, Veterinary Integrative Biosciences, Texas A&M University

**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.)*

<table>
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<tr>
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<th>Completion Date MM/YYYY</th>
<th>FIELD OF STUDY</th>
</tr>
</thead>
<tbody>
<tr>
<td>National Cheng-Kung University, Taiwan.</td>
<td>B.S.</td>
<td>07/1989</td>
<td>Biology</td>
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<tr>
<td>National Yang-Ming Medical College, Taiwan.</td>
<td>M.S.</td>
<td>07/1991</td>
<td>Anatomical Sciences</td>
</tr>
<tr>
<td>Kent State University affiliated with Northeastern Ohio Universities College of Medicine, Kent, OH. Dept. Neurobiology and Anatomy, University of Texas-Houston Medical School, Houston, TX. Dept. Biology and Biochemistry, University of Houston, Houston, TX.</td>
<td>Ph.D.</td>
<td>08/1996</td>
<td>Biomedical Sciences</td>
</tr>
<tr>
<td></td>
<td>Postdoc</td>
<td>02/1999</td>
<td>Neuroscience</td>
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<tr>
<td></td>
<td>Postdoc</td>
<td>07/2004</td>
<td>Neuroscience / Circadian Biology</td>
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**A. Personal Statement**

My lab focuses on retinal physiology in healthy and diabetic states, and how we can develop early detection protocols to prevent or deter the pathological development of diabetic retinopathy (DR). Diabetic retinopathy is a dual disorder with characterized vascular complications and neural degeneration, even though clinically, it is diagnosed and treated as a vascular disease. We have recently adopted a diet-induced obesity/diabetic mouse model to understand the physiological changes in both neural and vascular retina especially during the early on-set of DR pathogenesis. We found that mice under a high-fat-diet (HFD, 59.4% fat calories) for only 1 month have decreased neural retinal light responses compared to mice fed a normal diet (10% fat calories) in the absence of vasculogenesis (*IOVS* 2017, 58:106-118), even though the blood glucose levels of these HFD-mice are at the same level as the mice with normal diet. By 3 months of HFD, the HFD-mice are hyperglycemic with insulin-resistance and glucose-intolerance, and their neural retinal light responses are worsened (*IOVS* 2015, 56:2367-2380; *IOVS* 2017, 58:106-118), but we did not detect any microvascular changes until 5-6 months after HFD. These observations provide evidence that the neural retina is compromised ahead of vascular complications when animals are under chronic diabetic stress. By 6-7 months of HFD, these HFD-mice have developed retinal microvascular complications and neovascularization as manifested in human DR, including increased vasculogenesis, acellular capillaries, and “microaneurysm-like” structures (*PLOS ONE* 2016, 11 (6):e0157543; *IOVS* 2017, 58:106-118). Upon further analyses, we found that in HFD-induced early diabetic eyes, there is increased retinal and intra-ocular inflammation, a hallmark of diabetes (*IOVS* 2017, 58:106-118). We also found that in HFD-induced diabetic mice, microRNA-150 (miR-150) is significantly decreased in blood circulation (*PLOS ONE* 2016, 11 (6):e0157543) and in the retina (preliminary data). Deletion of miR-150 (miR-150−/−) further exacerbates HFD-induced DR vascular pathology in part by up-regulation of vascular endothelial growth factor receptor 2 (VEGFR2) in the retinal vasculature compared to the wild type (WT)-HFD mice (*PLOS ONE* 2016, 11 (6):e0157543). However, the gene encoding VEGFR2 (*KDR*) is not a direct target of miR-150, even though overexpression of miR-150 in retinal endothelial cells suppresses the protein expression of VEGFR2 (*PLOS ONE* 2016, 11 (6):e0157543). We have extensive experience in determining the direct targets of microRNAs (*JBC* 2009, 284: 25791-25803). In collaboration with our long-term collaborator Dr. Beiyan Zhou (U. Conn; *IOVS* 2015, 56:2367-2380; *PLOS ONE* 2016, 11 (6):e0157543), we will use tissue-specific miR-150 knockouts and knock-ins to determine how retinal miR-150
contributes to the pathogenesis of DR. We will further determine the signaling from miR-150 → VEGFR2 in this application.

**Publications/Research Products Most Relevant to This Proposal**


**B. Positions and Honors**

**Positions and Employment**

- **1991 - 1992** Research Assistant, Dr. Hwa-Min Hwang’s Laboratory, Department of Anatomy, Chung Gung Medical College, Taoyuan, Taiwan

- **1996 - 1999** Postdoctoral Research Fellow, Dr. Paul Kelly’s Laboratory, Department of Neurobiology and Anatomy, University of Texas-Houston Medical School, Houston, TX

- **1999 - 2000** Postdoctoral Fellow II, Dr. Stuart Dryer’s Laboratory, Department of Biology and Biochemistry, University of Houston, Houston, TX

- **2000 - 2004** Research Associate, Dr. Stuart Dryer’s Laboratory, Department of Biology and Biochemistry, University of Houston, Houston, TX

- **2004 - 2010** Assistant Professor, Department of Veterinary Integrative Biosciences, Texas A&M University, College Station, TX

- **2008 - 2010** Adjunct Assistant Professor, Department of Neurosciences and Experimental Therapeutics College of Medicine Texas A&M Health Science Center, Bryan/College Station, TX

- **2010 -** Associate Professor, Department of Veterinary Integrative Biosciences, Texas A&M University, College Station, TX

- **2010 -** Adjunct Associate Professor, Department of Neurosciences and Experimental Therapeutics College of Medicine Texas A&M Health Science Center, Bryan/College Station, TX

**Other Experience and Professional Memberships**

- **1989 - 1992** Member, the Association of Anatomists of Taiwan

- **1990 -** Member, Society for Neuroscience

- **2005 -** Member, Association for Research in Vision and Ophthalmology (ARVO)

- **2011 -** Member, European Biological Rhythms Society

- **2013 - 2014** Local Chapter President, Texas A&M Chapter of the Society for Neuroscience

- **2014 -** Member, International Society for Eye Research (ISER)

- **2015 -** NIH-NIOSH (National Institute for Occupational Safety and Health) study section *ad hoc* reviewer

**Honors and Awards**

- **1989** Graduate Student Scholarship Award (1989-1991), National Yang-Ming Medical College, Taiwan

- **1989** Annual Scientific Research Award, National Cheng-Kung University, Taiwan

- **1990** C. Yin, M.D. Memorial Scholarship Award, National Yang-Ming Medical College, Taiwan

- **1993** Tuition Scholarship (1993-1994), Northeastern Ohio Universities College of Medicine, OH

- **1994** Teaching Assistantship (1994-1996), Northeastern Ohio Universities College of Medicine, OH

- **1997** NIH Training Grant (1997-1999), Department of Neurobiology and Anatomy, University of Texas-Houston Medical School, Houston, TX

- **2001** NIH Individual National Research Service Award (NIH F32 EY 13920; 2001-2004), Houston, TX

- **2007** Montague Center for Teaching Excellence Scholar Award, Texas A&M University, College Station, TX

- **2014** Texas A&M University Institute for Neuroscience Service Award, Texas A&M University and Health Science Center, College Station, TX
C. **Contributions to Science**

1. **Characterizing how circadian clocks regulate photoreceptor physiology by regulating ion channels.**

Both cGMP-gated cation channels (CNGCs) and L-type voltage-gated calcium channels (LTCCs) are essential in phototransduction and light sensitivities of photoreceptors. While the CNGC is the last step of phototransduction, the LTCCs govern neurotransmitter release from most retinal neurons, including photoreceptors. Hence, understanding the circadian regulation of CNGCs and LTCCs at the molecular level provides knowledge on how photoreceptor circadian clocks contribute to photoreceptors and retina light sensitivities, as well as light- and dark-adaptation. During my postdoctoral training, I designed all and performed most of the experiments to characterize the circadian regulation of CNGCs in cone photoreceptors, which resulted in 5 publications (1 in *Neuron*, 2 in *J. Neurosci.*, 1 in *Brain Res.*, and 1 in *IOVS*). I found that the apparent affinities of CNGCs to cGMP in cone photoreceptors are under circadian control (higher at night than the day), while the total currents remain the same throughout the day. While Ras-MAP kinase and CaMKII are essential for this circadian rhythm, it is the tyrosine phosphorylation on the auxiliary subunit of cone CNGCs that is under circadian regulation thus modulating the CNGC affinities.

As an independent PI at Texas A&M University, my research team further characterized the circadian rhythmicity of LTCCs and calcium homeostasis in cone photoreceptors. We identified several signaling pathways (including PI3K-AKT, cAMP-PKA, Ras-MAP kinase, CaMKII, nitric oxide-cGMP-PKG, calcineurin, mTORC1, and AMPK) that are involved in the complex signaling network to regulate LTCCs, as well as how intracellular calcium mediates somatostatin-induced inhibition of LTCCs (Jian et al., *J. Neurophysiol.*, 2009, 102: 1801–1810). As the PI of this work, I oversaw and designed the research work and directed staff scientists and graduate students to perform the pertinent experiments.


2. **Discovering a new role for microRNAs as a regulator of ion channels.**

While we unraveled the complex signaling network involved in the circadian regulation of LTCCs, we combined bioinformatics strategies and molecular analyses and discovered that microRNA-26a (miR-26a) targets the 3’UTR of the LTCCα1C subunit to regulate its translation in a circadian manner. Our discovery was the first to illustrate a dual role of a single microRNA: regulating an ion channel expression at the post-translational level as well as regulating the circadian rhythm. As the PI of this work, I oversaw the entire project and directed staff scientists to design and perform the pertinent experiments.


3. **Demonstrating how an extracellular protein (retinoschisin) regulates ion channels.**

Retinoschisin is an extracellular protein that is secreted from photoreceptors and bipolar cells and is important in maintaining retinal architecture. Mutations of the retinoschisin gene (RS1) cause X-linked retinoschisis with congenital blindness. While LTCCs regulate the secretion of retinoschisin, retinoschisin promotes the membrane retention of LTCCs. We further characterized the physical interaction between the LTCCα1 subunit and retinoschisin using co-immunoprecipitation and mammalian two-hybrid assays. Transfection with a loss-of-function RS1 mutant in photoreceptors causes a decrease in LTCCs. As the PI of this work, I oversaw the entire project and directed staff scientists to perform the pertinent experiments.

Using bioinformatics and mass spectrometry (MS)-based proteomics, we recently discovered a novel bioactive peptide, **peptide Lv** (PLoS ONE 2012, 7(8): e43091), a small (40 amino acids in humans) secretory peptide that is expressed in major organs and tissues, including the retina and vascular endothelial cells. Peptide Lv augments the mRNA and protein expressions of LTCCs in retinal photoreceptors and cardiomyocytes. Peptide Lv interacts with vascular endothelial growth factor receptor 2 (VEGFR2), activates its downstream signaling, and promotes endothelial cell proliferation (BBA Molecular Cell Research 2015, 1853: 1154-1164).


5. Establishing chickens as a potential animal model for type 1 diabetes and characterizing the dysfunction of neural retina in obesity-induced type 2 diabetic mice.

There are various animal models (dogs, rodents, zebrafish) used to investigate diabetic retinopathy. While these animal models have certain characteristic diabetic phenotypes, none are without limitations. Chickens are diurnal species with cone-dominant retinas, which makes them suitable to study human cone photoreceptor-related degenerative diseases. While streptozotocin (STZ) successfully induces diabetes in dogs and rodents, previously, it failed to induce diabetes in adult birds. We took advantage of the fact that the pancreas is not fully developed in chicken embryos and injected STZ into the amnion layer in ovo at embryonic day 12 to successfully induce type 1 diabetes. We observed cataracts in STZ-injected chicken eyes, which occurs in ~24% of US patients with early on-set type 1 diabetes. Thus, this new model will complement the existing animal models for diabetic research. As the PI of this work, I oversaw the entire project and directed staff scientists to perform the pertinent experiments.

In collaboration with Dr. Beiyan Zhou (U. Conn), we established a high-fat-diet (HFD) induced diabetic mouse model, in which mice fed with a HFD regimen (59.4% fat calories) will develop diabetic retinopathy with both dysfunctional neural retinas (as shown by ERG) and pathological neovascularization (pilot data in this application). We further delineated the functional role of microRNA-150 contributing to HFD-induced DR (PLOS ONE 2016, 11 (6):e0157543). As the PI of this work, I oversaw the project and directed staff scientists to perform the pertinent experiments related with the retina, while Dr. Zhou oversaw the experiments that were related to the characterization of diabetic development in animals, as well as development of various miR-150 knockout/knock-in mouse lines. We have further reported on the effects of metformin in HFD-induced diabetic retina.


Complete List of Published Work in MyBibliography:

D. Research Support.
Ongoing Research Support
N/A

Completed Research Support

R21 EY023339-01 Ko (PI) 5/01/2013 – 4/30/2016
Project: Functional interactions among retinoschisin and its binding partners. The goal of this project is to investigate the functional interactions among retinoschisin, L-type voltage-gated calcium channel α1 subunit, and plasma membrane calcium ATPase isoform 1 (PMCA1).
Role: PI.

R01 EY017452-06A1 Ko (PI) 9/30/2012 – 8/31/2014
Project: Circadian rhythm in cone photoreceptors: cellular mechanisms. The goal of this project is to investigate the cellular mechanisms underlying the circadian output regulation of L-type voltage-gated calcium channels in chick retina cone photoreceptors.
Role: PI.

R01EY017452-01A1 Ko (PI) 4/01/2007 – 2/29/2012
Project: Circadian rhythm in cone photoreceptors: cellular mechanisms. The goal of this project was to investigate the cellular mechanisms underlying the circadian output regulation of L-type voltage-gated calcium channels in chick retina cone photoreceptors.
Role: PI.

F32 EY 13920 Ko (PI) 9/2001 - 9/2004
Project: Circadian regulation of cGMP-gated ion channels. The goal of this project was to characterize the circadian regulation of cGMP-gated ion channels and their underlying molecular signaling mechanisms in chick retina photoreceptors.
Role: NRSA Trainee and PI. Mentor: Stuart Dryer
BIOGRAPHICAL SKETCH

Provide the following information for the Senior/key personnel and other significant contributors in the order listed on Form Page 2. Follow this format for each person. DO NOT EXCEED FOUR PAGES.

NAME
JOE N. KORNEGAY

POSITION TITLE
PROFESSOR

VETERINARY INTEGRATIVE BIOSCIENCES
TEXAS A&M UNIVERSITY

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

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

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<td>UNIVERSITY OF GEORGIA, ATHENS</td>
<td>Residency</td>
<td>1976-79</td>
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<tr>
<td>UNIVERSITY OF GEORGIA, ATHENS</td>
<td>Residency</td>
<td>1979-82</td>
<td>VETERINARY PATHOLOGY</td>
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A. Personal Statement

My professional career has included various experiences, ranging from private veterinary practice to clinical residency and graduate training; to appointment as a faculty member with responsibilities extending across teaching, clinical service, and research; to administrative appointments as a department chair, hospital director, and dean; and to my current position, in which I have responsibilities primarily in research. Dating to my PhD dissertation, I have had a keen interest in the comparative aspects of disease and have characterized phenotypic features of various spontaneous animal disorders. My career as an academic clinician occurred in parallel with the emergence of cross-sectional imaging (computed tomography [CT] and magnetic resonance imaging [MRI]). As a result, I was fortunate to be involved in the early utilization of these techniques in naturally occurring neurologic diseases. For the past 30+ years, I have studied a spontaneous canine disease termed golden retriever muscular dystrophy (GRMD), which serves as an animal model for Duchenne muscular dystrophy (DMD) of humans. Both conditions are X-linked, occurring due to mutations in the DMD gene that codes for the dystrophin protein. An affected dog studied by our group until 40 months of age is the common sire of all dogs in GRMD colonies worldwide. We initially established our colony at North Carolina State University in the late ‘80s. It was moved to the University of Missouri-Columbia in 1994, the University of North Carolina at Chapel Hill (UNC-CH) in 2007, and Texas A&M University in 2012. Our research has defined key clinical and pathologic features of GRMD to both better understand disease pathogenesis and to also utilize these parameters in assessing treatment efficacy.


B. Positions and Honors

Employment

1973-1974 Associate Veterinarian, Weaver Animal Hospital, Perrysburg, OH
1974-1976 Associate Veterinarian, Memorial 610 Veterinary Clinic, Houston, TX
1976-1979 Resident (Neurology), University of Georgia, Athens
1979-1982 Resident (Pathology), University of Georgia, Athens
1982-1986 Associate Professor, North Carolina State University, Raleigh
1991-1992 Assistant Director of Services, North Carolina State University, Raleigh
1986-1994 Professor, North Carolina State University, Raleigh
1994-1999  Director, Veterinary Medical Teaching Hospital, University of Missouri-Columbia
1994-1999  Professor/Chairman, Veterinary Medicine & Surgery, University of Missouri-Columbia
1994-present Adjunct Professor, North Carolina State University, Raleigh
1994-2006 Investigator, Dalton Cardiovascular Research Center, University of Missouri-Columbia
1998-1999  Interim Dean, College of Veterinary Medicine, University of Missouri-Columbia
1999-2006 Dean, College of Veterinary Medicine, University of Missouri-Columbia
2006-2007 Visiting Research Professor, School of Medicine, University of North Carolina, Chapel Hill
2007-2012 Professor, School of Medicine, University of North Carolina, Chapel Hill
2012-present Adjunct Professor, School of Medicine, University of North Carolina, Chapel Hill
2012-present Professor, Interdisciplinary Program in Neuroscience, Texas A&M, College Station, TX
2012-present Professor, Veterinary Integrative Biosciences, Texas A&M, College Station, TX
2014-2016 Director, Texas A&M Institute for Preclinical Studies (TIPS)

Other Experience and Professional Memberships
1980-present American College of Veterinary Internal Medicine (ACVIM)
2012-present ACVIM Board of Regents, Successive offices of Vice President, President-Elect, President, Chair, and Past Chair (currently)
1993-1996 President, ACVIM Specialty of Neurology
1994-2006 Missouri Veterinary Medical Association, Executive Board
1998-2006 University of Missouri-Columbia European Union Center, Academic Cabinet
2003-2006 Board member, Missouri Innovation Center
2003-2005 Chair, University of Missouri-Columbia Planning and Design Construction Advisory Council
2004-2006 University of Missouri Strategic Planning and Resource Advisory Committee: Subcommittee on the Pursuit of Quality, Innovation, Incentives, Risks and Rewards
2004-2006 Mid-Continent Association for Agriculture, Biomedical Research and Education (MAABRE) Board of Directors
2008-2012 University of North Carolina at Chapel Hill IACUC
2008-present External Advisory Committee, Chairman, North Carolina State University Center for Comparative Medicine and Translational Research (CCMTR)

Honors
1985 Norden Distinguished Teacher Award, College of Vet Med, NC State
1991 Outstanding Extension Service Award, College of Vet Med, NC State
1995 Bourgelat Award, British Small Animal Veterinary Association
1998 Pfizer Award for Research Excellence, Pfizer Animal Health, College of Vet Med, Missouri
2003 Texas A&M University College of Veterinary Medicine Distinguished Alumnus Award
2005 Twelfth International Veterinary Congress Prize, American Veterinary Medical Association
2008 Robert W. Kirk Award for Professional Excellence (ACVIM)
2011 Honoris Causa Doctor, University of Liège (Belgium) (2011)
2014 Association of American Veterinary Medical Colleges (AAVMC) Recognition Lecture

C. Contributions to Science
1. A Canine Model of Duchenne Muscular Dystrophy. I have studied a spontaneous canine disease termed golden retriever muscular dystrophy (GRMD), which serves as an animal model for Duchenne muscular dystrophy (DMD) of humans, for over 30 years. Studies began while I was at the University of Georgia in 1981 and extend to my current appointment at Texas A&M. A dog studied by me beginning in 1981 is the common founder of all GRMD colonies worldwide. Both GRMD and DMD are X-linked, occurring due to mutations in the DMD gene. My research has defined key clinical and pathologic features of GRMD to both better understand disease pathogenesis and to also utilize these parameters in assessing treatment efficacy. In recent years, my laboratory and collaborators have studied various treatments (cell, molecular, and pharmacologic approaches) in affected dogs. Results of these preclinical studies should guide use of similar treatment strategies in DMD patients.

b. Sharp NJH, JN Kornegay, SD Van Camp, MH Herbstreith, SL Secore, S Kettle, W-Y Hung, CD


2. **Comparative Medicine/Pathology** – Dating to my PhD dissertation, I have had a keen interest in comparative medicine and pathology. In particular, I have defined key phenotypic features of spontaneous and experimental models of animal disease that mirror analogous conditions in humans. My PhD dissertation dealt with a form of herpes virus induced Marek’s disease termed transient paralysis (TP) that is seen spontaneously in certain inbred lines of chickens and for which susceptibility is linked to the chicken major histocompatibility complex (MHC). The inflammatory nature of the disease and its linkage to the MHC suggests potential relevance to multiple sclerosis. My experimental studies demonstrated that affected birds develop dramatic reversible brain edema, potentially due to cytopathic effects on oligodendrocytes. While in my PhD program and unrelated to this work on Marek’s disease, I became involved in studies of a spontaneous form of muscular dystrophy in golden retriever dogs (GRMD; see “A Canine Model of Duchenne Muscular Dystrophy” above). Our studies and those of Barry Cooper’s lab at Cornell University established that this is a genetically homologous model of DMD. Beyond these studies of transient paralysis in chickens and muscular dystrophy in dogs, I contributed to a number of other studies of animal diseases that model analogous conditions in humans, including as cited here, dysmyelination (Pelizaeus Merzbacher) and cerebellar hypoplasia, as well as those discussed under “imaging” and “comparative oncology” below.


3. **Cross-Sectional Imaging as a Diagnostic and Research Tool** – In initiating my faculty career as a veterinary neurologist at the College of Veterinary Medicine at North Carolina State University (NCSU) in 1982, I was fortunate to almost immediately begin working with neuroradiologists at Duke University on first computed tomography (CT) and later magnetic resonance imaging (MRI) studies in client-owned animals with spontaneous neurologic diseases. Indeed, the MRI studies at Duke allowed neuroradiologists to refine protocols on proprietary MRI scanners that had not yet been employed on human patients with analogous conditions. I continued to use CT and MRI in assessing animals with neurologic diseases over my 11-year appointment as a neurologist at NCSU, in many cases making sentinel observations on the imaging features of neurologic diseases. A number of these studies were published in the veterinary literature and greatly influenced the practice of my specialty. Later, upon taking an appointment at the School of Medicine at the University of North Carolina-Chapel Hill, I began using MRI as a biomarker in preclinical GRMD studies.


4. Paradoxical Muscle Hypertrophy in Muscular Dystrophy – Muscular dystrophy is a progressive disorder that should intuitively lead to muscle wasting and atrophy. However, paradoxically, in both DMD and GRMD, certain muscles hypertrophy. In the case of DMD, the muscle enlargement has been classified as “pseudohypertrophy,” due to a sense that the increase in size occurs due to deposition of fat and connective tissue, rather than muscle itself. However, with the advent of cross-sectional imaging, there is now a general consensus that certain muscles progress through a phase of true hypertrophy and only later undergo pseudohypertrophy and eventually atrophy. We have an analogous true hypertrophy in GRMD dogs using quantitative imaging and histopathologic studies. These studies have focused particularly on the cranial sartorius muscle, which undergoes dramatic hypertrophy, thus providing a model to elucidate genes/pathways involved in hypertrophy of dystrophic muscle. In collaboration with Eric Hoffmann’s laboratory, we have demonstrated that certain genes that are up-regulated in the cranial sartorius and that their expression levels correlate with cranial sartorius size, implying a cause and effect relationship. Separately, through a collaboration with Kathryn Wagner and Se Jin Lee at Johns Hopkins, cross breeding studies have been completed between GRMD dogs and whippets that have a mutation in their myostatin genes. We hypothesized that this mating would allow for more complete muscle regeneration and improve the GRMD phenotype, as has previously been shown in the dystrophin-deficient mdx mouse. But, GRMD dogs heterozygous for the myostatin mutation did not improve and, in fact, actually trended towards a more severe phenotype. This increase in disease severity appears to be due to preferential hypertrophy/atrophy of certain muscles, with resultant exaggeration of debilitating contractures and postural changes. Taken together, our studies demonstrate that myostatin inhibition may have untoward complications and may, therefore, inform treatment strategies for DMD and other muscle wasting disorders.


5. Comparative Oncology – Relating to my clinical responsibilities as a veterinary neurologist at NCSU, I assessed many dogs with naturally occurring brain and spinal cord tumors with histologic and biologic properties essentially identical to their counterparts in humans. The National Cancer Institute has recognized the value of such tumors as models for human cancer through its Comparative Oncology Program (https://ccrod.cancer.gov/confluence/display/CCRCOPWeb/Home). I collaborated extensively with oncologists and radiation oncologists at both NCSU and Duke on two NCI-supported Program Project Grants that included studies on brain neoplasia in dogs. Later, as Dean of the veterinary school at the University of Missouri, I took part in a campus wide initiative directed at achieving comprehensive cancer center status. In fact, for a while, I was Co-Director of Missouri’s cancer center and chaired the committee to recruit a cancer center director. As Director of the Texas A&M Institute for Preclinical Studies (TIPS), I worked with our own staff and oncologists at the veterinary school to utilize the PET-CT unit at TIPS for detection of metastases in client owned dogs with cancer. These collective experiences are in keeping with my broader work in comparative medicine (one medicine). I was recognized for these contributions through the 2014 “Recognition Lecture” of the Association of American Veterinary Colleges (AAVMC), “One man’s view of one health.”


**D. Research Support**

**Current**

**Solid GT**


Role: Joe N. Kornegay, Principal Investigator on the TAMU subcontract.

This study builds on the DoD grant, *Advanced Gene Therapy for Treatment of Cardiomyopathy and Respiratory Insufficiency in DMD*, being done in collaboration with Dr. Barry Byrne at the University of Florida (see below). With improvements in AAV culture methods, it is now possible to produce sufficient virus to allow systemic (vs localized, intrathoracic) treatment. Thus, we have revised the goals of the DoD grant to treat an expanded number of GRMD dogs (21 vs. 9) and to use systemic delivery. This will also entail additional outcome parameters to assess effects on appendicular skeletal muscle. The budget for this project will be routed through the University of Florida.

**Southwest National Primate Research Center, Texas Biomedical Research Inst**

Derivation of muscle progenitors from hPSCs to transplant in a dog model of DMD.

Role: Joe N. Kornegay, Principal Investigator

This is a collaboration with Tiziano Barberi who has recently relocated to the Southwest National Primate Center. Dr. Barberi is an international expert in human pluripotent stem cells (hPSCs). This study will assess four GRMD dogs injected intramuscularly with hPSCs.

**DMDRP Investigator-Initiated Research Award**

Byrne BJ (PI)

DoD Congressionally Directed Medical Research Programs (CDMRP), University of Florida (Texas A&M University Subcontract)

Advanced Gene Therapy for Treatment of Cardiomyopathy and Respiratory Insufficiency in DMD

Role: Joe N. Kornegay, Principal Investigator on the Texas A&M University subcontract.

This project will entail a subcontract between Texas A&M and the University of Florida to extend respiratory and cardiac AAV studies completed in murine and macaque models to the GRMD model.

**Zoetis-Morris Animal Foundation**

Cardiomyopathy in the golden retriever model of muscular dystrophy.

This is a fellowship for Dr. Sarah Schneider. I will be serving as her mentor for studies that correlate genomic and phenotypic features of the GRMD cardiomyopathy. Dr. Schneider is veterinarian and diplomate of the American College of Veterinary Pathologists.

**1R01AR064338-01A1**

Burkin DJ (PI)

NIH (NIAMS), University of Nevada-Reno (Texas A&M University Subcontract)

Laminin protein therapy for congenital muscular dystrophy.

Role: JN Kornegay, Principal Investigator on the Texas A&M subcontract

This project will define the PK properties of laminin-111 in normal dogs produced through the Texas A&M GRMD colony and are preparatory to move to human trials for congenital muscular dystrophy.
Glycosyltransferase Therapies for Myopathies (Renewal - R01 AR049722)
Role: JN Kornegay, Principal Investigator on the Texas A&M subcontract. Our collaborator, Paul Martin, has demonstrated that overexpression of GALGT2, a gene that encodes a glycosylation enzyme that alters sugars on the skeletal muscle membrane, boosts the expression of proteins that ameliorate disease. Previous studies have demonstrated therapeutic efficacy in three different mouse models of muscular dystrophy, including the mdx mouse model for DMD. Specific Aim 2 of this project will include studies to determine preclinical efficacy of AAV(rh.74)-MHCK7-GALGT2 in GRMD dogs.

Solid GT
Image and histopathologic assessment of GRMD dogs treated with AAV-microdystrophin constructs.
Role: JN Kornegay, Principal Investigator
This is a 2-year fellowship for Dr. Sharla Birch. I will be serving as her mentor for studies that correlate imaging and histopathologic studies in GRMD dogs treated with an AAV-micro-dystrophin construct. Dr. Birch is a veterinarian and has completed a residency in veterinary pathology. She is nearing completion of her PhD dissertation in Veterinary Pathobiology focused on the use of cross-sectional (CT and MRI) imaging as a biomarker for a sheep model of fetal alcohol syndrome. Her dissertation includes an additional project that correlates histopathologic and MRI findings in an ex vivo GRMD (pectineus) muscle model.

Safety and efficacy of systemic gene therapy in informative models for DMD.
Role: JN Kornegay, Principal Investigator on the Texas A&M University subcontract. This study extends a long-term collaboration between Drs. Kornegay and Stedman and will study immunological effects and outcome parameters in both GRMD and the dystrophin-null GSHPMD canine models following systemic AAV-mini-dystrophin gene therapy.
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: Julian Leibowitz, M.D.Ph.D.

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

POSITION TITLE: Professor of Microbial Pathogenesis and Immunology

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.)

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<td>B.A.</td>
<td>06/1968</td>
<td>Chemistry</td>
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<td>Albert Einstein College of Medicine, Bronx, NY</td>
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<td>Intern/Resident Pathology</td>
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A. Personal Statement

Over the past 34 years my research program has largely been concerned with utilizing biochemical and genetic approaches to studying both the replication and pathogenesis of coronavirus infections. Our focus has been on mouse hepatitis virus (MHV), the prototypical coronavirus. MHV provides rodent models relevant to important human diseases: multiple sclerosis, acute fulminant hepatitis, and the severe pulmonary infections caused by SARS-CoV and the newly emergent MERS-CoV. A major focus of my research program over the last 15 years has been to identify RNA structures that have an important role in coronavirus replication. Ultimately, through identifying and characterizing these cis-acting structures and the basis by which these structures interact with each other and with various trans-acting viral and host proteins to enable viral replication, we hope to identify specific targets for novel antivirals that can block replication by targeting these interactions. A second project utilizes the MHV-A59, MHV-JHM, MHV-1, and MHV-3 strains which vary widely in their pathogenetic potential and in organ tropisms, with MHV-A59 being moderately neurotropic and hepatotropic, MHV-JHM being strongly neurotropic, MHV-3 being hepatotropic, and MHV-1 being pneumotropic. Our approach to understanding these differences in pathogenesis and tropism has generally been genetic and molecular virologic with the goal of identifying the viral genes that are important for pathogenesis. We have utilized the MHV-1 platform to investigate the possibility that it might be possible to repurpose drugs that modulate the renin angiotensin system (RAS) for the treatment of viral pneumonia. My laboratory has recently been part of a collaborative team to explore the use of a microfluidic platform to rapidly define the B-cell repertoire after viral infection or vaccination using MHV as a model system for proof of concept. These research programs have provided multiple graduate training opportunities for students in molecular virology, viral immunology, viral pathogenesis, and pathology. In addition to my research endeavors I have been the director of the Texas A&M MD/PhD Program since 2004 with the training, leadership, and administrative tasks this entails.

B. Positions and Honors.

Positions:
1970-75. Medical Scientist Trainee, Albert Einstein College of Medicine, Bronx, N.Y.
1975-77. Intern and Resident, Department of Pathology, University of California, San Diego.
1979-83. Assist. Prof. of Pathology in Residence, University of California, San Diego.
1983-95. Assist. Prof., Assoc. Prof., Prof. of Pathology and Laboratory Medicine, University of Texas Medical School-Houston.
June, 1995-December, 2005. Prof. of Pathology and Laboratory Medicine, Texas A&M College of Medicine, College Station, TX.
Jan. 2006-present. Prof. of Microbial Pathogenesis and Immunology, Texas A&M College of Medicine, College Station, TX.
July 1998-present. Prof. of Veterinary Pathobiology, Texas A&M University, College Station, TX.
Oct. 2004-present. Director of Texas A&M MD/PhD Program

Other Experience and Professional Memberships
Member, American Society of Virology, 1981-present
Member, American Society for Microbiology, 1977-present
Member, American Association for the Advancement of Science, 1975-present
Member, Association of University Pathologists, 1991
Member, Veteran's Administration Merit Grant, Infectious Diseases Panel, 1994 -1997
NIH Ad hoc Peer Reviewer for Virology, Path B, CBNT, Neurology, and 7 Special Study Sections, 1987-2014
Peer Reviewer for Medical Research Council, UK, 2008

Honors and Awards:
Eta Mu Alpha, College Honor Society
New York State Regents Medical School Scholarship
Elected to the Pluto Club (Association of University Pathologists), 1991

C. Contribution to Science

1. My early work on coronavirus replication represented some of the first molecular biologic studies of this group of viruses. We identified key features of coronavirus replication: a 3' nested set arrangement of the coronavirus subgenomic mRNAs; identified the proteins encoded by these mRNAs; and showed that coronavirus replication was independent of the nucleus and could take place in enucleated cells. One of these studies (citation c) noted that there was an unusual structural arrangement at the 5'ends of the subgenomic mRNAs and set the stage for the discovery of the discontinuous synthesis of coronavirus mRNAs. I was the primary investigator or co-primary investigator on all of these studies.

2. In addition to the contribution described above, as part of my post-doctoral training with Dr. James Robb I performed a biochemical characterization of a set of temperature sensitive mutants of MHV, strain JHM. Subsequently my laboratory performed the earliest complementation testing of these mutants plus a second collection of mutants isolated by Haspel and Oldstone. These experiments suggested that at least six MHV gene products were required for MHV RNA synthesis. A later set of experiments expanded the complementation grouping to two additional panels of MHV-A59 temperature sensitive mutants and demonstrated that there were eight complementation groups amongst mutants with temperature sensitive defects in MHV RNA synthesis, suggesting that at least eight MHV gene products were required for MHV RNA synthesis. I was the primary investigator on all but the first of these studies.
3. In a series of studies performed in collaboration with Dr. Gary Levy we identified a macrophage procoagulant activity (a prothrombinase) that was induced by MHV-3 infection of macrophages from mice susceptible to lethal hepatitis, but not during infection macrophages from resistant mice. This macrophage prothrombinase activity was shown to play an important role in the pathogenesis of lethal hepatitis, and its inhibition by monoclonal antibody protected mice from otherwise lethal disease. We then used this antibody to molecularly clone the gene encoding this prothrombinase and identified this gene as Fgl2. Subsequent work demonstrated the induction of this gene in livers from humans with fulminant hepatitis. This line of research was initiated in my laboratory, and I served as the primary investigator or co-investigator in all of these studies. Later work by other workers has shown that in addition to prothrombinase activity Fgl2 is an immunoregulatory molecule secreted by Tregs and influences outcome in human viral hepatitis and in transplantation.


4. During the past several years the focus of my laboratory has been directed at identifying sequences and RNA secondary structural elements that are important in coronavirus replication. To date these studies have focused on the 5’ and 3’ cis-acting regions. We generated a novel secondary structural model of the coronavirus 5’UTR which has become the accepted model of these cis-acting sequences, demonstrated that the three stem-loops in the 5’UTR play a crucial role in coronavirus replication being required for discontinuous subgenomic mRNA synthesis. We showed that the individual stem-loops in the 5’UTR are functionally exchangeable amongst coronaviruses, thus it is the structure rather than the particular sequence that is required. Studies on the structure and function of the 5’UTR SL1 showed that there is a requirement for structural lability in the proximal region of SL1 and provided genetic evidence for an association of the 5’ and 3’ UTRs during replication, an event that had been hypothesized in a widely accepted model of coronavirus subgenomic RNA synthesis but for which data had been lacking until our work. We subsequently utilized SHAPE technology to generate biochemical support for a secondary structural model of the entire cis-acting region in the 5’end of the genome, a region that extends into the Orf1a coding region (citation d). This secondary structure model is conserved amongst the betacoronaviruses, including the SARS-coronavirus. Reverse genetic studies demonstrated that two of these secondary structural elements, SL5C and SL6, are not required for virus replication. We have also done biochemical and reverse genetic studies of the secondary structure of the MHV 3’UTR. The most recent of these have demonstrated a functional role in viral replication for a structural element in the 3’UTR which had not previously been investigated (citation c). I served as the primary investigator or co-investigator in all of these studies.


Complete List of Published Work in MyBibliography:

D. Research Support

Ongoing Research Support

1 R21 AI121807-01 Leibowitz (PI) 03/15/2016 – 02/28/2018 1.2 months
National Institutes of Health $125,000 first year direct costs

Conserved RNA Secondary Structures in three Betacoronaviruses: MHV, BCoV, and MERS-CoV
In this application we propose to study the RNA secondary structure of MERS-CoV and two related coronaviruses, mouse hepatitis virus (MHV) and bovine coronavirus (BCoV) with the goal of identifying and functionally characterizing novel conserved RNA secondary structures in the genomes of these viruses.

Texas A&M Foundation Gift Account 10/1/15-open not specified
Gift from Harley's Angels $1,044
This gift is to support exploratory research on a system for the rapid isolation of B-cells secreting anti-MHV or anti-FIPV neutralizing antibodies.

CST*R Pilot Grant. Leibowitz, PI, Han, Contact PI 3/1/17-2/28/18 not specified
TAMHSC $25,000 to Leibowitz
PRESCIENT: An Integrated System for Predicting Vaccine Efficacy
This application seeks to develop a microfluidic system suitable for high-throughput identification of B-cells secreting neutralizing antibody.

Texas A&M HSC Bridge Funding 10/1/15--open not specified
Approximately $2,400 remaining
These funds are to support ongoing research on MHV-1 pathogenesis, exploratory research on a system for the rapid isolation of B-cells secreting anti-MHV or anti-FIPV neutralizing antibodies, and to supplement 1 R21 AI121807.

Completed Research Support

Leibowitz (PI) 4/15/13-12/31/15
Texas A&M HSC CST*R Pilot Grant Program
Manipulation of the Renin-Angiotensin System as Therapy for Viral Pneumonias
This project used the MHV-1 mouse model of SARS and the PR8 mouse adapted influenza virus model of severe influenza virus pneumonia to investigate the repurposing of drugs that blockade of the renin angiotensin system (RAS) for the treatment of viral pneumonia.
Role in Project: Principal Investigator


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: Levine, Jonathan M.

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

POSITION TITLE: Professor, Helen McWhorter Chair, and Head of Department of Small Animal Clinical Sciences

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.)*

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<td>D.V.M.</td>
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<td>Veterinary Medicine</td>
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<td>Colorado State University, Fort Collins, CO</td>
<td>Diplomate ACVIM (Neurology)</td>
<td>01/2005</td>
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A. Personal Statement

I am a veterinarian with expertise in canine naturally occurring neurological diseases and the use of these model systems in translational research. Our laboratory has worked to define inflammatory events, validate outcome measures (eg, magnetic resonance imaging, gait analysis), and develop high-impact canine-based clinical trials in pet dogs with glioma and spinal cord injury. Many of our completed and on-going studies are multi-institutional collaborations with major medical centers to investigate therapies or basic biology that have significant human healthcare or societal impact. Our current research portfolio includes studies examining tumor immunophenotype and immunotherapy in dogs with naturally occurring glioma (collaboration with Amy Heimberger, MD Anderson and Roel Verhaak, Jackson Laboratories), determination of genomic factors modulating recovery in dogs with spinal cord injury (collaboration with Bob Grossman, Methodist Hospital and Natasha Olby NC State), and identification of anatomic/genetic determinants of olfaction in dogs (collaboration, Bob Wayne UCLA). Based on our record working with Dr. Heimberger on immunotherapies for dogs with glioma and investigators at other institutions on translational projects with high impact, we are well positioned to complete the proposed work.

B. Positions and Honors

**Positions and Employment**

<table>
<thead>
<tr>
<th>Positions and Employment</th>
<th>Institution, Location</th>
<th>Dates</th>
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<tbody>
<tr>
<td>Clinical Assistant Professor</td>
<td>Texas A&amp;M University, College Station, TX</td>
<td>2005-2006</td>
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<tr>
<td>Assistant Professor</td>
<td>Texas A&amp;M University, College Station, TX</td>
<td>2006-2012</td>
</tr>
<tr>
<td>Associate Professor</td>
<td>Texas A&amp;M University, College Station, TX</td>
<td>2012-2015</td>
</tr>
<tr>
<td>Helen McWhorter Chair</td>
<td>Texas A&amp;M University, College Station, TX</td>
<td>2014-present</td>
</tr>
<tr>
<td>Professor</td>
<td>Texas A&amp;M University, College Station, TX</td>
<td>2015-present</td>
</tr>
<tr>
<td>Department Head</td>
<td>Texas A&amp;M University, College Station, TX</td>
<td>2015-present</td>
</tr>
</tbody>
</table>
C. Contribution to Science (from a total of 89 publications)

1. We have worked to define the early inflammatory events following spinal cord injury in dogs. In particular, we have identified MMP-9, IL-8, MCP-1, and acute phase proteins as critical modulators of injury severity/recovery. We have also shown MBP release in the CSF is predictive of failure to recover in this model system.


2. We have defined new techniques for imaging the canine spinal cord using MRI and methodologies for quantifying abnormalities. Additionally, our group has identified relationships between abnormal MRI signal and severity of injury/recovery in dogs.


3. Our group has developed techniques to perform canine-based therapeutic trials in dogs with glioma and spinal cord injury to generate high quality pre-clinical data to inform human clinical trials. Many strategies we have worked with involve modulation of immune responses.


4. We have worked extensively to characterize the natural history and biology of canine gliomas. Our group has defined the MRI appearance of these tumors, validated minimally invasive brain biopsy techniques, and generated basic data concerning mechanisms of oncogenesis in dogs.


D. Research Support

Ongoing Research Support:

Mission Connect Foundation Levine G. (PI) 11/1/16-11/1/17
Mapping of chromosomal loci associated with injury severity and locomotor recovery following acute spinal cord injury in the dachshund dog
Role: Co-I
Goals: To characterize genomic alterations associated with spinal cord injury severity and long-term recovery.

NIH/NCI P30 CA 016672-40 Suppl DiPhinto (PI) 9/1/16-8/31/17
Sequencing of canine gliomas
Role: Co-I of Supplement (TAMU site PI)
Goals: To define genomic genetic alterations and associations with impaired immune responses, altered immune checkpoint expression, and neoantigen expression in canine gliomas.

NSF Wayne (PI) 1/1/15-1/1/18
Collaborative research: The genetic and anatomical determinants of olfaction in dogs
Role: Co-PI
Goals: To characterize anatomic facets of the olfactory system, nasal passage airflow, and variation in RNA profiles in different breeds of dogs.

AKC-CHF Bertocci (PI) 1/1/15-6/1/17
Development of a neuromusculoskeletal computer simulation gait model to characterize functional recovery in dogs with intervertebral disk herniation
Role: Co-PI
Goals: To develop a 3-dimesntional model of canine gait with predicted muscle activation in normal and injured dogs.

AKC-CHF G.Levine (PI) 1/1/15-6/1/17
Describing the kinetic and kinematic recovery of Dachshunds with spinal cord injury
Role: Co-PI
Goals: To longitudinally characterize recovery from spinal cord injury in a cohort of dogs using kinematics and kinetics and to characterize normal gait in matched controls.
Rapamycin Holdings  Levine (PI)  6/1/14-6/1/17
Phase I canine SCI study using eRapamycin
Role: PI
Goals: To examine the safety, pharmacokinetics, and pharmacodynamics of eRapa in dogs with spinal cord injury

MD Anderson Cancer Center  Levine (PI)  1/15/14-6/1/17
miR-124 delivery in dogs with glioma
Role: PI (Sub-award, Rose Foundation)
Goals: To examine the safety, pharmacokinetics, and pharmacodynamics of LUNAR-301 in healthy and tumor-bearing dogs. And, to examine the efficacy of LUNAR treatment in tumor-bearing dogs using MRI-based outcomes.

Completed Research Support (Last 5 years):
Mission Connect Foundation  Levine (PI)  5/1/15-5/1/16
Canine Summit on Spinal Cord Injury
Role: PI
Goal: To develop a consortium focused on translational applications of canine spinal cord injury. Attendees represented 8 institutions in Europe and the USA and included veterinarians, medical doctors, and basic scientists.

DOD SC100140  Noble (PI)  10/1/11-10/1/15
Matrix metalloproteinases as a therapeutic target to improve neurological recovery after spinal cord injury
Role: Co-PI
Goal: To conduct a Phase II canine study in dogs with spinal cord injury to examine the safety, pharmacokinetics, pharmacodynamics, and efficacy of a metalloproteinase inhibitor in spinal cord injured dogs.

UT Houston Health Science Center  Levine (PI)  3/1/11-3/1/13
Development of a canine brain tumor tissue bank
Role: PI
Goal: To establish a tissue bank and fund development of brain biopsy techniques in dogs with intracranial tumors.

Dana Foundation  Cooper (PI)  1/1/10-1/1/13
T-cell therapy for diffuse interstitial pontine glioma.
Role: Co-PI
Goal: To develop brain biopsy techniques in dogs and deliver CAR T cells to dogs with malignant glioma.
BIOGRAPHICAL SKETCH

Name: Jianrong Li, Ph.D.

Position/Title: Associate Professor

Education

<table>
<thead>
<tr>
<th>Institution and Location</th>
<th>Degree</th>
<th>Year Conferred</th>
<th>Field of Study</th>
</tr>
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<tbody>
<tr>
<td>Beijing Normal University</td>
<td>B.S.</td>
<td>1988</td>
<td>Chemistry</td>
</tr>
<tr>
<td>Beijing Normal University</td>
<td>M.S.</td>
<td>1991</td>
<td>Organic Chemistry</td>
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<tr>
<td>University of Hawaii</td>
<td>Ph.D.</td>
<td>1997</td>
<td>Biochemistry</td>
</tr>
<tr>
<td>University of Pittsburgh</td>
<td>Postdoc</td>
<td>2000</td>
<td>Mol. Cellular Biology</td>
</tr>
<tr>
<td>Children's Hospital, Harvard Medical School</td>
<td></td>
<td>2000-2005</td>
<td>Neurobiology</td>
</tr>
</tbody>
</table>

Research and Professional Experience

Appointments and Positions

1989-1990 Graduate Teaching Assistant, Dept. of Chemistry, Beijing Normal University
1991-1993 Instructor, Beijing Institute of Chemical Technology, P. R. China
1997-2000 Research Associate, School of Medicine, University of Pittsburgh
2000-2005 Instructor, Division of Neuroscience, Children's Hospital, Harvard Medical School
2006-2012 Assistant Professor, Dept. of Vet. Integrative Biosciences, Texas A&M University
2012- Associate Professor, Dept. of Vet. Integrative Biosciences, Texas A&M University

Faculty of Texas A&M Institute for Neuroscience

Other Professional Activities

2001- Member, Society for Neuroscience
2008- Member, American Society for Neurochemistry
2010 Panelist, National Science Foundation


2014- National Multiple Sclerosis Society Advisory Committee on Fellowship

Academic Honors and Awards

1985 & 1986 Outstanding Student Award at Beijing Normal University
1994-1997 Scholarships, Chun Ku & Soo Yong Huang Foundation, Hawaii
1995-1997 American Heart Association (AHA-Hawaii Affiliate) Predoctoral Fellowship
1999-2000 NIH Individual National Research Service Award (NRSA)
2001-2002 William Randolph Hearst Foundation Research Award
2002-2004 United Cerebral Palsy Foundation Research Award
2004-2006 United Cerebral Palsy Foundation Research Award
2004-2006 William Randolph Hearst Foundation Research Award
2005-2006 Priscilla and Richard Hunt Fellowship, Eleanor and Miles Shore Scholar in Medicine, Harvard Medical School
2014 Outstanding Scientific Achievement Award, College of Veterinary Medicine and Biomedical Sciences, Texas A&M University

Number pages consecutively throughout the Application. Do NOT use suffixes such as 6a, 6b...

Texas A&M Institute for Neuroscience

395


Publications

**Publications 2012-2017:**

**Original Research Reports:**


**Invited reviews and book chapters:**

Research Support

Ongoing Research Support

RG1057-05632 (Li - PI)  4/01/2016-3/31/2019
National Multiple Sclerosis Society
Stat3 in myeloid cells: a regulator of autoimmune demyelination
Role: PI
The goal of this proposed study is to investigate mechanisms by which Stat3 signaling in myeloid cells modulates adaptive immune responses in autoimmune demyelination mouse model of multiple sclerosis.

R21NS093487-01 (Li - PI)  6/15/2015-5/31/2017
NIH-NINDS
Role of caspase-8 in neuroinflammation, demyelination and myelin repair
Role: PI
This project aims to use conditional mutant mice to investigate non-apoptotic functions of caspase-8 in regulating microglial immune responses and the effect on oligodendrocyte regeneration in toxin models of de/remyelination.

R21EB021005 (Han - PI)  9/15/2015-06/30/2017
NIH-NIBIB (National Institute of Biomedical Imaging and Bioengineering)
A high-throughput microfluidic in vitro CNS myelination model towards drug screening
Role: Co-PI
The goal of this study is to establish a novel microfluidic high-throughput platform that employs brain aggregates for drug screen for remyelination.

Completed Research Support

R21NS077215 (Li - PI)  07/01/2012 - 6/30/2014 with NCE
NIH/NINDS
"Identification of Novel Small Molecules for CNS Myelin Repair"
Role: PI

"Role of Adhesion G protein-coupled receptors in glial cell development and myelination"
Role: Co-I

RG 4586A (Li - PI)  10/01/2011-09/30/2014 with NCE
National Multiple Sclerosis Society
"Role of Astroglial Galectin-9 in CNS Demyelination and Remyelination"
Role: PI

R56 NS060017-05A (Li - PI)  04/01/2012-03/31/2014
(R01 NS060017)
NIH-NINDS
Glial Interactions in Premyelinating Oligodendrocyte Destruction
Role: PI

FG 1937 (Steelman - PI)  07/01/2011-06/30/2014
National Multiple Sclerosis Society
"The role of the Tim-3/galectin-9 pathway in microglia activation and demyelination"
Role: mentor
BIOGRAPHICAL SKETCH

Provide the following information for the Senior/key personnel and other significant contributors in the order listed on Form Page 2. Follow this format for each person. DO NOT EXCEED FOUR PAGES.

<table>
<thead>
<tr>
<th>NAME</th>
<th>POSITION TITLE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peng Li</td>
<td>Professor</td>
</tr>
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<table>
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<tr>
<th>eRA COMMONS USER NAME (credential, e.g., agency login)</th>
<th>PIECE</th>
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</table>

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

<table>
<thead>
<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE</th>
<th>MM/YY</th>
<th>FIELD OF STUDY</th>
</tr>
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<tbody>
<tr>
<td>Xi'an Jiaotong University, Xi'an, China</td>
<td>B. E.</td>
<td>07/94</td>
<td>Information Engineering</td>
</tr>
<tr>
<td>Xi'an Jiaotong University, Xi'an, China</td>
<td>M. E.</td>
<td>07/97</td>
<td>Systems Engineering</td>
</tr>
<tr>
<td>Carnegie Mellon University, Pittsburgh, PA</td>
<td>Ph. D.</td>
<td>12/2003</td>
<td>Electrical and Computer Engineering</td>
</tr>
<tr>
<td>Carnegie Mellon University, Pittsburgh, PA</td>
<td>Postdoctoral</td>
<td>07/2004</td>
<td>Electrical and Computer Engineering</td>
</tr>
</tbody>
</table>

A. Personal Statement

I have established expertise in computational neuroscience, in particular, biophysically based modeling of neuronal networks, parallel numerical techniques for large-scale simulation of brain models, and computational modeling of epilepsy, and development of parallel neural network simulation tools on multi-core shared memory machines, massively parallel graphic processors and supercomputers. I have published a number of peer-reviewed publications in the above areas. More broadly, I have done significant work in integrated circuits and systems, VLSI hardware brain-inspired computing systems, modeling and analysis of dynamic properties of biological and electronic systems, characterization and design of biological and engineered memory systems, algorithms and tools for computer-aided design of integrated systems. My past and ongoing work has been funded by NSF, Semiconductor Research Corporation (SRC), a world-wide semiconductor research consortium, DoD through the Focus Center Research Program, Intel Corporation, Freescale Semiconductors and Texas Instruments.

B. Positions and Honors

Positions and Employment

2015- Professor, Department of Electrical and Computer Engineering, Texas A&M University, College Station, TX
2010- Associate Professor, Department of Electrical and Computer Engineering, Texas A&M University, College Station, TX
2011- (Courtesy) Member, Graduate Faculty, School of Graduate Studies, Texas A&M Health Science Center, College Station, TX
2011- (Courtesy) Member, Faculty of Neuroscience, Texas A&M University, College Station, TX
2004-2010 Assistant Professor, Department of Electrical and Computer Engineering, Texas A&M University, College Station, TX
2003-2004 Postdoctoral Fellow, Department of Electrical and Computer Engineering, Carnegie Mellon University, Pittsburgh, PA
Honors

2016  Best Paper Award
      53th IEEE/ACM Design Automation Conference (DAC), 2016
2016  ISCAS Honorary Mention Best Paper Award
      The Neural Systems and Applications Technical Committee of IEEE Circuits and Systems
2015  Fellow of the IEEE (Institute of Electrical and Electronics Engineers), elected in 2015
2014  William and Montine P. Head Fellow, College of Engineering, Texas A&M University, 2013-2014
2013  Best Paper Hat Trick Award (for receiving the conference best paper award three times), 50th
      IEEE/ACM Design Automation Conference (DAC), Austin, TX
2013  DAC Prolific Author Award, 50th IEEE/ACM Design Automation Conference (DAC), Austin, TX
2013  DAC Top 10 Author in Fifth Decade, 50th IEEE/ACM Design Automation Conference (DAC),
      Austin, TX
      on Computer-Aided Design, San Jose, CA
2011-2012 TEES Fellow Award, College of Engineering, Texas A&M University, College Station, TX
2011  Best Paper Award, IEEE/ACM Design Automation Conference (DAC), San Diego, CA
2008  Outstanding Professor Award, Department of Electrical and Computer Engineering, Texas A&M
      University, College Station, TX
2008  NSF Career Award, National Science Foundation
2008  Best Paper Award, IEEE/ACM Design Automation Conference (DAC), Anaheim, CA
2007  Inventor Recognition Award, Microelectronics Advanced Research Corporation
2006  Inventor Recognition Award, Microelectronics Advanced Research Corporation
2004  Inventor Recognition Award, Semiconductor Research Corporation
2003  Best Paper Award, IEEE/ACM Design Automation Conference (DAC), Anaheim, CA
2001  Inventor Recognition Award, Semiconductor Research Corporation

C. Selected Peer-reviewed Publications (Selected from over 180 peer-reviewed publications)

Supervised graduate students/post-doctoral fellows are delineated with an asterisk (*).

Most relevant
   and architectural exploration,” in ACM Journal on Emerging Technologies in Computing Systems, vo. 12,
   26, no. 11, pp. 2635-2649, Nov. 2015.
4. “Yan, B. and Li, P. The emergence of abnormal hypersynchronization in the anatomical structural network
   epileptic seizures and its implication on optimal therapeutic treatments. PLoS ONE 6(7): e22440.
   doi:10.1371/journal.pone.0022440
6. “Yan, B. and Li, P. Reduced order modeling of passive and quasi-active dendrites for nervous system
   accurate models on graphics processors. IEEE International Joint Conference on Neural Networks, pp.
   3184-3193, July 2011.

**Other relevant publications (in chronological order)**


**D. Research Support**

**Selected Ongoing Research Support**

**NSF/SRC**  
Li (PI)  
$499,113  
10/01/2016-9/30/19

**E2CDA: Type II: Self-Adaptive Reservoir Computing with Spiking Neurons: Learning Algorithms and Processor Architectures**

The this study is to develop computational models, learning algorithms, circuit architectures and VLSI design techniques to enable self-adaptive neuromorphic learning systems based on recurrent spiking neural networks.  
Role: PI

**SRC**  
Li (PI)  
$246,000  
11/01/2016-10/31/19
Hierarchical Analog and Mixed-Signal Verification Using Hybrid Formal and Machine Learning Techniques
The goal of this study is to develop algorithms and tools to provide scalable solutions to design verification of analog and mixed-signal integrated circuits by using a combination of formal and machine learning techniques.
Role: PI

NSF ECCS-1405774 Li (PI) E. Sanchez-Sinencio (Co-PI) $399,999 08/01/2014-7/31/17
Taming the Stability Challenge of Analog and Mixed-Signal Systems
The goal of this study is to develop theory, methods and design practice to ensure stability of a range of analog and mixed-signal systems.
Role: PI

Selected Completed Research Support

SRC/TxACE Li (PI) $240K 08/01/2013-7/31/16
Statistical Analog Design Property Checking
The goal of this study is to develop scalable statistical algorithms for feasible verification of analog design performances.
Role: PI
SRC: Semiconductor Research Corporation
TxACE: Texas Analog Center of Excellence

NSF CCF-1117660 Li (PI) $225K 08/01/11-07/31/15
Integrated Verification, Built-in Self-test and Tuning for Digitally-Intensive Analog Systems
The goal of this study is to develop formal and semi-formal techniques and methods for verifying and testing the design properties of digitally-intensive analog integrated circuits.
Role: PI

NSF CCF-0917204 Li (PI) 08/01/09-07/31/13
System-Theoretic Analysis and Design for Dynamic Stability of Memory Devices in Nanoscale CMOS and Beyond
The goal of this study is to develop theory, computational methods and tools to characterize dynamic stability of semiconductor and biological memories and enable design of such devices.
Role: PI

Editorial Board Memberships

5. Guest Editor, “Special Section Parallel CAD: Algorithm Design and Programming,” ACM Transactions on Design Automation of Electronic Systems (TODAES), 2010
Students

1. Peter Adkins, B.S.
2. Seungjai Ahn, M.S.
3. Patrick Cramer, B.S.
4. Paul Crouther, B.S.
5. Sai Rameshwar Devarakonda, M.S.
6. Boyuan Gong, M.S.
7. Yukun He, M.S.
8. Hanbin Hu, Ph.D.
9. Nithyashankari Gummidipoondi Jayasankaran, Ph.D.
10. Jimmy (Yingyezhe) Jin, Ph.D.
11. Yu Liu, Ph.D.
12. Amarnath Mahadevuni, M.S.
13. Sushirdeep Narayana, M.S.
14. Sai Sankeerth Nomula, M.S.
15. Deepika Ravipati, M.S.
16. Bala Rottela, M.S.
17. Myung Seok Shim, Ph.D.
18. Jong Hyun Park, B.S.
19. Ya (Tony) Wang, Ph.D.
20. Xin Zhan, Ph.D.
21. Weirui Zhang, Ph.D.
22. Qingshan Zheng, M.S.
23. Zhehui Zhou, M.S.

Refereed Journal Publications

Supervised graduate students/post-doctoral fellows are delineated with an asterisk (*).


20. [J41] [AICSP’13] *Yongtae Kim and Peng Li, “A 0.003-mm2, 0.35-V, 82-pJ/conversion ultra-low power CMOS all digital temperature sensor for on-die thermal management,” Analog Integrated Circuits and Signal Processing, volume 75, issue 1, pp 147-156, April 2013.


Edited Books


Book Chapters (114)


Refereed Conference Publications (128)
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:** Lockless, Steve W.

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

**POSITION TITLE:** Associate 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.*)

<table>
<thead>
<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
<th>Completion Date MM/YYYY</th>
<th>FIELD OF STUDY</th>
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<tbody>
<tr>
<td>Texas A&amp;M University, College Station, TX</td>
<td>B.S.</td>
<td>1994-1997</td>
<td>Molecular &amp; Cellular Biology</td>
</tr>
<tr>
<td>University of Texas Southwestern Medical Center, Dallas, TX</td>
<td>Ph.D.</td>
<td>1997-2002</td>
<td>Molecular Biophysics</td>
</tr>
<tr>
<td>The Rockefeller University, New York, NY</td>
<td>Postdoctoral Associate Research Associate</td>
<td>2002-2007</td>
<td>Molecular Neurobiology and Biophysics</td>
</tr>
<tr>
<td>The Rockefeller University, New York, NY</td>
<td>Research Associate</td>
<td>2007-2009</td>
<td>Chemistry</td>
</tr>
</tbody>
</table>

**Positions and Honors**

**Positions and Employment**

1998-2002 Graduate student in the Department of Pharmacology, University of Texas Southwestern Medical Center in the laboratory of Rama Ranganathan, M.D., Ph.D.

2002-2007 Postdoctoral Associate in the Department of Molecular Neurobiology and Biophysics, The Rockefeller University in the laboratory of Roderick MacKinnon, M.D.

2007-2009 Research Associate in the Department of Synthetic Protein Chemistry, The Rockefeller University in the laboratory of Tom Muir, Ph.D.

2009-2016 Assistant Professor in the Department of Biology, Texas A&M University

2016-present Associate Professor in the Department of Biology, Texas A&M University

**Honors and Awards**

1999 Sigma Xi, UT Southwestern

2000 Biophysics Program Award, UT Southwestern

1999-2002 Fellow, Biophysics Training Grant, UT Southwestern

**Contributions to Science**


* Authors contributed equally to this work.

Research Support
The Welch Foundation (6/1/16 – 5/30/19), Role PI
“Membrane Protein Regulation Through the Lipid Membrane”

National Institutes of Health R01 (8/1/15 – 3/31/20), Role: Collaborator
“Mechanisms of C. difficile spore germination”

National Institutes of Health R01 (1/1/17 – 11/30/17), Role: Subcontract
“Mechanisms of Gating and Permeation in the TrkH K+ Channels”
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: MacNamara, Annmarie

Era Commons User Name (credential, e.g., agency login): aemacnamara

Position Title: Assistant Professor of Psychology

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

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<tr>
<td>McGill University, Montreal, QC</td>
<td>AB</td>
<td>08/2001</td>
<td>Psychology</td>
</tr>
<tr>
<td>Glasgow School of Art, Glasgow</td>
<td>MFA</td>
<td>05/2006</td>
<td>Studio Art</td>
</tr>
<tr>
<td>Stony Brook University, Stony Brook, NY</td>
<td>MA</td>
<td>08/2009</td>
<td>Clinical Psychology</td>
</tr>
<tr>
<td>Stony Brook University, Stony Brook, NY</td>
<td>PHD</td>
<td>08/2013</td>
<td>Clinical Psychology</td>
</tr>
<tr>
<td>University of Illinois at Chicago,</td>
<td>Other training</td>
<td>06/2013</td>
<td>Predoctoral Clinical Psychology</td>
</tr>
<tr>
<td>Chicago, IL</td>
<td>NIH training</td>
<td>06/2015</td>
<td>Internship</td>
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<tr>
<td>University of Illinois at Chicago,</td>
<td>grant</td>
<td></td>
<td>T32 Postdoctoral Research Fellow</td>
</tr>
<tr>
<td>Chicago, IL</td>
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A. Personal Statement

My career path to date and my long term research goals have stemmed from a dedication to achieving an ever-more fine-grained understanding of the mechanisms underlying emotion dysfunction and its manifestation in anxiety disorders. My research to-date has used complementary, layered neurobiological methods (event-related potentials – ERPs, functional magnetic resonance imaging - fMRI, skin conductance response, eyeblink startle) in an effort to close gaps between overlapping phenotypic features and neurobiological dysfunction in anxiety. In the summer of 2016, I began as Assistant Professor in the Dept. of Psychology at Texas A&M University. Shortly after arriving, I established the Multimethod Affect and Cognition lab (MAClab). In the context of my funded NIH K23 award, I have been examining negative emotion processing across a spectrum of anxiety, characterized by increased comorbidity load and negative affectivity, using eyeblink startle simultaneously recorded with blood-oxygenated level dependent response (BOLD) in the fMRI scanner. While this work has extended my prior work on (categorical) anxiety disorders into a transdiagnostic framework, it has not positioned me to examine the unique and interactive influence of depression on negative emotion processing, or to examine the influence of positive affectivity on negative emotion processing in anxiety and depression. This NARSAD Young Investigator’s Award will fill these gaps in my research program, and is the ideal mechanism to leverage my prior experience and funded NIH K23 and put me firmly on the path toward submission of larger scale grants (e.g., an R01 to examine neurobiological means of classifying patients and predicting “real-world”/functional or treatment outcomes) in Year 2 of the Award.


B. Positions and Honors

Positions and Employment
2001 - 2002  Lab Manager, Concordia University, Montreal
2006 - 2007  Lab Manager, New York University, New York, NY
2007 - 2012  Graduate Researcher, Stony Brook University, Stony Brook, NY
2013 - 2013  Predoctoral Clinical Psychology Intern, University of Illinois at Chicago, Chicago, IL
2013 - 2015  T32 Postdoctoral Research Fellow, University of Illinois at Chicago, Chicago, IL
2015 - 2016  Visiting Research Assistant Professor, University of Illinois at Chicago, Chicago, IL
2016 - 2016  Assistant Professor, Texas A&M University, College Station, TX

Other Experience and Professional Memberships
2007 - 2007  Member, Society for Psychophysiological Research
2008 - 2012  Member, American Psychological Association
2013 - 2013  Member, Society for a Science of Clinical Psychology
2013 - 2013  Member, Anxiety and Depression Association of America
2017 - 2017  Member, Society for Affective Science

Honors
2008  Wisconsin Symposium on Emotion Fellow, University of Wisconsin, Madison, WI
2008  Travel Award, Graduate Student Organization, Stony Brook University, NY
2008  Travel Award, Dept. of Psychology, Stony Brook University, NY
2009  Two-week Training Course in fMRI, University of Michigan (NIH sponsored)
2010  Advanced Graduate Research Award, Dept. of Psychology, Stony Brook University, NY
2011  Paper of the Year (Cognitive, Affective and Behavioral Neuroscience), Psychonomic Society
2011  Travel Award, Determinants of Executive Function and Dysfunction, Boulder, CO
2013  Honorable Mention, Research Forum, College of Med, University of Illinois at Chicago, IL
2014  Top Poster, Research Extravaganza, Dept Psychiatry, University of Illinois at Chicago, IL
2014  Travel Award, Society of Biological Psychiatry
2017  Donald F. Klein Early Career Investigator Award, Finalist, Anxiety and Depression Association of America

C. Contribution to Science

1. In the early 2000s, studies showed that cognitive reappraisal could modulate emotional picture processing, as measured using event-related potential component, the late positive potential (LPP). However, questions remained as to the mechanism behind these effects. Through a series of studies, designed to clarify the mechanisms behind reappraisal's effects on the LPP, I showed that a) meaning change - the core component of reappraisal - is sufficient to modulate the LPP; b) that the effects of meaning change persist across time to affect subsequent encounters with the same pictures and c) that electroencephalographic (EEG) alpha power can be used as a measure of engagement of prefrontal brain regions during reappraisal of unpleasant stimuli. Together, this work helped dissect the elements underlying reappraisal's effects on the LPP.

2. Early studies of reappraisal using functional magnetic resonance imaging (fMRI) work revealed that cognitive reappraisal recruits domain-general prefrontal brain regions, such as the dorsolateral prefrontal cortex (dLPFC). Moreover, electrical stimulation of the dLPFC was found to reduce the LPP elicited by unpleasant pictures. Thus, it seemed possible that activation of the DLPFC via other means - e.g., using a task designed to elicit differential levels of activation across conditions - might also reduce the picture-elicited LPP. Indeed, across three separate studies, this is exactly what we found. These studies revealed that a) non-emotional, cognitively demanding tasks are sufficient to modulate the LPP; b) that these effects cannot be attributed to eye gaze toward/away from arousing picture regions and that c) working memory load effects on picture processing are attenuated among participants who are more anxious, suggesting impairments in dLPFC-mediated top-down modulation of picture processing/increased distractibility.


3. Another line of work has investigated how emotion generation goes awry in anxiety and trauma-related disorders. This work has revealed that a) generalized anxiety disorder (both categorical diagnosis and continuous symptoms) is characterized by increased attention towards threatening stimuli, as measured using the LPP; b) that some disorders, such as post-traumatic stress disorder (PTSD) may be characterized by reduced attention toward threatening faces and that c) comorbid depression may attenuate anxiety-related increases normally observed in the threat-elicited LPP. These results suggest that not all emotional disorders are characterized by heightened attention towards threatening stimuli, and underscore the importance of accounting for comorbidities when assessing threat-processing in the anxiety disorders.


4. Impaired emotion regulation is thought to underlie the internalizing disorders, however few studies have directly investigated the brain basis of this dysregulation (i.e., most studies have examined brain reactivity during passive picture viewing, trauma scripts or symptom provocation). My work innovated an emotion regulation paradigm in PTSD, and found that compared to controls, patients with PTSD recruit the dLPFC to a lesser extent during reappraisal of unpleasant pictures. Moreover, I found that 12 weeks of treatment of
with serotonin reuptake inhibitors (SSRIs) restores reappraisal-related recruitment of the dIPFC among those with PTSD. I also found that pre-treatment recruitment of the ventrolateral prefrontal cortex (vPFC) during reappraisal is predictive of treatment outcome, with greater initial deficits in this region predicting increased treatment gain (accounting for baseline symptom severity).


Complete List of Published Work in My Bibliography: [http://1.usa.gov/25xPu3R](http://1.usa.gov/25xPu3R)

D. Additional Information: Research Support and/or Scholastic Performance

**Ongoing Research Support**

K23 MH105553-01A1  
MacNamara, Annmarie Eileen (PI) 
09/17/15-08/31/19 
Brain-Behavior Markers of Negative Affectivity, Comorbidity in Anxiety Disorders 
Role: PI

**Completed Research Support**

T32 MH067631-09  
Rasenick, Mark M. (PI) 
04/01/03-06/30/15 
Training in the Neuroscience of Mental Health 
Role: TA

**Pending Research Support**

National Science Foundation  
Gutierrez-Osuna, Ricardo. (PI) 
08/01/17-07/31/20 
CHS: Medium: Collaborative Research: Managing stress in the workplace: Unobtrusive monitoring and adaptive interventions 
Role: Senior Personnel

NARSAD Young Investigator Award, Brain & Behavior Research Foundation  
MacNamara, Annmarie (PI) 
01/01/18-12/31/19 
Neurobehavioral Correlates of Depression and (Low) Positive Affectivity 
Role: PI
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:** Stephen A. Maren

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

**POSITION TITLE:** Professor of Psychology

**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.)*

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<tr>
<td>University of Illinois – Urbana-Champaign</td>
<td>BS</td>
<td>1989</td>
<td>Psychology</td>
</tr>
<tr>
<td>University of Southern California, Los Angeles, CA</td>
<td>MS</td>
<td>1991</td>
<td>Neurobiology</td>
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<tr>
<td>University of Southern California, Los Angeles, CA</td>
<td>PhD</td>
<td>1993</td>
<td>Neurobiology</td>
</tr>
<tr>
<td>University of California, Los Angeles, CA</td>
<td>--</td>
<td>1996</td>
<td>Behav Neuroscience</td>
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**A. PERSONAL STATEMENT**

My area of research expertise concerns the neurobiology of learning and memory, particularly memory for emotional events such as those established during Pavlovian fear conditioning. For over 20 years my laboratory has worked to define the neural circuits underlying the encoding and retrieval of fear memories. This work has used electrophysiological, behavioral, and immunohistochemical methods to implicate a broad network of brain structures including the amygdala, hippocampus, and medial prefrontal cortex in the regulation of emotional memory. Our most recent work has implicated this network in the contextual control of extinguished fear memories, which is a form of emotion regulation that is disrupted in anxiety disorders including post-traumatic stress disorder. Specifically, the hippocampus is essential for the relapse or “renewal” of fear when an extinguished stimulus is encountered outside the extinction context. The renewal of fear appears to involve reciprocal prefrontal-amygdala circuits that regulate the fear expression. My extensive work in this area provides critical expertise in the neural substrates of fear conditioning and extinction in animal models. Importantly, we have now successfully implemented pharmacogenetic approaches using DREADDs to dissect, in a circuit-specific fashion, the function of hippocampal-prefrontal circuits in the contextual control of learned fear.

**B. POSITIONS AND HONORS**

**Professional Experience**

- 1993 - 1996 Postdoctoral Fellow, University of California, Los Angeles, CA
- 1996 - 2001 Assistant Professor, University of Michigan, Ann Arbor, MI
- 2001 - 2006 Associate Professor, University of Michigan, Ann Arbor, MI
- 2006-2012 Professor, University of Michigan, Ann Arbor, MI
- 2012-present Professor, Texas A&M University, College Station, TX

**Honors and Awards**

- 1987 University of Illinois Edmund J. James Scholarship
- 1987 – 1989 University of Illinois Psychology Honors Program
- 1989 University of Illinois *cum laude* with Distinction in Psychology
- 1989 Phi Beta Kappa Honor Society
- 1989-1993 University of Southern California Dean’s Fellowship
C. CONTRIBUTIONS TO SCIENCE

Dr. Maren has made many seminal contributions to understanding the neurobiological basis of learning and memory. This work has spanned several levels of analysis from receptors and synapses to neural circuits and behavior. His most significant contributions include: 1) demonstrating for the first time that LTP is expressed by increases in the number of postsynaptic glutamate receptors, 2) discovering neuronal correlates of emotional learning and memory in the amygdala, 3) discovering that synaptic plasticity (e.g., long-term potentiation or LTP) in the amygdala supports emotional memory formation, 4) discovering competition and compensation in neural circuits for emotional memory, and 5) defining a neural circuit for the contextual control of emotional memory. Each of these novel contributions has driven both empirical and theoretical work in the field. Collectively, this work has been foundational to understanding the synaptic and circuit mechanisms underlying learning and memory. Maren is currently a Fellow of the American Psychological Association (APA) and the Association for Psychological Science and is the Editor-in-Chief of Behavioural Brain Research. He was the 2001 recipient of the APA Distinguished Scientific Award for an Early Career Contribution to Psychology (Behavioral Neuroscience) and is Past-President of the Pavlovian Society.

Full bibliography:

1) **Long-term potentiation increases postsynaptic glutamate receptor number.** After the discovery of LTP in the early 1970s, there was vigorous debate over the synaptic mechanisms mediating its expression. Two possible mechanisms had been proposed: an increase in presynaptic glutamate release or an increase in the number of postsynaptic glutamate receptors. In the early 1990s, there was considerable evidence suggestive of increased glutamate release, though extant findings could be still accounted for by postsynaptic receptor mechanisms. To address this question, I used selective radiolabeled glutamate receptor ligands (which differentiated NMDA and AMPA receptors) to quantify glutamate receptor populations after LTP induction in vivo (Tocco et al., 1992; Maren et al., 1993). This work, which formed the core of my PhD dissertation, revealed that LTP induction in the hippocampus resulted in a selective increase in the number of postsynaptic AMPA receptors that was highly correlated with LTP magnitude. This finding has now been replicated and extended by several other groups, and an increase in AMPA receptor density is now considered the core mechanism for the expression of LTP. I consider this a seminal discovery in understanding the synaptic mechanisms of memory expression; indeed, AMPA receptor insertion (indexed by GluA1, for example) is now used routinely as a “biomarker” for synaptic sites of memory storage.

Tocco, G., **Maren, S.**, Shors, T. J., Baudry, M., & Thompson, R. F. (1992). Long-term potentiation is associated with increased [H]AMPA binding in rat hippocampus. *Brain Research, 573*:228-234


2) **Amygdaloid neuronal correlates of emotional learning.** In the mid-80s, there was a revolution in the neuroscientific study of emotion in the brain. Joe LeDoux, Bruce Kapp, Mike Davis and others had identified

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Texas A&M Institute for Neuroscience

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the amygdala as an important hub in the learning and memory of emotional memories, but little was known about the function of amygdala neurons in this process. To address this question, I conducted the first single-unit recording study of basolateral amygdala (BLA) neurons during an avoidance task in rabbits; this work formed the basis of my undergraduate honors thesis. I discovered that BLA neurons were among the first to acquire learning-related activity in the brain during conditioning and that amygdala engagement waned as the task became well-learned (Maren et al., 1991). This finding provided the first physiological evidence for two-process models of avoidance learning that posited that avoidance first requires a rapid acquired Pavlovian fear memory (mediated by the amygdala), followed by instrumental habits. It provided the basis for decades of subsequent work exploring the synaptic mechanisms emotional learning in the BLA, which was identified in this early work as a critical locus for fear memory.


3) **Long-term potentiation in the amygdala and emotional memory.** In the early 90s, work on the mechanisms of LTP induction and expression opened avenues for exploring the contributions of these processes to learning and memory. Mike Davis had shown that NMDA receptor antagonists infused into the amygdala prevented the acquisition of fear conditioning, an effect presumably mediated by disruption of LTP induction. To explore this question, I examined whether LTP induction in the amygdala depended on NMDA receptor activation. Early in my post-doctoral work, I developed a novel in vivo LTP preparation to examine plasticity at hippocampal inputs in the BLA. Using this preparation, I was the first to demonstrate that LTP induction in the amygdala in vivo required NMDA receptors—and I also made the novel discovery that NMDA receptors in the BLA were generally involved in neuronal excitability (Maren and Fanselow, 1995). The latter finding made a novel prediction that NMDA receptors should be involved in both the acquisition and expression of fear memories, a result that I later confirmed (Maren et al., 1996). Collectively, these findings were critical for establishing the link between NMDA receptors and synaptic plasticity in the amygdala in vivo to memory formation in behaving animals. This work is now a core element of current synaptic models of fear conditioning, which are centered on LTP in the BLA.


4) **Competition and compensation in fear learning circuits.** In 1992, Mike Fanselow reported that hippocampal (HPC) damage in rats produced a profound time-limited retrograde amnesia for context memories that reproduced the amnesia found in humans with HPC lesions. Because human amnesiacs also have a severe anterograde amnesia (an inability to form new declarative memories), it was believed that HPC lesions should also impair the acquisition of new context memories. To address this question, I began work as a post-doc that was later continued as an Assistant Professor exploring anterograde amnesia for emotional memories after HPC or BLA lesions. The work produced outcomes that were not anticipated by existing neurobiological model of memory formation. Specifically, I observed an absence of anterograde amnesia for contextual memories in animals with either HPC or BLA damage (Maren et al., 1997; Maren, 1998; Maren 1999). This led to a re-conceptualization of the memory processes (and underlying neural circuits) mediating the formation of emotional memory. That is, animals could acquire emotional memories using either rapidly-acquired configural representations (HPC- and BLA-dependent) or more slowly acquired elemental representations (HPC- and BLA-independent). This work has had a major influence on understanding the architecture of memory: the nature of the associative representations acquired by an animal is determined by the memory circuits recruited to (and available for) memory storage.


5) **Neural circuits for contextual control of fear.** Memory retrieval is heavily influenced by the context in which it occurs. In emotional learning and memory tasks, considerable work indicates that memories of safety in particular—that is memories acquired during extinction (when a cue no longer predicts shock) are particularly context-dependent. My laboratory has spent over a decade mapping the neural circuits for contextual control, and we have identified the key elements and connections in these circuits mediating contextual memory retrieval (Corcoran and Maren, 2001; Knapska and Maren, 2009; Orsini et al., 2011; Knapska et al., 2012). This circuit centers on the HPC, medial prefrontal cortex (PFC), and BLA. We have discovered that the hippocampus mediates the return or renewal of extinguished fear that occurs outside the safe context. Hippocampal projections engage reciprocal PFC-BLA circuits that either excite or suppress fear, behavioral outcomes that are dependent on the context in which a fear signal is encountered. This work identifies specific neural connections that can be manipulated to foster fear suppression and dampen fear renewal, information that will be central to developing novel translation strategies for treating clinical disorders of fear and anxiety. Moreover, the work has broad implications for understanding memory disorders, including Alzheimer’s Disease, which are characterized by failures in contextual memory retrieval.


D. RESEARCH SUPPORT

**Ongoing**

R01 MH065961 Maren (PI) 2/1/2015 – 1/30/2020

Neural Substrates of Contextual Memory in Fear Extinction

The long-term goals of this project are to understand the neural systems that mediate the context-dependence of memory, particularly memory for traumatic events. This proposal seeks to characterize the neural circuits and cellular mechanisms by which the hippocampus gates the expression of fear memories after extinction in rats. This work has broad significance for understanding flexible memory representations in the brain and potential clinical interventions to increase the generality and permanence of fear inhibition after extinction-like therapies (e.g., exposure therapy).

Role: PI

McKnight Memory and Cognitive Disorders Award Maren (PI) 2/1/2015 – 1/30/2018

This project focuses on prefrontal-hippocampal interplay in contextual memory retrieval and, in particular, the context-dependence of extinction memories. The major aim of the project is to elucidate the role of the nucleus reuniens, a midline thalamic region by the prefrontal cortex projects to the hippocampus. It is hypothesized that this pathway is critical for cortical circuits to retrieve contextual memories from the hippocampus to guide behavioral performance in a context-specific manner.

Role: PI
NAME: Vani Anshu Dawson Mathur

eRA COMMONS USER NAME: VMATHUR

POSITION TITLE: Assistant Professor, Department of Psychology & Institute for Neuroscience (TAMIN)

EDUCATION/TRAINING

<table>
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<td>Boston University, Boston, MA</td>
<td>B.S.</td>
<td>05/2005</td>
<td>Human Physiology</td>
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<td>Northwestern University, Evanston, IL</td>
<td>M.S.</td>
<td>06/2009</td>
<td>Psychology &amp; Neuroscience</td>
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<td>Northwestern University, Evanston, IL</td>
<td>Ph.D.</td>
<td>06/2012</td>
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<tr>
<td>Johns Hopkins University, Baltimore, MD</td>
<td>post-doc</td>
<td>07/2014</td>
<td>Interdisciplinary Pain Research</td>
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<tr>
<td>University of Maryland, Baltimore, MD</td>
<td>post-doc</td>
<td>07/2015</td>
<td>Neural &amp; Pain Sciences</td>
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Neuroimaging of Pain

Pain Psychophysics

Experimental Social Psychology

B. Positions and Honors

Research Positions
2012-14 Postdoctoral Fellow, Interdisciplinary Training Program in Biobehavioral Pain Research, Johns Hopkins University, Baltimore, MD
2014-15 Postdoctoral Fellow, Neural & Pain Sciences, University of Maryland, Baltimore, MD
2015-Present Assistant Professor of Diversity Science and Well-Being Department of Psychology: Social and Personality Area & Diversity Science Cluster Institute for Neuroscience (TAMIN) Texas A&M University

Honors
2003: National Society of Collegiate Scholars
2004: Golden Key International Honour Society
2005: Cum Laude, Boston University
2006: Women’s Health Research award, Massachusetts General Hospital
2007: Northwestern University Graduate School Research Fellowship
2009: Society, Biology, and Health Cluster Fellowship, Northwestern University
2010: Midwest Pain Society Robert G. Addison and E. Richard Blonsky Research Grant
2010: Philip Brickman Endowment Fellowship, Northwestern University
2012: Northwestern University Graduate Research Grant
2012: Northwestern University Graduate School Career Development Award
2012: Pain Disparities Young Investigator Award, Pain & Disparities SIG, American Pain Society
2012: Interdisciplinary Biobehavioral Pain Research Fellowship, Johns Hopkins University
2013: NIH Health Disparities Loan Repayment Program
2015: Texas A&M University ADVANCE Scholar

C. Contribution to Science

1. Social experiences of discrimination and pain. The overarching goal of my research program is to identify mechanisms underlying social modulation of pain that contribute to pain disparities. Pain disparities are prevalent and well documented. Despite decades of research revealing the profound extent and effects of these disparities, little is known about underlying mechanisms. Examination of the social determinants of pain disparities holds promise to identify social mechanisms that contribute to and help maintain pain disparities. Discrimination is a social determinant that appears to be a significant driver of health disparities, and initial evidence suggests it plays a role in pain sensitization as well. During my post-doctoral fellowship I initiated an analysis of discrimination experienced by patients with sickle cell disease which was part of an ongoing study conducted by my mentor Jennifer Haythornthwaite. In the resulting paper we describe some of the first evidence that racial discrimination in health care settings is associated with enhanced self-reported clinical pain as well as enhanced temporal summation of pain – indicative of pain facilitation. In my own lab, we are extending this line of inquiry to identify the mechanisms underlying the relationship between social insults and pain sensitization. Preliminary findings are being presented at national pain and social psychology conferences this year. Additionally, my colleagues and I were invited by the American Pain Society scientific planning committee to turn our proposed symposium into one of the two longer workshops at the annual meeting. This is evidence of the perceived importance and potential impact of this work on the field.


2. Racial differences in neural empathic response. My master’s thesis research contributed early evidence of racial differences in neural empathic response as well as identified psychosocial factors associated with
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these differences. This work was initially inspired by racial biases observed in helping behavior – specifically in the response observed during the wake of Hurricane Katrina. The resultant publication from my thesis was one of the first papers, in what is now a rapidly growing and influential research area, on the interaction of intergroup processes and empathic brain response. My research in this area has demonstrated that there are racial differences in neural empathic response, but that these differences are not always consistent with outgroup antipathy. Rather, our first paper demonstrated that, while there was no difference in empathic response to racial ingroup or outgroup members in brain regions associated with pain perception, African American participants demonstrated enhanced brain response within the medial prefrontal cortex (mPFC, a region often associated with social cognitive processes including perspective taking) in response to the suffering of ingroup others. Across African American and White participants, mPFC activity predicted enhanced ingroup empathy and altruistic motivation. As the first paper to identify a potential neural mechanism associated with intergroup differences in empathic brain response, this paper contributed to an increased understanding of social-cognitive processes and patterns of brain response underlying intergroup biases in empathy. Additionally, this work contributed to a now growing subfield exploring mechanisms underlying bias in altruistic and compassionate behavior. To further our understanding of the mechanisms that may lead to ingroup favoritism or outgroup antipathy, my collaborators and I have extended this work to examine the effects of other sociocultural factors that enhance neural empathic response in intergroup contexts (e.g., egalitarianism, degree of racial group identification, other-focusedness).


3. Demonstration that implicit biases exist in pain perception. Racial disparities in pain management are well documented and prevalent. Initial approaches to decrease these disparities relied heavily on education and raising awareness of disparities. However, after more than a decade of this approach, inequities persist. One possibility is that, while clinicians consciously strive to provide equitable treatment, implicit biases may affect low level processes, such as pain perception, and operate under the level of conscious regulation. As part of my dissertation, I demonstrated - using controlled experimental social psychological methods - that implicit biases do exist in the low level perception of pain in another person. Participants read identical vignettes describing the pain of a patient. When vignettes were preceded by a rapid-presentation (under the level of conscious recognition) of the face of a Black person, pain perception ratings were lower than those for the identical vignettes preceded by a White face. This effect was not present when faces were explicitly presented along with vignettes, suggesting that implicit biases may persist and affect perception even in the context of explicit motivation to respond without bias. This research is critically important given the continued prevalence of racial disparities in pain and pain treatment. Though researchers previously proposed the potential contribution of implicit biases on pain treatment, this was the first study to demonstrate that implicit biases affect lower levels of perception – which may be more insidious than biases that affect higher level processes such as treatment decisions, and should influence the level at which interventions to combat disparities in pain are targeted. The perceived future impact of this recent paper in the field is indicated by its selection as the Journal of Pain Featured Journal Club Article (May 2014 [http://www.jpain.org/](http://www.jpain.org/)) and inclusion in the outreach focused arm of the Pain Research Forum (June 2016 [http://relief.news/all-pain-is-not-equal/](http://relief.news/all-pain-is-not-equal/)).


4. Altered brain structure and function in chronic migraine. As part of my postdoctoral training in pain imaging under the mentorship of David Seminowicz, I worked on a series of studies examining structural and functional brain alterations in migraine, the relationship between migraine-related brain changes and clinical outcomes, and the effectiveness of mindfulness meditation interventions on reversing these changes. My primary contribution to this work was data analysis and interpretation. Migraine has traditionally been considered an episodic disorder. However, brain alterations in migraine provide compelling evidence that it
may be better characterized as a chronic pain disorder. In recent and ongoing work we have identified 1) alterations in gray matter volume; 2) disrupted resting state functional connectivity between the default-mode network and pain-, cognitive-, visual-, and sensorimotor-related networks; and 3) altered cognitive processing among patients with chronic migraine during interictal (non-attack) periods. This suggests that migraine restructures key brain networks, thus altering the processing of cognitive and pain-related information. Furthermore, we have found an association between altered resting state and cognitive brain function and increased pain catastrophizing – a maladaptive cognitive coping strategy often associated with poorer pain-related clinical outcomes. Results suggest pain catastrophizing and other pain-related cognitions may be tractable targets for interventions to potentially slow or reverse the alterations in brain structure and function, and affect clinical outcomes. Ongoing studies in Dr. Seminowicz’s lab are building upon these initial findings.


**Complete List of Published Work in My Bibliography**

**D. Research Support**

**Ongoing:**
- Texas A&M Division of Research PESCA Grant
  - *Neural Mechanisms of Attention to Pain Cues*
  - 05/01/17-4/30/18
  - Role: Co-Principal Investigator with Brian Anderson

**Completed in the last three years:**
- National Institutes of Health Ruth L. Kirschstein National Research Service Award T32 NS 070302-08
  - Program Directors: Gayle G. Page, RN, DNSc & Jennifer A. Haythornthwaite, PhD
  - Goal: Prepare fellows to address the complex challenge of pain through interdisciplinary training including coursework and collaborative mentorship.
  - Role: Postdoctoral Fellow (July 2012-July 2014)

- National Institutes of Health 1R21NS074017-01A1
  - Racial disparities in pain experience: Neuroimaging and behavioral investigations
  - Goal: Use functional neuroimaging (fMRI) and behavioral paradigms to examine effects of race on neural response to pain experience as well as effects of racial stereotypes and prejudice on perception and diagnosis of pain in a medical and non-medical setting.
  - Principal Investigator: Joan Y. Chiao, Ph.D.
  - Role: Co-Investigator, Dissertation (2011-2013)
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: Uel Jackson McMahan

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

POSITION TITLE: 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.)

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<td>Westminster College, Fulton, MO</td>
<td>B.A.</td>
<td>05/1960</td>
<td>Biology</td>
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<tr>
<td>University of Tenn. Medical Units, Memphis, TN</td>
<td>Ph.D.</td>
<td>05/1964</td>
<td>Anatomy</td>
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NOTE: The Biographical Sketch may not exceed five pages. Follow the formats and instructions below.

A. Personal Statement

The proposed study relies on mapping the macromolecular architecture of synaptic basal lamina at neuromuscular junctions by electron tomography and identifying macromolecules containing the C-terminus of neural agrin by histochemistry. My lab was among the first to develop a software package (EM3D) for imaging by electron tomography macromolecules such as those in the basal lamina. We have used it for studying the structure and function of the macromolecular assembly known as active zone material and other macromolecular components of the active zone of axon terminals for more than 15 years. I have directly participated in all of the studies, from tissue preparation for electron tomography to data collection to computational tomographic analysis. The studies in this series required considerable effort and skill in pattern recognition and considerable quantitation, all of which required considerable time. Thus the frequency of publication has not been as great as for studies requiring different sorts of skills. Prior to undertaking the development of electron tomography for understanding the structure and function of active zone material, my lab spent decades on studies aimed at understanding how the axon terminals at developing and regenerating neuromuscular junctions direct the formation and maintenance of the postsynaptic apparatus on muscle fibers. These studies involved a variety of cell biological/biochemical approaches including histochemistry and led to the discovery and initial characterization of agrin, which is now widely recognized to be the principal signaling molecule secreted by axon terminals to direct the formation and maintenance of the postsynaptic apparatus. The C-terminus of the protein is the portion active in this process.

B. Positions and Honors

Positions
1965-67  Instructor, Department of Anatomy, Yale University School of Medicine.
1967-72  Instructor, Department of Neurobiology, Harvard Medical School.
1972-75  Assistant Professor, Department of Neurobiology, Harvard Medical School.
1975-77  Associate Professor, Department of Neurobiology, Harvard Medical School.
1977-02  Professor of Neurobiology, Stanford University School of Medicine.
1986-91  Director, Interdepartmental Neurosciences Ph.D. Program, Stanford University.
1987-92  Chairman, Department of Neurobiology, Stanford University School of Medicine.
1989-90  Chairman, Committee on Graduate Studies, Stanford University.
2002-08  Professor of Neurobiology and of Structural Biology, Stanford Univ Sch. of Med.
2008-  Emeritus Professor of Neurobiology and of Structural Biology, Stanford University
2008-13  Head, Department of Biology, Texas A&M University
2008-  Professor, Department of Biology, Texas A&M University

Other experience and professional memberships
1984  Principal Instructor, Stanford Summer Course-3wk: Cell & Molecular Biol. of the Synapse.
2000-  Lecturer, IBRO-VLTP Courses in Neuroscience-9da: Nigeria (2X), Cuba, Vietnam (2X), Iran, Argentina, Poland (2X), China (4X), Turkey, Uganda (2X), Costa Rica, Romania, Kenya, Jordan, Russia, Estonia, India, Ecuador, Cameroon, Guatemala, Ethiopia, Chile, Sri Lanka, Latvia, Paraguay, Iraq (Kurdistan), Albania, Israel (East Jerusalem), Brazil.
2003-  Director, International Brain Research Organization’s Visiting Lecture Team Program.

Honors
1973-77  Research Career Development Award (NIH).
1984-91  Jacob Javits Neurosciences Investigator Award (NIH).
1991-98  Jacob Javits Neurosciences Investigator Award (NIH).
1998  Fondation IPSEN/Fondation de France Prix (Plasticite Neuronale) shared with Dr. G. Fischbach (Harvard) and Dr. H. Betz (MPI, Frankfurt).

C. Contribution to Science

1. Showing that the position of individual axon terminals at synapses in autonomic ganglia and of neuromuscular junctions can be observed in living isolated preparations by Nomarski DIC optics. Although DIC optics had been used in a previous publication by others to examine the trafficking of large organelles, this was its first application to the study of cellular topography, in general, and synapses, in particular, in live preparations, and it led to the widespread use of DIC optics for these purposes today.

   Showing that the muscle fiber's surface directly opposite the axon terminal at neuromuscular junctions (the postsynaptic membrane) has a far greater sensitivity to the direct iontophoretic application of the neurotransmitter acetylcholine than it does a few micrometers away. This study, which relied on the use of Nomarski DIC optics to visualize axon terminals on muscle fibers, together with a study published by others the same year, which relied on α-bungarotoxin labeling, provided the first direct evidence that receptors for neurotransmitter are highly concentrated in the postsynaptic membrane.


2. Determining that acetylcholinesterase, which degrades acetylcholine after its interaction with the muscle
fiber has terminated, is a component of the portion of the muscle fiber's basal lamina sheath that occupies the synaptic cleft between the axon terminal and muscle fiber.

Finding that after damage to a motor nerve in the frog, regenerating axon terminals grow to precisely cover the portion of a muscle fiber's surface that was formerly opposite the original axon terminals. It had been shown around the turn of the twentieth century that regenerating axon terminals reinnervate the general area of a muscle fiber that was the home of the original axon terminals, the endplate region. Knowledge about the precision of reinnervation of the narrow postsynaptic membrane within the endplate region was an essential step toward learning that the synaptic basal lamina contains synaptogenic proteins, which ultimately led to the discovery of agrin as described below.

Demonstrating that when a frog muscle is damaged in a way that causes the muscle fibers to degenerate but leaves their basal lamina sheaths intact, including that which lies between the axon terminal and the postsynaptic membrane, damaged axons regenerate to precisely cover the sites on the sheaths formerly occupied by the original axon terminals despite the absence of muscle fibers.


3. Showing that the portion of the muscle fiber's basal lamina sheath that occupies the synaptic cleft at the frog's neuromuscular junction contains synaptogenic proteins that induce the accumulation of synaptic vesicles and the formation of active zones in regenerating axon terminals, which are major constituents of the presynaptic apparatus of axon terminals at normal and regenerating neuromuscular junctions and are directly involved in the exocytosis of the neurotransmitter acetylcholine during synaptic transmission.

Finding that the portion of the muscle fiber's basal lamina sheath that occupies the synaptic cleft at the frog's neuromuscular junction contains synaptogenic proteins that induce regenerating muscle fibers to form on their surface aggregates of acetylcholine receptors and acetylcholinesterase, which are major constituents of the postsynaptic apparatus at normal and regenerating neuromuscular junctions and are required for synaptic transmission.


4. Isolating, identifying and initially characterizing the protein agrin, and proposing that agrin secreted by nerve terminals into the synaptic cleft directs the formation and maintenance of the postsynaptic apparatus on muscle fibers. This led to many studies by others on agrin's structure and mechanism of action including the discovery of its muscle fiber receptors and the observation that genetic defects in or autoimmune action against agrin and its receptors correlate with myasthenias (congenital myasthenia and myasthenia gravis) in some humans suffering the disease.


5. Using electron microscope tomography on sections from frog and mouse neuromuscular junctions to learn
that the dense aggregates of proteins attached to the presynaptic membrane of typical synapses, known as
active zone material, contain an organized network of elongate macromolecules. Our results have led to the
concept that the macromolecules help dock synaptic vesicles at the presynaptic membrane in an orderly and
specific way. That is, certain active zone material macromolecules connect to specific sites on the membrane
of undocked synaptic vesicles, orienting a predetermined fusion domain in the vesicle membrane toward the
presynaptic membrane while bringing the two membranes together. Our results also indicate that the
macromolecules regulate the priming of each synaptic vesicle after it has docked and they anchor calcium
channels in the presynaptic membrane near docking sites. Certain of the macromolecules most likely contain
proteins that mediate the calcium-induced exocytosis of acetylcholine from the docked vesicles into the
synaptic cleft upon arrival of a nerve impulse. The studies relied on development of the software package
EM3D.

synaptic vesicle docking by different classes of macromolecules in active zone material. PLoS ONE
vesicles with the macromolecules in active zone material that direct vesicle docking. PLoS ONE
8(7):e69410. doi: 10.1371/journal.pone.0069410. * co-primary authors.
(2009) Macromolecular connections of active zone material to docked synaptic vesicles and

Complete List of Published Work in MyBibliography:


D. Research Support

I was the PI on the National Institute of Neurological Disorders and Stroke Grants NS014506 and NS007158
and the National Institute of Mental Health, Human Brain Project/Neuroinformatics Grant MH068065.
**NAME:** MARY MEAGHER

eRA COMMONS USER NAME (credential, e.g., agency login): m-meagher

**POSITION TITLE:** Professor, Psychology and Neuroscience

**EDUCATION/TRAINING**

<table>
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<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
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<td>Nazareth College of Rochester</td>
<td>B.S.</td>
<td>05/1982</td>
<td>Psychology major/Biology minor</td>
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<tr>
<td>University of North Carolina at Chapel Hill</td>
<td>Ph.D.</td>
<td>05/1989</td>
<td>Psychology major (Behavioral Neuroscience)/Neurobiology minor</td>
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<tr>
<td>Texas A&amp;M University</td>
<td>Postdoc</td>
<td>05/1993</td>
<td>Clinical Psychology</td>
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<tr>
<td>San Antonio VA Medical Center</td>
<td>APA Clinical Internship</td>
<td>08/1994</td>
<td>Clinical Psychology</td>
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**A. Personal Statement**

The goal of this project is to identify pain gene expression signatures in a mouse xenograft model of endometriosis. This proposal builds on a successful R21 collaboration with Dr. Arosh where we investigated the role of prostaglandin E2 signaling on endometriosis pain. The results published in the *Proceedings of the National Academy of Sciences* in 2015. In that study, mechanical hyperalgesia was assessed by stimulating the pelvic floor with calibrated von-Frey filaments to determine the force required to elicit a behavioral withdrawal response. We showed that peritoneal endometriosis decreased pelvic floor withdrawal threshold, indicating increased mechanical pain sensitivity. Moreover, we showed that mechanical sensitivity was correlated with growth of endometriosis lesions. We also showed that inhibition of EP2/EP4 increased pelvic floor pain threshold, indicating a decrease in mechanical pain sensitivity. These findings support the validity of this approach to pain measurement. As a behavioral neuroscientist, I bring expertise in the assessment of pain behaviors and the neural basis of pain processing. My research program uses both mouse and rat models to study pain mechanisms. My early research investigated how exposure to stressors can either inhibit or facilitate pain by engaging endogenous modulatory systems at multiple levels of the neural axis. Notably, we were one of the first laboratories to elucidate the role of the prefrontal cortex, amygdala, BNST, and dorsolateral PAG in mediating stress-induced hyperalgesia. My expertise in this area will help me to assist Dr. Arosh in dissecting different brain regions associated with ascending and descending pain processing. My more recent research investigates the neuroimmune mechanisms mediating the adverse behavioral and pathogenic effects of stress on a mouse model of multiple sclerosis. I also bring expertise in clinical health psychology, with a focus on pain. My recent work examines the effects of adverse life events and alcohol abuse on pain sensitization using quantitative sensory testing procedures combined with psychophysiological, endocrine, inflammatory, and genomics approaches to elucidate the underlying mechanisms.

This exploratory R21 proposal represents a new line of research that leverages Dr. Arosh’s well-characterized animal model of endometriosis to identify CNS gene expression signatures. As co-I, my role will be to assist Dr. Arosh in the measurement of the pain behaviors and in the dissections of brain regions involved in pain processing. I will help to train the graduate students involved in this project. Through my experience as PI and co-I on several interdisciplinary NIH, NMSS, and NSF projects, I have developed strong collaborative and organizational skills. Together, these factors support the feasibility of this project and the likelihood of success.

**B. Positions and Honors**

**Positions and Employment**

1989-1994 Visiting Assistant Professor, Department of Psychology, Texas A&M University
1994 Assistant Professor, Department of Psychology, Texas A&M University
1994- Texas A&M Institute of Neuroscience, Interdisciplinary Faculty Member
Other Experience

1998 NIH Interdisciplinary Workshop in Psychoneuroimmunology Participant, March 26-30
2004-06 American Psychological Association, Committee on Animal Research & Ethics
2005-06 NIH Biobehavioral Mechanisms of Emotion, Stress & Health [MESH] Study Section
2008-10 American Psychological Association, Scientific Leadership Representative
2009-11 NIH Chronic Fatigue Syndrome and Fibromyalgia [ZRG1 CFS-M] Study Section
2011-13 NIH Biobehavioral Mechanisms of Emotion Stress and Health (MESH) Study Section
2015-16 NIH Biobehavioral Mechanisms of Emotion Stress and Health (MESH) Study Section
2017 NIH Biobehavioral and Behavioral Processes [ZRG1 BBBP-Z (04) M ] Study Section

Honors and Awards

2003 American Psychological Association Fellow - Behavioral Neuroscience/Division 6
2004 Women’s Progress Faculty Award, Texas A&M University
2009 American Psychological Association Fellow - Clinical Psychology/Division 12
2009 Research Excellence Award, TAMU Women’s Former Students Association
2008-12 Cornerstone Fellow, College of Liberal Arts, Texas A&M University
2015 American Psychological Science Fellow

Society Memberships:
Society for Neuroscience, American Psychological Association, American Pain Society, International Association for the Study of Pain, Psychoneuroimmunology Research Society, American Psychological Society

C. Contributions to Science

1. My early animal research investigated the organization of endogenous pain inhibitory systems using the stress-induced hypoalgesia paradigm. Stressor severity was found to determine whether forebrain, brainstem or intraspinal systems mediate stress-induced hypoalgesia using spinal nociceptive reflex measures. We also showed that Steven’s power law could be used to predict when these inhibitory systems are engaged. Additional research showed that spinal systems exhibit simple forms of learning and memory-like phenomena similar to those observed in invertebrate organisms by Kandel and colleagues. Specifically, we demonstrated that Pavlovian conditioned antinociception can be observed after spinal transection. This line of work involved a collaboration with Jim Grau who’s expertise in Pavlovian conditioning and stress-induced hypoalgesia complemented my training in behavioral neuroscience and my graduate work in Michela Gallagher’s laboratory. Two graduate (**) and undergraduate co-authors (**) were placed in postdoctoral and faculty positions while one was placed in an MD/PhD program at UTMB where he completed his PhD under Bill Willis (Chen).

   d. Meagher MW, **Chen P, **Salinas JA, Grau JW (1993). Activation of the opioid and nonopioid hypoalgesic systems at the level of the brainstem and spinal cord: Does a coulometric relation predict the emergence or form of environmentally-induced hypoalgesia? Behav Neurosci 107, 493-505. PMC: 8392349

2. Later studies revealed that exposure to aversive stimuli could sometimes induce pain facilitation on supraspinal measures of pain (e.g., vocalization thresholds and associative learning measures). These publications examined the conditions under which pain facilitatory versus inhibitory processes are engaged and the neural systems that mediate shock-induced hyperalgesia. We showed that mild to moderately-intense shocks enhanced pain by engaging the prefrontal cortex, amygdala (CeA, BNST) and dIPAG, whereas more severe shocks inhibited pain by activating brainstem systems. These early findings fit with subsequent human research indicating that long-term sensitization of these defensive circuits by trauma and emotional learning contributes to pain facilitation in clinical pain disorders. Five graduate student co-
4. My human pain laboratory uses quantitative sensory testing (QST) methods to conduct basic mechanistic studies in humans, providing a bridge between animal laboratory and clinical pain research. Early studies sought to characterize the emotional state associated with the induction of stress-induced hypoalgesia versus hyperalgesia in healthy humans using shock, anticipation of shock, startling noise, and Pavlovian fear conditioning. Other studies used affective pictures to modulate pain. We found that pain facilitation was linked to the induction of anxiety, or anxious apprehension, while pain inhibition was linked to highly arousing fear-alarm reactions. These studies were among the first to demonstrate the pain modulatory

3. I have been PI or co-I on NIH and National Multiple Sclerosis Society grants investigating the effects of psychosocial stressors on an animal model of multiple sclerosis, Thie"er’s virus induced demyelination. This project involves a collaboration with CJR Welsh, a neuroimmunologist with expertise in Thie"er’s virus that complements my background in behavioral neuroscience, stress, and psychoneuroimmunology research. Our last R01 (Meagher PI) examined the role of proinflammatory cytokines in mediating the adverse effects of social stress on disease course. We showed that central interleukin-6 (IL-6), released during repeated social stress, exacerbated disease course by priming neuroinflammation and by suppressing virus-specific T cell responses in CNS. We also showed that early life events can alter disease severity when mice are subsequently infected during adolescence. We found that neonatal maternal separation exacerbated disease course by disrupting viral clearance from CNS. In contrast, subsequent work indicated that brief neonatal handling had protective effects, leading to blunted corticosterone responses to stress and decreased autoimmune disease severity in non-stressed adolescent mice. However, neonatal handling led to increased disease severity when paired with later social stress during adolescence. These findings suggest that early life experiences leading to hypo-responsiveness of the HPA axis interact with later social stress to increase vulnerability to infectious and autoimmune disease. Recent findings indicate that exposure to social stress prior to infection increases pain behavior and impairs hippocampal-dependent memory consolidation during the demyelinating phase of disease. My work on neuroinflammation, fatigue and pain had important implications for cancer treatment-induced symptoms, and led to an invited Nature Reviews Clinical Oncology with Robert Dantzer. My skills in pain assessment also led to a R21 and PNAS article with Joe Arosh investigating the role of prostaglandin E2 signaling in a mouse model of endometriosis. My graduate student co-authors* have been placed in tenure-track, postdocs, and industry research positions. Two student co-authors (Johnson, Vichaya) were awarded NSF fellowships and Johnson also received an F31 NRSA fellowship.

4. My human pain laboratory uses quantitative sensory testing (QST) methods to conduct basic mechanistic studies in humans, providing a bridge between animal laboratory and clinical pain research. Early studies sought to characterize the emotional state associated with the induction of stress-induced hypoalgesia versus hyperalgesia in healthy humans using shock, anticipation of shock, startling noise, and Pavlovian fear conditioning. Other studies used affective pictures to modulate pain. We found that pain facilitation was linked to the induction of anxiety, or anxious apprehension, while pain inhibition was linked to highly arousing fear-alarm reactions. These studies were among the first to demonstrate the pain modulatory
effects of emotion using human experimental procedures. This led to an invited review published in *Current Opinion in Psychiatry*. Both graduate student co-authors* are now tenured associate professors (U. Tulsa, U. Mississippi). Notably, my former doctoral student, Jamie Rhudy, extended this line of work by examining the effects of emotion on spinal versus supraspinal measures of pain in humans.


5. Psychosocial or physical stressors have been shown to increase pain severity and chronicity. However, the mechanisms underlying this association remain disputed. Animal research suggests that activation of the stress axis by uncontrollable stressors or alcohol abuse can increase pain sensitivity and persistence by enhancing peripheral and central sensitization mechanisms. Using QST methods, my laboratory recently examined whether these findings translate to humans. We found that young adults reporting early life adversity show enhanced pain sensitivity on tests assessing “central sensitization”, a neurobiological mechanism that contributes to the development and maintenance of chronic pain. Importantly, we found that this stress-induced increase in pain sensitivity could be reversed through a psychological intervention that helps the individual process their most traumatic experience. Most recently, we found that early childhood adversity increases the risk of developing chronic pain in young adults. The long-term goals of this work are to: (a) identify the mechanisms underlying the increase in chronic pain following adverse life events, and (b) evaluate whether psychological and pharmacological interventions that target these mechanisms can prevent the development of chronic pain. A recent line of work examines the role of the stress axis in mediating the effects of binge drinking on muscle pain sensitization in young adult binge drinkers. My doctoral student, Dokyoung You, was recently awarded an F31 NRSA fellowship to investigate this topic. My graduate student co-authors* have been placed in tenure-track (U Conn), postdoc (with Gebhardt, Dantzer, Cleeland), and VA clinical and research positions (e.g. Providence VAMC, Brown Univ Medical School, San Antonio Military Medical Center).


For a complete listing of Meagher’s publications see: [https://www.researchgate.net/profile/Mary_Meagher](https://www.researchgate.net/profile/Mary_Meagher)

**D. Research Support**

Ongoing Research Support:

**F31AA023709-01A1**

Effect of Alcohol Withdrawal on Pain Sensitization  
National Research Service Award, Individual Predoctoral Fellowship  
Role: Sponsor for Dokyoung Sophia You

**One Health Grant**

Mechanisms mediating pain sensitization following sexual assault  
Role: PI

**2 T32 OD011083-06**

Institutional Training Grant for Comparative Biomedical Research Training for Veterinarians.
**Completed Research Support:**

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<td>R21 DA034285-01</td>
<td>Ditre</td>
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<td>Effects of Smoking Abstinence on Pain Reactivity: A Human Experimental Model</td>
<td>8/01/12</td>
<td>03/31/16</td>
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<td>R21 HD066248-01A1</td>
<td>Arosh</td>
<td>Meagher</td>
<td>Prostaglandin E2 Signaling in Growth and Pain of Endometriosis</td>
<td>10/01/11</td>
<td>03/31/15</td>
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<td>TBSI Seed Grant</td>
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<td>Effects of psychogenic stress on pain after spine surgery: the moderating/mediating role of cytokines.</td>
<td>6/01/12</td>
<td>03/31/16</td>
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<td>T32OD011083-05</td>
<td>Kier</td>
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<td>Role: Training Faculty, “Comparative Biomedical Research Training for Veterinarians”</td>
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<td>R01NS060822</td>
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<td>Role of social stress-induced cytokines in exacerbating an animal model of MS</td>
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<td>NIH/T32 MH65728-01</td>
<td>Gonzalez-Lima</td>
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<td>Behavioral Neuroscience Minority Training Grant Role: Executive Committee and training faculty</td>
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<td>R01AG07805</td>
<td>Griffith</td>
<td>Meagher</td>
<td>Physiology of cholinergic basal forebrain neurons</td>
<td>10/1/05</td>
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<td>NSF Graduate Fellowship</td>
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<td>Role: Sponsor for Elisabeth Good-Vichaya, “Exaggerated pain states in Theiler’s virus infection”</td>
<td>2005-2008</td>
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<td>NSF Graduate Fellowship</td>
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<td>RG3128</td>
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<td>Sensitization: Behavioral properties &amp; neural mechanisms</td>
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<td>Conditioned changes in pain reactivity: The variables determining the direction and the form of the conditioned response</td>
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A. Personal Statement

My role in the proposed project is that of PD/PI. The objective of the proposed work is to develop a reliable fatigue assessment survey and a personalized fatigue prediction tool for the oil and gas extraction (OGE) workers. I have the expertise, leadership, and motivation necessary to successfully carry out the proposed work. I am Assistant Professor in the Department of Environmental and Occupational Health in the School of Public Health Texas A&M Health Science Center, graduate faculty with the Texas A&M Institute for Neuroscience at Texas A&M University, director of the Neuroergonomics & Biomechanics Lab, and co-director of the Texas A&M Ergonomics Center. My research is focused on exploring how work-related factors (physical and mental workload), individual (i.e., obesity, aging) and psychosocial factors influence human capacity, particularly worker fatigue, which has resulted in several peer-reviewed journal publications.

Fatigue in OGE workers is implicated as a critical cause of occupational injuries/accidents, and is a grave risk factor for operator health and safety. The absence of evidence-based comprehensive fatigue monitoring approaches in this highly hazardous environment is deeply troubling. This industry has often witnessed the detrimental impacts of competitive business demands on safety behaviors (or lack of) at the individual and organizational levels. To be able to develop an effective assessment tool that has a strong chance of being adopted by the OGE industry, where applicable safety regulations are not in place, is highly motivating to me for two reasons. First, I am an occupational safety and health faculty in the Texas A&M School of Public Health and historically a majority of our graduate students are future oil and gas occupational health and safety professionals. Important knowledge gained with the proposed research can better educate (and safeguard) our future safety practitioners to address and mitigate fatigue concerns in the OGE workforce. Second, my expertise and experience with fatigue-related research have previously employed detailed physiological understanding of the impact of fatigue on worker capability, and I plan to utilize these competencies (particularly ambulatory physiological monitoring) to develop a practical, feasible, and safe fatigue assessment method tailored to OGE workers. I have conducted several laboratory- and field-based fatigue investigations, and I am strongly qualified to conduct the proposed work in challenging OGE environments.

I currently serve as a PI on a 3-year NIH grant focusing on fatigue in obese and older adults, a 1-year National Academies of Sciences grant focusing on fatigue in offshore oil and gas workers, and several industry research projects on understanding work-related risk factors on worker discomfort and productivity. In addition to serving as a Co-I on several federal and industry grants, I also served as a Co-PI on a 2-year NIOSH-funded project to develop revised force-endurance models that account for the shift in worker demographics to an older and more obese worker population.
I will oversee the general project management of the proposed work and will mentor and supervise the postdoctoral personnel in data collection, analyses, and inferences. Dr. S. Camille Peres, Co-I, is a trained cognitive psychologist and an experimental design/statistics expert. She will provide her guidance in survey development, qualitative methods, and statistical analyses. Moreover, her recent experiences leading industry-led research projects to understand human factors challenges in the oil and gas extraction industry will tremendously benefit translate the proposed study “outputs” into “outcomes”. Drs. Lawley and Erraguntla will provide their expertise in developing machine learning models and in managing the large/complex data sets. Dr. Sam Mannan, Co-I and senior mentor to the project, will provide guidance in strengthening our existing industry-academic partnerships and in understanding the potential implications of the study outputs and outcomes on safety regulations. Dr. Mannan is a renowned researcher in process safety in petrochemical industries and has been integral in developing the API 755 Fatigue Risk Management Systems guidelines for onshore OGE operations. Finally, Dr. Miller will serve as a consultant on this proposal and will provide his expertise in practical fatigue assessments with psychophysiological data. I am confident that Drs. Mannan’s and Miller’s mentorship and guidance will enhance our study protocols, analyses, and implications for practices. I am confident that the research team has the appropriate experiences and expertise in successfully conducting this study.


### B. Positions

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<tr>
<td>Summer 2011</td>
<td>Instructor, Industrial and Systems Engineering, Virginia Tech</td>
</tr>
<tr>
<td>2011–2012</td>
<td>Assistant Professor, Cognitive and Learning Sciences, Kinesiology and Integrative Physiology, Michigan Technological University.</td>
</tr>
<tr>
<td>01/2013–Present</td>
<td>Assistant Professor, Environmental and Occupational Health, Texas A&amp;M Health Science Center; Director, Neuroergonomics &amp; Biomechanics Laboratory</td>
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<tr>
<td>07/2013–Present</td>
<td>Co-Director, Texas A&amp;M Ergonomics Center</td>
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<tr>
<td>05/2014</td>
<td>Graduate Faculty, Texas A&amp;M Institute for Neuroscience (TAMIN)</td>
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### Professional service

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<tr>
<td>2012-present</td>
<td>Editorial board, <em>Work: A Journal of Prevention, Assessment and Rehabilitation</em></td>
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<tr>
<td>2014-2016</td>
<td>Guest Editor, “<em>Integrating Physical and Cognitive Ergonomics</em>”, IIE Transactions on Occupational Ergonomics and Human Factors</td>
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<tr>
<td>2015</td>
<td>Reviewer, National Science Foundation (NSF) Graduate Research Fellowships</td>
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<td>2016</td>
<td>Reviewer, National Institutes of Health, Aging Systems and Geriatrics Study Section</td>
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### Honors and Awards

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<td>2008</td>
<td>Alpha Pi Mu Honor Society</td>
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</table>
C. Contribution to Science

Worker fatigue: To reduce the increasing costs of fatigue injuries and to improve workers’ quality of life, efforts are needed to help understand the causes of injuries and to facilitate development of safety guidelines and interventions. My efforts, listed below, outlined a comprehensive understanding of the interactive effects of physical and mental fatigue on (ambulatory) physiological responses, such as muscle activity, heart rate, heart rate variability, and muscle oxygenation, and their perceptions of workload/fatigue. These findings may facilitate the development of task design strategies to help reduce the risk of workplace injuries and to increase worker safety.

Our pilot study with oil and gas industry has provided the rationale and basis for our study hypothesis, and while our publication is being prepared for journal submission, I have presented the pilot study findings to the Houston Human Factors and Ergonomics Society.


Neurophysiological fatigue: In addition to the traditional physiological indicators of fatigue (i.e., EMG, heart rate, heart rate variability), brain imaging during fatigue development indicated that compared to the control, a critical reduction in oxygenation in the bilateral prefrontal cortex was observed during submaximal fatigue-inducing contractions associated with mental fatigue at exhaustion. Using a neuroergonomics approach, my studies provide the evidence that neural interference at the prefrontal cortex may influence physical fatigue development during tasks that are associated with extreme mental stress.


Changing workforce characteristics: Although the health consequences of obesity have gained increasing attention in the literature, there are still major limitations in what is known regarding the effects of obesity on physical and mental fatigue. My research with obesity and fatigue outcomes employs both brain and behavior assessments to
understand if obesity is associated with changes in brain function that impact an obese individual’s physical and cognitive capabilities to perform work safely.

1. Mehta, RK. & Cavuoto, LA. (Accepted). Relationship between BMI and fatigability is task-dependent. Human Factors.

**Occupational and Environmental Health:** One of the main focuses of my laboratory is to conduct examinations on environmental factors impacting health and safety. Other ergonomic research includes developing/implementing interventions to improve healthy behaviors in workplaces.


**Complete List of Published Work in MyBibliography:**

### D. Research Support

**Ongoing Research Support**

*Factoring in the Human in Offshore Operations: Forces for Scenario Planning*

<table>
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<th>Project Code</th>
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<td>2000007355</td>
<td>Mehta</td>
<td>PI</td>
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National Academies of Science, Engineering, and Medicine
The objective of the project is to systematically explore different scenarios to determine fatigue-related variability in operator performances and physiological responses during simulated drilling operations.
Role: PI

*Obesity, Stress, and Neuromuscular Functioning in the Elderly*

<table>
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<td>1R15AG047553 - 01A1</td>
<td>Mehta (PI)</td>
<td>PI</td>
<td>9/2015-8/2018</td>
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</table>

NIH-NIA
This proposal will 1) determine the impact of obesity on upper and lower extremity neuromuscular fatigue and associated neural activation patterns in older adults, and 2) identify the neural mechanisms that contribute to impaired motor function under stress with obesity older adults, that can inform development of intervention strategies to promote brain (and subsequently) physical health.
Role: PI
Fatigue risk management research in the energy sector
Seed Grant Mehta (PI) 2015-2017
School of Public Health/Mary Kay O’Connor Process Safety Center
This project aims to strengthen industry-academic partnerships and collect pilot data to examine critical research gaps in fatigue monitoring and management in the energy sector.
Role: PI

Texas A&M Health Science Center Occupational Health & Safety Program
5T03OH009410-05 Benden (PI) 7/2012-6/2017
NIOSH
The project will deliver focused training in occupational safety and health, with available concentrations in safety, health, and ergonomics for masters students.
Role: Co-I

Cognitive ergonomics assessment of office furniture
Research Contract Mehta (PI) 2/2017-8/2017
Herman Miller
The objective of the grant is to evaluate the cognitive impacts and physical discomfort associated with the use of various office furniture.
Role: PI

Validation of an ergonomic assessment tool
Research Contract Mehta (PI) 1/2016-12/2016
Office Ergonomics Research Committee
The objective of the grant is to validate a self-report tool sensitive enough to evaluate ergonomic risks of electronic devices.
Role: PI

Select Completed Research Support

Revised Force-Endurance Models for the US Workforce
1R03OH010547-01 Cauuto (PI) 9/2014-8/2016
NIOSH
This project determined the extent of endurance differences based on obesity level to provide ergonomics practitioners with a revised tool to enable the prediction of endurance time inclusive of personal factors.
Role: PI (sub-contract)

Influence of workplace stress and personal factors on musculoskeletal disorders
5T42OH008421 Mehta (PI) 7/2012-6/2013
CDC NIOSH through Southwest Center for Occupational and Environmental Health
The goal of this pilot study was to evaluate the influence of stress on obesity- and age-related differences in fatigability and cognitive function. Outcome measures included endurance and fatigue-related responses (strength decline, changes in EMG, heart rate, blood pressure, joint steadiness, and perceived exertions).
Role: PI
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: Menet, Jerome S

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

POSITION TITLE: Assistant Professor of Biology

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.)

<table>
<thead>
<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
<th>Completion Date MM/YYYY</th>
<th>FIELD OF STUDY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Science &amp; Technology University, Lille, France</td>
<td>B.S.</td>
<td>06/1998</td>
<td>Cell Biology &amp; Physiology</td>
</tr>
<tr>
<td>Louis Pasteur University, Strasbourg, France</td>
<td>Master</td>
<td>06/1999</td>
<td>Neuroscience</td>
</tr>
<tr>
<td>Louis Pasteur University, Strasbourg, France</td>
<td>Ph.D.</td>
<td>10/2003</td>
<td>Neuroscience</td>
</tr>
<tr>
<td>Brandeis University, MA</td>
<td>Post-doc Research Specialist</td>
<td>12/2003-11/2009</td>
<td>Molecular Biology</td>
</tr>
<tr>
<td>Brandeis University, MA and Howard Hughes Medical Institute</td>
<td></td>
<td>12/2009-08/2013</td>
<td>Molecular Biology</td>
</tr>
</tbody>
</table>

A. Personal Statement

The goal of the proposed research is to characterize the mechanisms by which the two major circadian transcription factors, CLOCK and BMAL1, regulate the rhythmic expression of their target genes to enable biological functions to perform optimally at the most appropriate time of the day. Our proposed experiments capitalize on our recent findings and will test the hypothesis that CLOCK:BMAL1 cooperates with transcription factors to 1) bind DNA in a tissue-specific manner and 2) regulate the rhythmic expression of its target genes.

I have the motivation, expertise and leadership necessary to successfully carry out the proposed work. I have worked in the field of circadian biology for 18 years, and, as a postdoctoral fellow at Brandeis University, I more particularly investigated the molecular basis of circadian rhythms in insects and mammals. Over the last 6 years, I initiated independent projects (first as a HHMI Research Specialist, currently as an Assistant Professor) to determine the role of the molecular clock in the generation of circadian rhythms at the genome-wide level in the mouse. I set up a wide-range of molecular and biochemical techniques that include next-generation sequencing (RNA-Seq, ChIP-Seq, Nascent-Seq and MNase-Seq) and produced several high-profile peer-reviewed publications. I recently started my own lab in the Department of Biology and the Center for Biological Clock Research at Texas A&M University to continue my work on the molecular basis of circadian rhythms in the mouse. In particular, we are expanding our recent findings indicating that the molecular clock transcriptional output is heterogeneous and that it promotes rhythmic decondensation of the chromatin (Menet et al., eLife, 2012; Menet et al., Genes Dev., 2014), two findings that laid the groundwork for the proposed research. I have trained three graduate students, Alexandra Trott, Joshua Beytebiere and Ben Greenwell, who will work on this specific project during their Ph.D. thesis. In particular, Alexandra Trott has characterized CLOCK:BMAL1 transcriptional output heterogeneity, and Joshua Beytebiere investigated the tissue-specificity of CLOCK:BMAL1 DNA binding. We are currently writing the manuscripts for both projects, and they should be submitted by the end of Fall 2016.

In summary, I have demonstrated a record of productive research in the field of molecular circadian rhythms, and I will capitalize on my expertise to successfully carry out the proposed project.
B. Positions and Honors

Positions and Employment

1998-1999  Master student, Louis Pasteur University, Strasbourg, France
1999-2003  Ph.D. student, Louis Pasteur University, Strasbourg, France
2002-2003  Lecturer, Louis Pasteur University, Strasbourg, France
2003-2009  Post-doctoral Fellow, Michael Rosbash laboratory, Brandeis University, Waltham, MA
2009-2013  HHMI Research Specialist, Michael Rosbash laboratory, Brandeis University, Waltham, MA
2013-   Assistant Professor, Department of Biology, Texas A&M University, TX

Other Experience and Professional Memberships

2008- Member of the Society for Research on Biological Rhythms
2013- Member of the Center for Biological Clocks Research, Texas A&M University, TX
2013- Faculty of Genetics, Texas A&M University, TX
2014-  Faculty of Neuroscience, Texas A&M Institute for Neuroscience, Texas A&M University, TX

Grant Review

2014- Ad hoc reviewer, National Science Foundation, Division of Molecular and Cellular Biosciences
2015 NIH, NDPR (Neurodifferentiation, Plasticity, and Regeneration) Study Section Review Panel, June roster
2016 Modulation Panel, Division of Molecular and Cellular Biosciences, National Science Foundation

Journal Review


Honors and Awards

1999-2002  Ph.D. fellowship from the French Ministry of National Education and Research
2004-2005  Long-term EMBO post-doctoral fellowship
2008 Young Investigator Award, French-speaking Society of Chronobiology
2009 Talk selected for the Hot Topic symposium of the 11th congress of the EBRS
2011 Hot Topic presentation at the Chronobiology Gordon Research Conference
2013 Chair of the Chronobiology Gordon Research Seminar
2013 Hot topic presentation at the Chronobiology Gordon Research Conference

C. Contribution to Science

1. My early publications come from my Ph.D., during which I directly addressed the role of the master circadian clock located in the suprachiasmatic nucleus (SCN) of the hypothalamus in the encoding of the photoperiodic information (duration of the light phase per 24 hours) that is critical for animals to survive seasonal environmental change. I showed that the SCN encodes the photoperiodic information, and that the underlying mechanisms involve the molecular clockwork and its transcriptional feedback loops, as well as some specific subdivisions within the SCN. I also demonstrated that another structure of the circadian system, the intergeniculate leaflets of the thalamus, modulates this integration of the photoperiod by the SCN. Altogether, these publications contributed to demonstrate that the circadian system not only encodes the daily environmental variation to drive 24-hr rhythms, but also integrates the photoperiodic information. Importantly, these data indicate that the functioning of the circadian system is regulated by the photoperiod and that the circadian clock rhythmic outputs from the SCN are likely altered by the seasons.

I performed the experiments in the studies for which I am the first author and performed some experiments in the study for which I am the second author. As for all my first author papers, I wrote the manuscripts, which were then edited by all co-authors.


2. In the early stage of my postdoc, I worked on projects that investigated the dogma (as it stood in 2008) that post-translational modifications of the negative components of the circadian clock (i.e., the circadian repressors) were solely responsible for circadian repression and determination of the circadian period. We provided evidence for novel mechanisms using the *Drosophila* system. First, we showed that the transcriptional activity of the circadian positive transcription factors CLOCK and CYCLE also controls the pace of the clock and is vital for proper period determination (Kadener et al., 2008). Then, we demonstrated that circadian repression does not solely rely on post-transcriptional events such as phosphorylation and inactivation of the transcriptional activators, but rather involves direct repression of CLOCK:CYCLE by PERIOD while still bound to DNA (on-DNA) and is followed by a stoichiometric sequestering of CLOCK:CYCLE by PERIOD (off-DNA) (Menet et al., 2010). This on-DNA repression mechanism, which we were the first to characterize, has now been found in circadian systems of other organisms.

I contributed to the design of both projects, and performed many of the experiments and data analysis (especially for the paper for which I am the first author).


3. The molecular circadian clock drives the rhythmic expression of hundreds of genes to regulate biological functions under clock control. A major aspect of my research aimed at characterizing the genes targeted by the molecular clock in both *Drosophila* and the mouse (see also contributions 4 and 5). We were the first to characterize CLOCK:CYCLE (the master transcriptional regulator of the *Drosophila* circadian clock) direct target genes at the genome-wide level and address circadian transcriptional regulation. We found that CLOCK:CYCLE rhythmically binds to at least 800 sites with maximal binding in the early night. This binding is followed by the recruitment of the circadian repressor PERIOD 4 to 6 hours later, indicating that the majority of CLOCK:CYCLE targets are regulated similarly to core circadian genes. Importantly, we also showed that CLOCK has specific targets in different tissues. This data provided support to a novel concept for the regulation of circadian transcription: important CLOCK partner proteins and/or mechanisms contribute to gene-specific and tissue-specific regulation. This concept was validated three years later in paper from the Stark group, for which I wrote a dispatch in Current Biology with Dr. Paul Hardin.

I contributed to the design, the experiments and the analysis of the data presented in the Abruzzi paper.


4. Rhythmic mRNA expression is a hallmark of circadian rhythms and is commonly assumed to reflect rhythmic transcription. We addressed this assumption in two organisms (*Drosophila* and mouse) by directly measuring the circadian dynamics of transcription using Nascent-Seq (genome-wide sequencing of nascent RNA). We surprisingly found that many rhythmically expressed transcripts manifest poor transcriptional rhythms. Our data thus indicate an unexpectedly prominent contribution of post-transcriptional regulation to circadian mRNA expression, and reveal a more pervasive role of the molecular circadian clock in driving rhythmic gene expression. Moreover, the analysis of rhythmic transcription in the mouse revealed that the rhythmic DNA binding profile of the transcription factors CLOCK and BMAL1 does not determine the
transcriptional phase of most target genes. This result indicates that, in the mouse, the recruitment of CLOCK:BMAL1 to its target genes’ promoter is not sufficient to activate transcription, and likely reflects gene-specific collaborations of CLOCK:BMAL1 with other transcription factors.

I significantly contributed to the design and analysis of both projects, carried out all experiments performed in the mouse, and wrote the paper published in eLife.


5. CLOCK:BMAL1 sits are the top hierarchy of the mammalian circadian clock, and is thought to activate rhythmic gene expression upon binding to its target genes’ promoter. In this project, we showed that the rhythmic DNA binding of CLOCK:BMAL1 promotes the rhythmic removal of nucleosomes at its DNA binding sites. The mechanisms include CLOCK:BMAL1 binding to nucleosomes and rhythmic chromatin modifications such as the incorporation of H2A.Z. Strikingly, this rhythmic chromatin remodeling mediates the rhythmic binding of other transcription factors adjacent to CLOCK:BMAL1, suggesting that the activity of these other transcription factors contributes to the genome-wide CLOCK:BMAL1 heterogeneous transcriptional output. These data therefore indicate that clock regulation of transcription mainly relies on the rhythmic regulation of chromatin accessibility (and not just activation of transcription upon DNA binding) and suggest that the concept of pioneer function extends to acute gene regulation.

I led this project and performed most of the experiments, all the data analysis and wrote the manuscript.


Complete List of Published Work in MyBibliography (25 papers total):
http://www.ncbi.nlm.nih.gov/sites/MyCBI/1bsUyWj-
yJ_k_/bibliograpahy/41694429/public/?sort=date&direction=ascending

D. Research Support

2014: Texas A&M Center for Biological Clock Research Seed Fund
“Role of the daily food intake rhythm in the synchronization of circadian clocks”
PI: Jerome Menet; co-PIs: David Earnest, Chaodong Wu
Award period: January 2014-December 2015. Total direct costs: $14,000.

2015: Texas A&M University and CONACYT Collaborative Research Grant Program
“Molecular characterization of the adverse effects of shift work on metabolic and cardiovascular functions”
PI: Jerome Menet; co-PIs: Ruud Buijs
Award period: September 2015-August 2016. Total direct costs: $24,000.
NAME: Merlin, Christine

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

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.)

<table>
<thead>
<tr>
<th>INSTITUTION AND LOCATION</th>
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<th>FIELD OF STUDY</th>
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<tr>
<td>University Pierre and Marie Curie Paris 6, France</td>
<td>B.S</td>
<td>05/2001</td>
<td>Biology</td>
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<td>University Pierre and Marie Curie Paris 6, France</td>
<td>M.S</td>
<td>05/2003</td>
<td>Invertebrate physiology</td>
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<tr>
<td>University Pierre and Marie Curie Paris 6, France</td>
<td>Ph.D.</td>
<td>12/2006</td>
<td>Insect physiology</td>
</tr>
<tr>
<td>University of Massachusetts Medical School, USA</td>
<td>Postdoctoral</td>
<td>07/2013</td>
<td>Neurobiology</td>
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</table>

A. Personal statement

I am a circadian biologist with 10 years of research experience and I recently started my own independent laboratory in the Department of Biology and the Center for Biological Clock Research at Texas A&M. The main focus of my research is to understand the genetic basis of complex behaviors with an emphasis on how organisms perceive and respond to external stimuli and how biological clocks control these behaviors. In my laboratory, we use the migratory monarch butterfly as a model system to investigate the molecular mechanisms underlying animal seasonal adaptation and migration. To gain insights into the role of circadian clocks in these processes, we use integrative approaches from genes to behavior. We have recently started developing nucleases-mediated gene targeting approaches to manipulate its genome, which has been sequenced and annotated, and we are very keen in using next-generation sequencing to identify clock-controlled genes, among which some are likely to be candidate migratory genes.

B. Positions and Honors

Positions and Employment

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<td>2007-2013</td>
<td>Postdoctoral Fellow, Department of Neurobiology, UMass Medical School, MA</td>
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<tr>
<td>2013</td>
<td>Assistant Professor, Department of Biology, Texas A&amp;M University, TX</td>
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<td>2013</td>
<td>Faculty, Center for Biological Clocks Research, Texas A&amp;M University, TX</td>
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<tr>
<td>2014</td>
<td>Faculty, Neuroscience Institute, Texas A&amp;M University, TX</td>
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<tr>
<td>2014</td>
<td>Faculty, Genetics Interdisciplinary Program, Texas A&amp;M University, TX</td>
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Other Experience and Professional Memberships

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<tr>
<td>2009-2013</td>
<td>Associate member, Faculty of 1000 Biology</td>
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<tr>
<td>2013</td>
<td>Review editor, Frontiers in Ecology and Evolutionary Biology, Chemical Ecology</td>
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<tr>
<td>2014</td>
<td>Member, Society for Research on Biological Rhythms</td>
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<tr>
<td>2014</td>
<td>Member, NSF Insect Genetic Technology Network</td>
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<td>2016</td>
<td>Member, Genetics Society of America</td>
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Honors and Awards
C. Contributions to Science

1. Pioneered the development of genomic and genetic tools in the monarch butterfly. The discovery that North American migratory monarch butterflies use mammalian-like clock components to help them navigate to their overwintering sites in Mexico established monarchs as a powerful model system for understanding timekeeping mechanisms in higher organisms and how circadian clocks regulate physiology and behavior. Yet the lack of functional genomic tools limited our ability to link genes to unique aspects of monarch physiology and behavior, both in the context of circadian rhythms and seasonal migration. To overcome this limitation, a high quality draft genome sequence and genome editing tools were developed for loss-of-function studies in vivo. During my postdoctoral studies at UMASS Medical School, I was part of a four-member team that sequenced and annotated the monarch genome. I contributed to generating all the material to be sequenced, annotating genes encoding chemosensory receptors and interpreting all data. In parallel, I pioneered the use of Zinc Finger nucleases for genome targeted mutagenesis in vivo, focusing on the mammalian-like cryptochrome (Cry2) as a proof of concept. This work produced the first monarch clock gene knockout, and genetically defined CRY2 as the major transcriptional repressor in the monarch molecular clock transcriptional feedback loop. In my own lab, we have now developed TALENs and CRISPR/Cas9 for generating monarch gene knockouts with substantially greater targeting efficiencies. Together, this work has established strategies for the routine use of reverse-genetics in the monarch, and will allow us to use it as a viable model system to understand circadian clockwork mechanisms relevant to mammals. It also provides a framework for genetic analyses in other lepidopterans and “non-model” insects that could eventually lead to chronobiological control of pests to food crop and disease vector insects that have human hosts.


2. Demonstrated that peripheral clocks in insect antennae drive olfactory rhythms and orientation behaviors. Circadian clocks synchronize animal physiology and behavior to the 24-hour light:dark cycle. However, the mechanisms through which circadian clocks control insect physiology and behavior are not well understood. My early work as a graduate student focused on understanding how circadian clocks in the olfactory sensing organ, the antenna, control the rhythmic receptivity of males to female sex pheromones in moths. I discovered that antennae possess circadian clocks and that physiological responses in antennae to sex pheromones are under clock control, likely by clocks in antennae rather than in the brain. As a postdoctoral fellow at UMASS Medical School, I extended my work on antennal clocks to a complex behavior that occurs on an annual basis: monarch butterfly migration. Monarch butterflies use a sun compass to navigate to their overwintering sites in Mexico, and circadian clocks compensate for the positional change in the sun as it crosses the sky during the day. The clocks responsible for this time-compensation were assumed to be located in the brain. I discovered that the antennae are in fact necessary for proper orientation, and possess autonomous circadian clocks that provide the primary timing component of the sun compass orientation mechanism. This work demonstrated for the first time that peripheral clocks could regulate brain function and complex behavior. Temporal regulation of proper brain function by peripheral clocks could be more pervasive than anticipated in animals, and our finding could thus have important implications to understand the impact of peripheral clocks disruption on behavioral disorders.


**Complete List of Published Work in NCBI My Bibliography (19 papers total):**

D. Research Support

**Ongoing**

NSF IOS1456985 Merlin (PI) 05/01/2015-04/30/2018

Circadian clock control of seasonal migration
The goal of this study is to identify how the circadian clock controls seasonal changes in physiology and behavior using the migratory monarch butterfly as a model.
Role: PI.
NAME: Rajesh C. Miranda, PhD

POSITION TITLE: Professor

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

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.)

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<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
<th>Completion Date MM/YYYY</th>
<th>FIELD OF STUDY</th>
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<tr>
<td>St. Xavier's College, Bombay, India</td>
<td>B.A.</td>
<td>1982</td>
<td>Psychology</td>
</tr>
<tr>
<td>Bombay University, Bombay, India</td>
<td>M.A.</td>
<td>1984</td>
<td>Clinical Psychology</td>
</tr>
<tr>
<td>University of Rochester, Rochester, N.Y.</td>
<td>M.A.</td>
<td>1987</td>
<td>Biopsychology</td>
</tr>
<tr>
<td>University of Rochester, Rochester, N.Y.</td>
<td>M.S.</td>
<td>1988</td>
<td>Neurobiology</td>
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<tr>
<td>University of Rochester, Rochester, N.Y.</td>
<td>Ph.D.</td>
<td>1989</td>
<td>Biopsychology/Neurobiology</td>
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</table>

A. Personal Statement:

My own research has focused on maternal-fetal health, and on the impact of maternal alcohol exposure on fetal brain development. We focus primarily on fetal neural stem cells as a unique target of developmental vulnerability and on the biology of a class of regulatory small RNA molecules, called microRNAs. In 2007, we were the first research group in the fields of teratology, toxicology and addiction biology to identify microRNAs as mediators of ethanol’s teratogenic effects (PMCID: PMC2915840). Since then, we have identified and assessed the functions of teratogen-sensitive miRNAs associated with fetal neural stem cell maturation, as well as miRNAs associated with neural adaptation to degeneration.

Pertinent to the current proposal, Dr. Sohrabji and I have a long-term and ongoing collaboration, including significant collaboration on my NIH-funded project (R01AA024659), and 14 co-authored peer-reviewed publications, including publications on miRNAs (PMC4386587, PMC3290559) and epigenetics (PMC4622874) in stroke outcomes. In this proposal, I will contribute to the design of experiments on miRNA biology including cellular analyses of miRNA function. Relevant to the current proposal, my laboratory has expertise with studies of plasma miRNAs in human disease, an important component of the current proposal with publications (PMC5102408) and current grant support (R21AA024055 and U01 AA014835 (subcontract)) in this area.


B. Positions and Honors

Positions and Employment
1983-1984  St. Xavier's College, Bombay India, University Grants Commission teaching assistant
1983  University of Rochester, Depts. of Psychology and Neuroscience: Teaching assistant.
1990-1994  Columbia University College of Physicians and Surgeons, Instructor, Medical Neuroanatomy
1995-2000  Texas A&M University, Dept. Human Anatomy and Medical Neurobiology, Assistant Professor.
2000-2009  Texas A&M, Health Science Center, Dept. Neurosci & Expt. Therapeutics, Associate Professor
2009-present  Texas A&M Health Science Center, Professor
2005-present  Texas A&M University, Department of Psychology, Adjunct Professor

Service and Memberships
1995-present  Member of the Faculties of Neuroscience, Reproductive Biology and Toxicology at TAMU
1999-present  Member, Center for Environmental and Rural Health, Texas A&M University
2002-2003  Ad-hoc reviewer, NIH, ALTX-3 and NAL study section
2004-2007  Member, NIH, NAL (Neurotoxicology and Alcohol) study section
2006-2009  Ad. Hoc. Member, NIH AA-1, ZAA1-BB98, NCF, MNG & AA-4 study sections
2009-2012  Member, NIH AA-4 study section.
2012-2015  Chair, AA-4 study section
2009-2011  Treasurer, vice president Fetal Alcohol Spectrum Disorders Study Group (FASDSG)
2011-2012  President, Fetal Alcohol Spectrum Disorders Study Group
2012-present  Member of the Steering Committee on FASD prevention at the Texas HHS Office for Prevention of Developmental Disabilities (TOPDD)
2013-present  Co-chair of the FASD surveillance and epidemiology workgroup, TOPDD.

C. Contributions to Science (underlined authors are co-investigators on the current proposal)

1. MiRNAs as mediators of ethanol effects. My laboratory was the first to identify miRNAs as mediatory factors in the fields of toxicology, teratology and drug addiction. Ethanol is often viewed as a ‘dirty drug’ that alters the expression of many genes and developmental processes. Since miRNAs control the translation of networks of genes, evidence for miRNA involvement in teratology has the potential for promoting the development of unifying theories for teratogenesis. Moreover, nicotinic acetylcholine receptors, GABAa receptors and epigenetic factors also control the expression of ethanol-sensitive miRNAs. These data may ultimately facilitate the development of therapeutic protocols to reverse teratogenesis.


2. MiRNAs for diagnosis and therapy. MiRNAs are intracellular regulators of networks of protein coding genes. However, recent evidence shows that they are also secreted into biofluids and constitute an endocrine signal. We have shown for the first time that secreted miRNAs may be used for diagnosis of fetal alcohol exposure and other diseases and for therapy.

3. Fetal Neural stem cells (NSCs) are vulnerable to ethanol exposure. We initiated experiments in this area to test the hypothesis that ethanol induced apoptosis and other death mechanisms in fetal NSCs. To our surprise, we found that NSCs are depleted not because of cell death, but because ethanol promotes aberrant maturation. These data are important for the perspective advanced in this current proposal, that NSCs can be re-programmed to increase neurogenic capacity following episodes of ethanol exposure.


4. Cell death mechanisms control early neural development. We were one of the early groups to show that receptor-mediated apoptosis was common during early neural development. We also showed that gonadal hormones and teratogens could control cell death pathways.


5. Neurotrophic factor autocrine loops in the developing brain as mediators of developmental actions of estrogen. As a post-doctoral fellow in the laboratory of Dr. Toran-Allerand, Columbia University, we were the first group to provide evidence for the existence of neurotrophin autocrine signaling pathways in the developing nervous system and showed that gonadal hormones could control neurotrophin signaling pathways.


b. Sohrabji F, Miranda RC, Toran-Allerand CD (1994) Estrogen differentially regulates estrogen and
D. Research Support
Ongoing Research Support
1R01NS074895 (Sohrabji, PI) 9/01/2011-05/30/2017 (nce)
Neuroprotection in the Aging Female Brain
I serve as a co-investigator on this project. The overall goal of this application is to determine the interaction of estrogen and IGF-1 in the context of stroke and neuroprotection in middle age females, using an animal model. No overlap with present proposal.

1R01ES020276 (Sohrabji, PI) 9/15/11-5/31/17 (nce)
Epigenetics of the Aging Astrocyte: Implications for Stroke
I serve as a co-investigator on this project. Major goals: The overall goal of this application is to identify aging- and stroke-related epigenomic changes in astrocytes (in response to RFA ES 10-002). No overlap with present proposal.

U01 AA014835 (Chambers, PI; Miranda, sub-contract PI) 06/01/2012-05/31/2017
Early Identification of Affected Children and Risk Factors for FASD in Ukraine
I serve as a sub-contract PI on this proposal. My role is to screen for plasma miRNA biomarkers for alcohol exposure in mothers of an FASD cohort in the Ukraine. The purpose is to identify maternal plasma miRNA biomarkers in early pregnancy, that predict future infant developmental outcomes. This proposal will correlate the expression of miRNA biomarkers with other epigenetic markers of maternal alcohol exposure. OVERLAP: None.

Craig H. Neilsen Foundation (Grau, PI) 11/01/2014-10/31/2017
How and when does peripheral input affect recovery after SCI?
I serve as co-investigator on this proposal. The experiments focus on the role of pain and inflammatory signals on pyroptosis in the spinal cord and recovery of function. No overlap with present proposal.

1R21AA024055-01 (S Jacobson, RC Miranda, multi-PI) 06/01/2015-05/31/2017
MicroRNAs As Biomarkers Of Exposure And Effect In Fetal Alcohol Spectrum Disorders
I serve as co-PI on this proposal. The focus is on identifying plasma microRNA biomarkers in infants that predict neurocognitive deficits. No overlap with present proposal.

R01HD086765 (K Larin, RC Miranda, Multi-PI) 01/01/2016-12/31/2020
Optical Coherence Tomography to Study Effect of Poly-Drug Exposure on Fetal Brain Development
I serve as multi-PI on this award. The purpose is to develop a whole animal quantitative microscopy method to detect structural and dynamic in vivo effects of alcohol and nicotine exposure on fetal blood flow and brain development. No overlap with present proposal.

1R01AA024659-01 (Miranda, PI) 03/10/2016-02/28/2021
Title: Prenatal microRNA neuro-therapeutics for fetal alcohol exposure
Role: PI. This proposal focuses on identifying molecular biological principles (miRNA-transcription factor networks) and pharmacological approaches (nicotinic acetylcholinergic) to reprogram neural stem cells to overcome deficits in brain growth due to early (1st trimester-equivalent) fetal alcohol exposure. The goal of this project is to find ways to stimulate the growth of fetal neural stem cells that have been previously exposed to alcohol, as a means to minimize the damaging effects of fetal alcohol exposure.
Title: Effect of inflammation on recovery and pain after spinal cord injury
Role: co-I. Goals: Prior work has shown that engaging pain (C) fibers soon after spinal cord injury undermines behavioral recovery and fosters the development of neuropathic pain. NS091723 grant will use a peripheral irritant (capsaicin) to engage pain fibers. We will determine when pain input affects recovery, whether it leads to tissue loss by inducing apoptosis or pyroptosis, and whether blocking pyroptosis attenuates its effect on recovery.

Completed Research Support
RO1 AA13440 (Miranda, PI) 03/01/2002-08/31/2015
Fetal Alcohol exposure and neurodevelopment
I served as PI for this project. AA13440 investigates (1) the role of alcohol exposure on the control of receptor-neural stem cell maturation. (2) The involvement of miRNAs as critical mediators of ethanol’s effects on stem cell maturation.

U24 AA014811 (Riley, PI; Miranda, sub-contract PI) 8/1/2009-5/31/2015
NIAAA/CIFASD Consortium Developmental Project
Circulating microRNA biomarkers of Fetal Alcohol Exposure
I served as a sub-contract PI on this proposal. The proposed experiments focused on identifying novel miRNA biomarkers of maternal and fetal alcohol exposure and studying the functions of secreted miRNAs.
A. Personal Statement

In this project, we will screen melanocortin-5 receptor (MC5R) blockers for target activity in vitro, as well as behavioral efficacy and oral bioavailability in mice. This will build on my previous work. My predoctoral work partly focused on influences of melanocortins on hypothalamic-pituitary-adrenal activity and emotional behavior in hamsters. As a postdoctoral scientist, I focused on the role of MC5R in emotional behavior in hamsters and mice. As an independent scientist, I discovered that MC5R controls depression-related status in mice, developed animal models of depression and anxiety, as well as assays for rodent depression- and anxiety-related behaviors. The scientific literature on non-selective melanocortin blockers, beginning in 1974, shows that they outperformed antidepressants for therapeutic efficacy, onset of efficacy, and frequency of side effects in humans and laboratory animals. They also had potential for low price points. Unfortunately, this promising work was limited by the technology (e.g., a need for intramuscular or intravenous delivery), intellectual property (e.g., a single expiring patent), and business model (e.g., only one candidate in the pipeline). Nonetheless, early-generation melanocortin blockers clearly outperformed antidepressants in clinical trials. I acquired selective MC5R blockers, while at Texas A&M University in 2014, and determined that they possess at least three characteristics of their non-selective predecessors (i.e., high efficacy, rapid action, and potentially low price points). I founded Akhu Therapeutics in 2015, and we acquired an exclusive worldwide license from the University of Arizona in 2016 to develop treatments for mood and other CNS disorders from an extensive series of peptide MC5R blockers. These peptides possess four important advantages over their predecessors. They have pharmacologic selectivity, stability in biological systems, oral bioavailability, and protection by nine patent applications, and I am a co-inventor. My experience, including the management of research teams with 5 to 15 personnel for 15 years makes me highly motivated and prepared to lead this project.


B. Positions and Honors
Positions and Employment

2001-04   Assistant Professor, Psychiatry, Weill Medical College of Cornell University, New York, NY
2005-06   Adjunct Assistant Professor, Biology, California State University, San Marcos, CA
2007-15   Assistant Professor of Bioenergetics, Texas A&M University, College Station, TX
2015-     CEO and President, Akhu Therapeutics, Inc., College Station, Texas

Other Experience

1991      NIDDK Travel Award, American Psychological Society Meeting, Orlando, FL
1994      Eli Lilly Travel Award, Society Neuroscience Meeting, New Orleans, LA
1999      Endocrine Society Travel Award, Endocrine Society Meeting, San Diego, CA
1999      NIH/NIMH Travel Award, Am Coll Neuropsychopharmacology
1999      Invitee, Am Coll Neuropsychopharmacology Meeting, Acapulco, MX
2000      Invitee, Am Coll Neuropsychopharmacology Meeting, San Juan, PR
2001      Invitee, Am Coll Neuropsychopharmacology Meeting, Waikoloa, HI
2002      Invitee, Am Coll Neuropsychopharmacology Meeting, San Juan, PR
2003      Invitee, Am Coll Neuropsychopharmacology Meeting (declined)
2007-     Graduate Faculty, Intercollegiate Faculty of Nutrition, Texas A&M University
2008-     Affiliate Member, Institute for Obesity Research, Texas A&M University
2009-     Graduate Faculty, Institute for Neuroscience, Texas A&M University
2010-12   Fellow, Mexican-American and Latino Research Center, Texas A&M University
2012      Invitee, Capitol Hill Day, Society for Neuroscience Gov & Public Affairs Cmte, Wash, DC
2012      Early Career Reviewer Program, NIH, Neuroendocrinology, Neuroimmunology, Rhythms, and Sleep (NNRS) Study Section
2013      Early Career Reviewer Program, NIH, Neuroendocrinology, Neuroimmunology, Rhythms, and Sleep (NNRS) Study Section
2015      Invitee, Capitol Hill Day, Society for Neuroscience Gov & Public Affairs Cmte, Wash, DC

Professional Memberships
American Physiological Society
Endocrine Society
New York Academy of Science
Sigma Xi
Society for Neuroscience
Organization for the Study of Sex Differences
Texas Academy of Science

C. Contributions to Science

1. I began to develop expertise that is relevant to this project under the mentorship of Huda Akil at the University of Michigan. As a doctoral student, funded by a King/Chavez/Parks Fellowship, I learned to clone stress-related genes and assess their expression.
2. I later conducted NIH-funded postdoctoral research under the mentorship of Catherine Rivier and Wylie Vale (Salk Institute). In that position, I studied neuroendocrine mechanisms of stress using molecular, antisense, and neuroanatomic approaches (e.g., ICV cannulations and in situ hybridization). I continued NIH-funded postdoctoral training under the mentorship of Roger Cone at Oregon Health and Science University where I investigated feeding, social, and emotional behaviors, metabolism & physiology, and MC5R control of emotional behaviors in mice.

3. As an independent investigator, I have developed rodent models of anorexia, anxiety, and depression. I have also developed assays for emotional behaviors in rodents, and assessed neural and endocrine signaling and expression that is relevant to emotional behavior and physiology.

D. Research Support

Current Research Support

Bootstrapping  
Founder: Business Development of Akhu Therapeutics  
Project goal: Cover startup, incorporation, and in-licensing costs.  
Role: CEO  
Funding Start Date: 06/01/15

Indiegogo Reward-Based Crowdfunding  
Campaign: Early Business Development  
Project goal: Cover costs of relocating startup to California  
Role: CEO  
Campaign Start Date: 06/15/16
Pending Research Support

NIH/National Institute of Mental Health
SBIR Phase I: Development of Melanocortin-5 Receptor Blockers for Depression
Project goal: Select MC5R blocker for preclinical efficacy and oral bioavailability
Role: CEO
Funding Start Date: 12/15/17

EquityNet Equity-Based Crowdfunding
Campaign: Early Drug Development
Project goal: Initiate preclinical toxicology study
Role: CEO
Campaign Start Date: 09/15/17

Research Support Completed During the Last Three Years

Texas AgriLife Research and National Institute of Food and Agriculture, USDA (H-9230)
Signature Research Program Award: Nutritional Interventions in Metabolic & Emotional Disorders
Project goal: Study Neurobiological and physiological responses to nutrients.
Role: PI
Funding Dates: 01/01/07 to 05/31/15

C.O. and Willie Ruth Foerster Undergraduate Research Fellowship Program
Award: Nutrition and Neuroscience Interactions
Project goal: Engage undergraduates in research projects.
Role: PI
Funding Dates: 04/01/11 to 04/30/15
Justin Moscarello, PhD  
Assistant Professor  
Department of Psychology  
Institute for Neuroscience  
Texas A&M University  
jmm31@tamu.edu

**Education & Training**

**Degrees**

<table>
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<th>Year</th>
<th>Degree</th>
<th>Institution</th>
<th>Dissertation Title</th>
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<tr>
<td>2010</td>
<td>PhD Psychology</td>
<td>University of California, Santa Barbara</td>
<td>The role of the medial prefrontal cortex and nucleus accumbens in motivation and reinforcement</td>
</tr>
<tr>
<td>2003</td>
<td>BA Physical Anthropology</td>
<td>UCSB</td>
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**Academic Honors**

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<tr>
<td>2009</td>
<td>Harry J. Carlisle Award for Outstanding Graduate Student</td>
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<tr>
<td>2006</td>
<td>Advanced to PhD candidate with distinction</td>
</tr>
<tr>
<td>2003</td>
<td>Graduated magna cum laude and with distinction in major</td>
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<tr>
<td>2002-03</td>
<td>Dean’s Honor List</td>
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**Positions & Employment**

**Faculty**

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<th>Year</th>
<th>Position</th>
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<th>Details</th>
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<tr>
<td>2017</td>
<td>Assistant Professor</td>
<td>Department of Psychology</td>
<td>Institute for Neuroscience, Texas A&amp;M University</td>
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**Research**

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<th>Details</th>
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<tr>
<td>2014-16</td>
<td>Senior Research Scientist</td>
<td>Center for Neural Science, New York University</td>
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<tr>
<td>2010-14</td>
<td>Postdoctoral Fellow</td>
<td>Mentor: Professor Joseph LeDoux, Center for Neural Science, NYU</td>
</tr>
<tr>
<td>2004-10</td>
<td>Graduate Student Researcher</td>
<td>Mentor: Professor Aaron Ettenberg, Department of Psychological &amp; Brain Sciences, UCSB</td>
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<tr>
<td>2003-04</td>
<td>Laboratory Technician</td>
<td>Ettenberg Lab, Department of Psychological &amp; Brain Sciences, UCSB</td>
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Teaching & Mentorship

2010-16  **Undergraduate Mentor**  
Center for Neural Science, NYU  
Trained undergrad researchers in lab procedures.  
Mentored undergrad researchers writing honors theses.

2008-09  **Lecturer/Instructor of Record**  
Course title: Psychopharmacology of Drugs of Abuse  
Department of Psychological & Brain Sciences, UCSB  
Developed syllabus and all course materials, delivered all lectures.

2004-09  **Laboratory Instructor**  
Department of Psychological & Brain Sciences, UCSB  
Courses include Neural Development, Neuropharmacology, Introduction to Biopsychology, Motivation, Cognition, Psychopathology.  
Graded papers and exams, delivered guest lectures, lead lab exercises.

2004-09  **Teaching Assistant**  
Department of Psychological & Brain Sciences, UCSB  
Courses include Neuroanatomy, Neuroendocrinology, Methods in Biopsychology, Animal Learning.  
Lead lab exercises, graded papers and exams, delivered guest lectures.

Institutional Service

2006-09  **Graduate Student Member of IACUC**  
UCSB

Research Funding

2017-19  **NARSAD Young Investigator Award**  
Brain & Behavior Foundation  
Title: Neural Mechanisms of Resilience  
Total Award: $70,000

2011-14  **Postdoctoral National Research Service Award (NRSA)**  
National Institute of Mental Health (#F32 – MH094061)  
Title: The role of medial prefrontal cortex in active avoidance behavior  
Total award: $155,466

2008-09  **Predoctoral National Research Service Award (NRSA)**  
National Institute on Drug Abuse (#F31 – DA024505)  
Title: Dopamine terminal regions interact as a function of motivation & reinforcement  
Total award: $63,399

2007  **Dean’s Fellowship**  
College of Letters & Sciences, UCSB  
Total award: $15,000
Publications

Empirical Papers in Preparation

Moscarello JM, Diaz-Mataix L, LeDoux JE. Active avoidance recruits a prefrontal cortex-nucleus reuniens pathway to suppress Pavlovian reactions.

Moscarello JM, LeDoux JE. The associative structure of active avoidance memory.

Published Empirical Papers


*denotes shared 1st authorship


**Reviews in Preparation**


**Published Reviews and Book Chapters**


*denotes shared 1st authorship


**Invited Talks**

2017 Department of Neuroscience and Experimental Therapeutics, Texas A&M University. Title: Neural pathways of active avoidance behavior.

2017 Winter Conference on Neural Plasticity, Grenada. Title: Avoidance learning recruits a PFC-nucleus reuniens pathway to suppress conditioned freezing

2016 Department of Psychology, NYU. Title: The associative structure of active avoidance memory in rat.

2016 Pavlovian Society Meeting, Jersey City, NJ. Title: Investigating the associative structure of active avoidance memory

2016 Department of Psychology, Texas A&M University. Title: Mastering fear: the neural substrates of signaled active avoidance behavior.

2015 Society for Neuroscience, Washington DC. Title: Active avoidance recruits a prefrontal-hippocampal circuit for the suppression of innate defensive reactions.
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: Naomi Nagaya, PhD

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

POSITION TITLE: Research assistant professor of Psychology

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.)

<table>
<thead>
<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
<th>Completion Date MM/YYYY</th>
<th>FIELD OF STUDY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stanford University, Stanford, CA</td>
<td>B.S.</td>
<td>06/1984</td>
<td>Biological Sciences</td>
</tr>
<tr>
<td>University of Southern California, Los Angeles</td>
<td>Ph.D.</td>
<td>12/1993</td>
<td>Biological Sciences (Neurobiology)</td>
</tr>
<tr>
<td>Geffen School of Medicine, University of California, Los Angeles</td>
<td>Postdoctoral</td>
<td>6/1996</td>
<td>Neuroscience</td>
</tr>
<tr>
<td>University of Michigan Medical School, Ann Arbor</td>
<td>Postdoctoral</td>
<td>6/2001</td>
<td>Neuroscience</td>
</tr>
<tr>
<td>University of Michigan, Ann Arbor</td>
<td>Researcher</td>
<td>6/2011</td>
<td>Neuroscience</td>
</tr>
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A. Personal Statement

I have had a longstanding interest in the role of hormones in regulating behavior. I have a broad background in neuroscience spanning from molecular approaches to the understanding of ion channel biogenesis, structure and function to plasticity in synaptic structure and efficacy but have recently transitioned into the study of animal behavior. My current research interests include the role of sex steroids in the regulation of learned fear and anxiety as well as molecular and cellular mechanisms of sex differences in fear-related behavior. My work utilizes Pavlovian fear conditioning in rodents and involves analysis from behavioral, cellular, and molecular perspectives.


B. Positions and Honors

Positions and Employment
1993-1996 Fellow, Dept. of Physiology, Geffen School of Medicine, University of California, Los Angeles
1996-2001 Fellow, Dept. of Neurology, University of Michigan School of Medicine, Ann Arbor
Texas A&M Institute for Neuroscience
2001-2011 Research Investigator, Dept. of Molecular, Cellular, and Developmental Biology, University of Michigan, Ann Arbor
2007-2012 Lecturer, Depts. of Molecular, Cellular, and Developmental Biology and Psychology, University of Michigan, Ann Arbor
2012- Research Assistant Professor, Dept. of Psychology and Institute for Neuroscience, Texas A&M University, College Station

Honors
1988 Student, NIMH-sponsored summer course on Neural Systems and Behavior, Marine Biological Laboratory, Woods Hole, MA
1989 Travel Fellowship for Minority Neuroscientists, Society for Neuroscience
1991 Women in Neuroscience Travel Award, Society for Neuroscience
1996 Second Prize (Basic Science), Laverna Titus Young Investigators Forum, American Heart Association, Greater Los Angeles Affiliate

Memberships
1988- Society for Neuroscience
1989- American Association for the Advancement of Science
1993-2014 Biophysical Society
2013- Texas A&M University Chapter of the Society for Neuroscience
2013- Pavlovian Society
2014- Organization for the Study of Sex Differences

C. Contributions to Science

1. My graduate work focused on the study of synaptic plasticity at the frog neuromuscular junction with the focus of my dissertation being the hormonal regulation of plasticity in synaptic function in a sexually dimorphic frog muscle. This work supported the idea that synapses in adult vertebrates (amphibians, in this case) retain plasticity in terms of both structure and function and perhaps represent the persistence of mechanisms typically associated with development.

2. My postdoctoral work focused on various aspects of ion channel biogenesis, structure, and function. I characterized the role of a chaperone protein as well as an important step in the biosynthetic pathway of multisubunit voltage-gated potassium channels (supported by the American Heart Association, Greater Los Angeles Affiliate Postdoctoral Research Fellowship). I identified protein domains important for zinc binding in both GABA_A receptors (supported by the Epilepsy Foundation of America/American Epilepsy Society Research Fellowship) and P2X_2 receptors and demonstrated their functional roles in channel pharmacology and subunit interactions.
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: PETER P. NGHIEM

POSITION TITLE: ASSISTANT PROFESSOR (TENURE-TRACK), VETERINARY INTEGRATIVE BIO SCIENCES, TEXAS A&M UNIVERSITY

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.)

<table>
<thead>
<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
<th>Completion Date MM/YYYY</th>
<th>FIELD OF STUDY</th>
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<tbody>
<tr>
<td>Texas A &amp; M University (TAMU)</td>
<td>DVM</td>
<td>05/2008</td>
<td>Medicine &amp; Surgery</td>
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<tr>
<td>The University of Georgia (UGA) Small Animal Teaching Hospital</td>
<td>Internship</td>
<td>07/2009</td>
<td>Medicine &amp; Surgery</td>
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<td>Children’s National Medical Center (CNMC)</td>
<td>Fellowship</td>
<td>08/2010</td>
<td>Vet Neurology</td>
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<td>CNMC</td>
<td>F32 NRSA</td>
<td>08/2013</td>
<td>Molecular Medicine</td>
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<tr>
<td>The George Washington University (GWU)</td>
<td>Ph.D.</td>
<td>01/2014</td>
<td>Molecular Medicine</td>
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<tr>
<td>CNMC</td>
<td>Post-doc</td>
<td>07/2014</td>
<td>Molecular Medicine</td>
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A. Personal Statement

My experience with the canine models for muscular dystrophy dates back to 2009, where I discovered a Cavalier King Charles spaniel with dystrophin deficiency while working as a veterinarian at the University of Georgia. My fascination with muscular dystrophy led to completion of a Ph.D. under Dr. Eric Hoffman (with Dr. Joe Kornegay as a co-mentor), evaluating molecular mechanisms of differential muscle involvement in the golden retriever muscular dystrophy (GRMD) dog, a model for Duchenne muscular dystrophy (DMD). During this time, I was funded with an F32 NRSA mechanism by the National Institute of Arthritis and Musculoskeletal Diseases (NIAMS) and also the NIH Loan Repayment Award. The NIH-funded research has led to several publications on the GRMD dog (see below). Completing a post-doctoral fellowship under Dr. Eric Hoffman and also working as a Senior Scientist at Pfizer allowed me to further sharpen my molecular techniques, creating a career path to becoming a principal investigator. Since October 2015, I have assumed a tenure-track Assistant Professor position in Neuroscience at Texas A&M University with a focus on the canine Duchenne muscular dystrophy models. I am keenly interested in evaluating phenotypic and molecular outcome measures in the canine models for DMD, including muscle force, surgical muscle biopsy, 6-min walk test, among others.


A. Positions and Honors

Positions and Employment
2015-Present  Tenure-track Assistant Professor, TAMU
2014-2015   Senior Scientist, Pfizer Worldwide Research and Development, CT
2013-2014   Post-doc, CNMC, D.C.
2011-2014   Clinical Laboratory Veterinarian (Back-up), GWU, D.C.
2010-2013   F32 NRSA Fellow, NIH-NIAM and CNMC, D.C.
2009-2010   American College of Vet Internal Medicine, Post-doc Neurology Research Fellowship, CNMC
2008-2009   Post-doctoral Clinical Internship, University of Georgia Veterinary School, GA

Other Experience and Professional Memberships
2015   Muscular Dystrophy Association Conference, D.C.
2011-2014 Institute for Animal Care and Use Committee, Active Member, GWU, D.C.
2013   Muscular Dystrophy Association Conference, D.C.
2012   NIH Regional Seminar on Program Funding, NIH, D.C.
2011   NIAMS 25th Anniversary Scientific Symposium, NIH, Bethesda
2011-2012 Licensed Veterinarian in Washington, D.C.
2008-2010, ’15 Member, American Veterinary Medical Association

Honors
2013   Research Poster Winner, GWU, D.C.
2012   Invited Oral Speaker and Poster at FASEB Osteopontin Biology Conference, VT
2011-2013 NIH Pediatric Loan Repayment Program, NIH-NIAMs
2008-2009 Pfizer Specialty Award: Internal Medicine, TAMU, TX
2006-2007 Danny L. Davis Memorial Award, TAMU, TX
2005-2008 General Veterinary Scholarship, TAMU, TX
2005-2006 Gentle Doctor Benefit Scholarship, TAMU, TX
2004-2008 Sandra-Austin Endowed Scholarship, TAMU, TX
2004-2008 Diversity Scholarship, TAMU, TX
2004-2006 Student Body President, TAMU CVM, TX

B. Contribution to Science
1. Characterization of naturally occurring disease models.
   Since the identification and phenotypic characterization of the Cavalier King Charles Spaniel with dystrophin deficiency that I discovered in 2009, I have characterized its DMD gene mutation with next generation sequencing of the whole genome. The affected dog has a mutation in DMD exon 42, which is in a secondary hot spot area of the DMD gene. I am evaluating options to perpetuate this line for research purposes.

   My research work as a young investigator pertains to the identification of genetic modifiers in Duchenne muscular dystrophy. We identified through genome-wide mRNA and microRNA profiling and proteomic (mass spectrometry) profiling several key signatures associated with paradoxical muscle hypertrophy in the GRMD model. These include AKT1, myotrophin, alpha-dystroglycan, spectrin, myostatin and target microRNAs (first author publication, “Sparing of the dystrophin-deficient cranial sartorius muscle is associated with classical and novel hypertrophy pathways in GRMD dogs”). Other modifiers of interest include osteopontin.


3. Canine and Feline Neurology

As a veterinary trainee, I was drawn to the field of neurology and neuromuscular disorders, where I had a keen interest in canine and feline disorders. I assisted in several neurology-based studies, published several review articles, and co-authored several book chapters in the canine/feline neurology field (opportunities given to me by former mentors at TAMU and UGA). As detailed above, I began to focus on neuromuscular disorders, specifically muscular dystrophy, when I discovered a Cavalier King Charles Spaniel with dystrophin deficiency. From here, I have contributed several publications in the field of animal models, precisely the canine models for Duchenne muscular dystrophy, which include the GRMD dog and the initial publication detailing the German short-haired pointer muscular dystrophy dogs.


4. Research Support

Active Research Support
Application of an immortalized canine muscle line and SIAC to muscular dystrophy studies. Solid GT (Gene Therapy). JN Kornegay, Principal Investigator-2% effort; PP Nghiem, Co-Principal Investigator-50% effort; 04/01/17–03/31/2018; $92,484 Total; $77,070 Direct.

Completed Research Support
Pediatric Loan Repayment Award (PI; mentor Eric Hoffman) 11/01/2011-11/01/2013
NIAMS/ Children’s National Medical Center
The goals of this award were to further define the functional and molecular relationships between muscle imbalance and joint contractures in pediatric patients with Duchenne muscular dystrophy.

5F32AR060703-01-03 Post-doctoral Fellowship (PI; mentor Eric Hoffman) 07/01/2010 - 06/30/2013
NIH-NIAMS / Children’s National Medical Center
Joint contractures in golden retriever muscular dystrophy: A model for Duchenne muscular dystrophy. The goals of this F32 were to further define the functional and molecular relationships between muscle imbalance and joint contractures in Duchenne muscular dystrophy.
ACVIM Post-Doctoral Neurology Research Fellowship (PI, mentor S. Schatzberg) 07/01/2009 - 06/30/2010
ACVIM / UGA & CNMC
Molecular pathophysiology of dystrophin-deficient muscles: Golden retriever muscular dystrophy.
The major goal of this fellowship was to provide funding for one year of post-doctoral research to study molecular genetics, bioinformatics, and biochemistry, utilizing a canine model for Duchenne muscular dystrophy.
NAME: Orr, Joseph M  

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

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.)

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<th>DEGREE (if applicable)</th>
<th>Completion Date MM/YYYY</th>
<th>FIELD OF STUDY</th>
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<tr>
<td>University of Michigan, Ann Arbor, MI</td>
<td>MS</td>
<td>08/2008</td>
<td>Cognition &amp; Cognitive Neuroscience</td>
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<tr>
<td>University of Michigan, Ann Arbor, MI</td>
<td>PHD</td>
<td>12/2011</td>
<td>Cognition &amp; Cognitive Neuroscience</td>
</tr>
<tr>
<td>University of Colorado Boulder, Boulder, Colorado</td>
<td>Postdoctoral Fellow</td>
<td>06/2015</td>
<td>Cognitive Neuroscience</td>
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A. Personal Statement

I am a new Assistant Professor at Texas A&M University in the Department of Psychology and the Texas A&M Institute for Neuroscience. I have built a line of research investigating the psychological and neural mechanisms underlying executive function. Specifically, my research seeks to understand the mechanisms underlying voluntary task selection according to abstract goals, and the shielding of these mechanisms from distraction and competing goals. Over the course of my graduate and post-doctoral research training, I have developed the expertise necessary to carry out the proposed research. I will serve as the project director and principal investigator on this proposal in direct collaboration with Co-PI, Dr. Jessica Bernard. We have assembled a research team of co-Investigators and Consultants with critical theoretical and technical expertise to ensure the success of the proposed work. As a doctoral student at the University of Michigan working with Drs. Daniel Weissman and William Gehring, I received training in functional magnetic resonance imaging (fMRI) and Event-Related Potentials, and developed a strong theoretical grounding in cognitive psychology. In my dissertation research I began to develop my current line of research examining the internal and external factors underlying voluntary task selection. I continued to develop this line of research in my NIDA-funded NRSA postdoctoral fellowship at the University of Colorado Boulder working with Dr. Marie Banich. While working with Dr. Banich I gained further experience in fMRI experimental design and analysis, and received training in additional neuroimaging techniques for white matter imaging and resting state connectivity. I further developed expertise in connectivity through a collaboration with Dr. Vijay Mittal working on identifying biomarkers of disease progression in adolescents at risk for psychosis using multi-modal neuroimaging (functional, structural, and diffusion MRI).


B. Positions and Honors

Positions and Employment
2015 - Assistant Professor, TEXAS A&M UNIVERSITY, College Station, TX

Other Experience and Professional Memberships
2004 - 2013 Student Member, Society for Neuroscience
2006 - Member, Cognitive Neuroscience Society
2013 - Member, Society for Research in Psychopathology
2014 - Member, Organization for Human Brain Mapping
2016 - Member, Psychonomic Society

Honors

C. Contribution to Science

1. Internal and external mechanisms underlying voluntary task selection. Cognitive flexibility underlies our ability to rapidly update our goals and behaviors in response to changes in internal and external information. Traditionally, cognitive neuroscience has examined cognitive flexibility through paradigms that involve rapidly alternating between two or more tasks, as instructed by the experimenter. However, it is unclear whether these same mechanisms play a role in situations where task choice is voluntary, or under the control of free-will. Using behavior and brain imaging methods, I have shown that voluntary task selection involves separate brain mechanisms from externally-directed task selection, namely the frontal pole of the brain. Further, my work has shown that participants are better able to ignore distracting external information when they freely choose the task as opposed to the task being instructed. Moreover, the frontal pole is more activated on trials where participants voluntarily choose to go against external information rather than choose to follow external information.


2. Multimodal brain connectivity for the identification of putative biomarkers of psychopathology. During my post-doctoral research, and now as an assistant professor, I have gained expertise in using combinations of structural and functional brain connectivity to investigate psychopathology. While this work has focused on investigating individuals with non-clinical levels or risk for psychosis, I have also been involved in investigations of disrupted connectivity in eating disorders, and my recent focus has been investigating the effects of alcohol and marijuana use on the structure and function of the brain. This work on substance abuse has involved data from 900 participants in the Human Connectome Project, and I will apply this experience to validate the connectivity algorithms being developed in the...
current project to data from the Human Connectome Project. I currently have a peer-review manuscript under revision that showed that white matter integrity, i.e., the strength of structural connections of the brain, shows a negative relationship with exposure to marijuana; that is, those who used more marijuana in their life and/or started using at a younger age, show reduced structural connectivity compared to those with less of a history of marijuana use. These same effects are observed in the shape of subcortical structures implicated in addiction, e.g., the accumbens.


c. Bernard JA, Orr JM, Mittal VA. Abnormal hippocampal-thalamic white matter tract development and positive symptom course in individuals at ultra-high risk for psychosis. NPJ Schizophr. 2015;1PubMed PMID: 26120591; PubMed Central PMCID: PMC4479398.


3. Although it is clear that the lateral prefrontal cortex plays an important role in executive function, its functional organization in less well understood. The prefrontal cortex is a large region of the brain, consisting of at least 10 discrete cytoarchitectonic areas. Moreover executive function is a diverse construct consisting of multiple domains such as working memory, inhibition, set shifting, problem solving, reasoning, multitasking, etc. While there have been a number of fruitful endeavors aimed at parcellating the prefrontal cortex based on functional connectivity patterns, these studies have not been aimed at developing/enhancing models of the functional organization of the prefrontal cortex. We have used a number of complimentary techniques including functional connectivity and diffusion tractography in combination with behavioral batteries in order to gain insights into the neural underpinnings of executive function. For example, using functional and diffusion MRI, we showed that inhibitory control of cognitive, emotional, and motor processes involved a common region of the right dorsolateral prefrontal cortex. Further, in a series of studies involving functional MRI, diffusion MRI, and meta-analysis I showed how the structure and function of the frontal pole supports a role for abstract goal-directed behavior.


Complete List of Published Work in My Bibliography:
http://bit.ly/1GAsxiD

D. Additional Information: Research Support and/or Scholastic Performance
**Completed Research Support**

L30 DA038580-01   Orr, Joseph M (PI)   07/01/14-06/30/16
Neural mechanisms of cognitive flexibility
Role: PI

F32 DA034412-03   Orr, Joseph M (PI)   04/01/13-07/31/15
Organization and timecourse of the neural mechanisms for cognitive flexibility
Role: PI
Curriculum Vitae

MARK GRAY PACKARD

CONTACT
Department of Psychology
Texas A & M University
979-764-8601 (H)
979-845-9504 (W)
979-845-7172 (Fax)
e-mail: markpackard@.tamu.edu

EDUCATION

1984 University of California, Santa Barbara
   B. A. Zoology
   B. Sc. Biopsychology

1986-1987 McGill University
   M. Sc. Experimental Psychology

1988-1991 McGill University
   Ph. D. Experimental Psychology

1991-1993 University of California, Irvine
   Center for the Neurobiology of Learning and Memory
   Post-Doctoral Fellow

PROFESSIONAL CAREER

1991-1993 Post-doctoral fellow, University of California, Irvine,
   Center for the Neurobiology of Learning and Memory

1993-6/1998 University of New Orleans
   Assistant Professor, Psychology
   (early tenure promotion to Associate Professor, approved
   Louisiana State Univ. Board of Regents 1/98, effective date 8/98)

7/1998-6/2001 Yale University
   Assistant Professor, Psychology

7/2001- 6/2002 Yale University
   Associate Professor, Psychology
8/2002-8/2005  Texas A & M University
Associate Professor, Psychology

8/2005-present  Texas A & M University
Full Professor, Psychology

AWARDS/RECOGNITION

Morgan Most Promising Researcher in Psychology, Undergraduate Award
Department of Psychology, University of California, Santa Barbara, 1984

Undergraduate Honors student in Biopsychology, Distinction in the Major
Department of Psychology, University of California, Santa Barbara, 1984

Early Career Achievement Award for Excellence in Research
University of New Orleans, 1995 (campus-wide, single faculty member recipient)

Yale University Junior Faculty Fellowship, 2000

“Essential Science Indicators top 1 %” Thompson Scientific (Analytical tracking of
research performance of 3 million worldwide scientists’ and lists the top one
percent of authors in terms of total publication citations, discipline of behavioral

Elected Fellow Association of Psychological Sciences, 2012
Elected Fellow American Psychological Association, 2013

GRANT REVIEW ACTIVITIES

Federal

U.S. Veterans Administration
Behavioral Neuroscience Program
external grant reviews 1998, 1999

National Science Foundation
Integrative and Behavioral Neuroscience Program

National Institutes of Health
B-Start Cognitive Neuroscience
external grant reviews 2001, 2002
NIH National Institutes of Mental Health
Special Emphasis Panel, Minority Training Grants
Panel meeting Washington, D.C. Fall, 2001
National Science Foundation

Behavioral Neuroscience and Endocrinology Grant Review Panel
Panel meeting Washington, D.C. Fall, 2002

National Science Foundation
Behavioral Neuroscience and Endocrinology Grant Review Panel
Panel meeting Washington, D.C. Spring, 2003

National Science Foundation
Behavioral Neuroscience and Endocrinology Grant Review Panel
Panel meeting Washington, D.C. Fall, 2003

National Science Foundation
Behavioral Neuroscience and Endocrinology Grant Review Panel
Panel meeting Washington, D.C. Spring, 2004

National Institutes of Health
Training Grant and Career Development Grant Review Panel, National Institutes of Neurological Disorders and Stroke
Panel meeting Washington D.C. Spring, 2004

National Institutes of Health (NIDA)
Neurotoxicology and Drug Abuse Grant Review Panel
Panel meeting, Washington DC Fall, 2004

National Science Foundation
Behavioral Neuroscience and Endocrinology Grant Review Panel
Panel meeting Washington, D.C. Fall, 2004

National Institutes of Health
Training Grant and Career Development Grant Review Panel, National Institutes of Neurological Disorders and Stroke
Panel meeting Washington, D.C. Fall, 2004

National Institutes of Health (NIDA)
Drug Abuse Special Emphasis Grant Review Panel
Panel meeting, Washington DC Spring, 2005
National Science Foundation
Behavioral Neuroscience and Endocrinology Grant Review Panel
Panel meeting Washington, D.C. Spring, 2005

National Institutes of Mental Health
NRSA and Postdoctoral Award Grant Review Panel Member
Panel meeting Washington, D.C. Fall, 2005

National Institutes of Health (NIDA)
Neurotoxicology and Drug Abuse Panel
Panel meeting, Washington DC Fall, 2005

National Institutes of Health
B-Start Grant Program
(external grant reviewer, 2004, 2005)

National Institutes of Mental Health
NRSA and Postdoctoral Award Grant Review Panel Member
Panel meeting Washington, D.C. Spring, 2006

National Institutes of Mental Health
Special Emphasis Panel Basic Neuroscience Conte Centers
Panel meeting Washington, D.C Fall 2006

National Institutes of Health
Behavioral Neuroscience, ICFN-7
Panel meeting, Fall, 2007

National Science Foundation
Systems Neuroscience: Modulation
Panel Meeting, Washington DC, Fall 2007

National Institutes of Health (NIDA)
Drug Abuse Special Emphasis Grant Review Panel Member
Panel meeting, Fall, 2007

Agency: National Institutes of Health (NIDA)
Drug Abuse Special Emphasis Grant Review Panel Member
Panel meeting, Fall, 2008

National Science Foundation
Systems Neuroscience: Modulation
Panel Meeting, Washington DC, Fall 2008
Agency: National Science Foundation (NSF)
Neural Systems Cluster: Behavioral Neuroscience
Panel member/meeting, Fall 2010

Agency: National Institutes of Health (NIDA)
Drug Abuse Special Emphasis Grant Review Panel Member
Panel meeting, Fall, 2012

Agency: National Science Foundation (NSF)
Neural Systems Cluster

**International**

United States-Russia Joint Behavioral Neuroscience Grant Program

United States- Israel Binational Science Foundation Grant Program

U.S. Civilian Research & Development Foundation, United States – Russia
Cooperative Grants Program, Behavioral Neuroscience

**Foundation**

Alzheimer's Association Grant Review and Medical Advisory Board (1998-
present)

**PROFESSIONAL MEMBERSHIPS**

Society for Neuroscience

American Psychological Society

Society for Behavioral Neuroendocrinology

American Psychological Association

International Society for Behavioral Neuroscience
EDITORIAL BOARDS

Hippocampus
Frontiers in Systems Neuroscience
Brazilian Journal of Neuropsychology

JOURNAL REFEREE (Ad-Hoc)

Proceedings of the National Academy of Sciences
Behavioral Neuroscience
Neurobiology of Learning and Memory
Physiology and Behavior
Pharmacology, Biochemistry, and Behavior
Neuroscience Letters
Journal of Neuroscience Research
Learning and Memory
Experimental Brain Research

Journal of Neuroscience
Behavioral Brain Research
Brain Research
European Journal of Pharmacology
Hormones and Behavior
European Journal of Neuroscience
Psychopharmacology
Neuroscience
Brain Research Bulletin

INVITED SEMINARS

Department of Neurology, University of California, Irvine, 1991
Center for the Neurobiology of Learning and Memory, UC Irvine, 1991
Department of Psychology, University of New Orleans, 1993
Department of Psychology, Tulane University, 1994
Department of Neuroscience, LSU Medical School, 1994
Department of Neurology, LSU Medical School, 1994
Department of Anatomy, LSU Medical School, 1996
Department of Psychology, Yale University, 1998
Neuroscience Department, Pfizer Incorporated, 1998
Department of Psychology, University of California, Santa Barbara, 1998
Department of Psychobiology, University of California, Irvine, 1998
Department of Psychology, Columbia University, 2000
Neurobiology of Learning and Memory Conference, Utah University, 2000
Department of Psychology, University of Oregon, 2001
Department of Psychiatry, Columbia University, 2001
Department of Psychology, University of Connecticut, 2001
Neuroscience Department, Pfizer Incorporated, 2001
Department of Psychology, Columbia University, 2001
Department of Psychology, McGill University, 2001
Center of Neurobiology and Behavior, Columbia University, 2001
Neurobiology of Learning and Memory Conference, Utah University, 2002
Department of Psychology, CUNY, 2002
Department of Psychology, Texas A & M University, 2002
Department of Psychology, University of Texas, 2002
Department of Psychology, University of Illinois at Chicago, 2004
Department of Neurobiology and Anatomy, University of Texas Medical School, 2004
Neurobiology of Learning and Memory Conference, Utah University, 2004
Neurobiology of Learning and Memory Conference, Utah University, 2005
Neurobiology of Learning and Memory Conference, Univ. of California, Irvine, 2006
Department of Neuroscience, University of South Carolina Medical School, 2007
Brain Research Meeting: Stress, Disease and Coping, Washington, DC, 2008
Society for Neuroscience Satellite Symposium, New Orleans, 2012
Neurobiology of Stress and Memory Conference, University of Texas, Dallas, 2012
Society for Biological Psychiatry Annual Conference, San Francisco, 2013
Department of Psychology, University of Texas, 2014
Department of Psychology, University of Southern Illinois, 2014
American Psychological Association Annual Conference, Washington DC, 2014
Society for Biological Psychiatry Conference, San Francisco, 2014

**Invited Seminars, International Conferences:**
McDonnell-Pew Foundation Conference, Montreal, Canada, 1997
FESBE Conference, Xacambu, Brazil, 1999
European Society for Behavioral Neuroscience Conference:
Emotional Modulation of Memory Symposium, Marsille, France, 2001
European Society for Behavioral Neuroscience Conference:
Basal Ganglia and Cognition Symposium, Barcelona, Spain, 2003
International Society for Behavioral Neuroscience, Sardinia, Italy, 2009

**Conference Organizer:**
Amygdala Interactions with other Brain Regions in Learning,
University of California, Irvine, 2001
Co-organizer with Dr. Larry Cahill, International Conference

**TEACHING**

**Undergraduate**
- Introductory Psychology
- Physiological Psychology
- Experimental Design
- Introductory Statistics
- Motivation
- Comparative Psychology
- Honors Psychology

**Graduate**
- Psychopharmacology
- Neurobiology of Learning and Memory
- Neurobiology of Multiple Memory Systems
- Physiological Psychology
JOURNAL PUBLICATIONS (REFEREED)


70. Packard, M. G. Anxiety, cognition, and habit: A multiple memory systems


**BOOK CHAPTERS**


BIOGRAPHICAL SKETCH

Provide the following information for the Senior/key personnel and other significant contributors in the order listed on Form Page 2. Follow this format for each person. DO NOT EXCEED FOUR PAGES.

<table>
<thead>
<tr>
<th>NAME</th>
<th>POSITION TITLE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vladislav M. Panin</td>
<td>Professor</td>
</tr>
</tbody>
</table>

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

EDUCATION/TRAINING (Begin with baccalaureate or other initial professional education, such as nursing, and include postdoctoral training.)

<table>
<thead>
<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
<th>YEAR(s)</th>
<th>FIELD OF STUDY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moscow State University, Moscow (USSR)</td>
<td>B.S., M.S.</td>
<td>1981-1987</td>
<td>Biophysics</td>
</tr>
<tr>
<td>Moscow State University, Moscow (USSR)</td>
<td>Ph. D.</td>
<td>1987-1990</td>
<td>Biophysics</td>
</tr>
<tr>
<td>Waksman Institute, Rutgers University, Piscataway, NJ</td>
<td>Postdoc</td>
<td>1995-2001</td>
<td>Developmental Biology &amp; Glycobiology</td>
</tr>
</tbody>
</table>

A. Personal Statement

The broad, long-term goal of my research is to understand the role of glycosylation in the development and physiology of the nervous system and reveal pathological mechanisms of neurological diseases associated with glycosylation defects. I accumulated 26 years expertise of working with Drosophila model system, including significant expertise in the fields of glycobiology, genetics, developmental biology and neurobiology. My research interests concentrate on evolutionarily conserved functions of glycoprotein sialylation in the nervous system development, neuromuscular functions and neurophysiology. Research of my laboratory focuses on elucidating genetic, cellular and molecular mechanisms of the sialylation pathway. My laboratory is well equipped and prepared for this research. Our previous studies discovered Drosophila sialyltransferase, the first sialyltransferase reported in protostomes. We revealed a novel pathway of glycoprotein sialylation that regulates neural transmission in the Drosophila nervous system. Our studies demonstrated that Drosophila sialylation is mediated by evolutionarily conserved enzymes, and characterized functions of several sialylation genes in vivo. Working together with mass spectrometry experts, we pioneered identification and characterization of sialylated glycans and analyses of Drosophila glycoproteome. Our laboratory, along with another group, discovered that Drosophila CMP-Sia synthetase, a key enzyme of the sialylation pathway, is uniquely localized in the cell secretary compartment, which established a novel family of CSAS enzymes working in ER-Golgi compartments. Our recent research revealed that sialylation is required for tolerance to oxidative stress, while defects in sialylation result in neurodegeneration. Our preliminary experiments discovered a novel sialylation-mediated regulatory mechanism involved in glia-neuron communication. We will capitalize on these results in the proposed project by focusing on function of sialylation in oxidative stress tolerance and neuronal viability pathways. We will apply advanced neurobiological and genetic approaches to elucidate involvement of sialylation in glia-neuron communication and the control of neural excitability. In collaboration with Christina Woo (Harvard University) we will use click chemistry strategy to identify and characterize sialylated glycoproteins. We will continue collaboration with Michael Tiemeyer (CCRC Georgia) in functional analyses of sialylated glycove of Drosophila. Via established collaboration with Dr. Zoran’s lab, we will explore sialylation-mediated mechanisms of glia-neuron communication in mammalian culture models. In summary, our project provides a unique opportunity of combining genetic tools, unsurpassed by those available in other model systems, with advanced neurobiological strategies in vivo, and approaches of glycobiology and chemical biology, which will facilitate elucidation of novel conserved mechanisms underlying functions of glycoprotein sialylation in the nervous system and will shed light on pathomechanisms of related diseases.


**B. Positions and Honors**

**Positions and Employment**

<table>
<thead>
<tr>
<th>Year</th>
<th>Position and Institution</th>
</tr>
</thead>
<tbody>
<tr>
<td>1984-1987</td>
<td>M.S. Trainee, Moscow State University, Moscow, Russia</td>
</tr>
<tr>
<td>1987-1990</td>
<td>Ph.D. Student, Moscow State University, Moscow, Russia</td>
</tr>
<tr>
<td>1990-1992</td>
<td>Junior Research Scientist, Engelhardt Institute of Molecular Biology, Drosophila Molecular</td>
</tr>
<tr>
<td></td>
<td>Genetics Laboratory, The Russian Academy of Sciences, Moscow, Russia</td>
</tr>
<tr>
<td>1992-1995</td>
<td>Research Scientist, Senior Research Scientist, Laboratory of Neurogenetics and Developmen</td>
</tr>
<tr>
<td></td>
<td>t Biology, Institute of Gene Biology, The Russian Academy of Sciences, Moscow, Russia</td>
</tr>
<tr>
<td>1994</td>
<td>Visiting Scientist, Laboratory of Mammalian Genetics and Neurobiology, Institut Pasteur,</td>
</tr>
<tr>
<td></td>
<td>Paris, France</td>
</tr>
<tr>
<td>1995-2001</td>
<td>Postdoctoral Research Associate, Waksman Institute of Microbiology, Rutgers University,</td>
</tr>
<tr>
<td></td>
<td>Piscataway, New Jersey, USA</td>
</tr>
<tr>
<td>2002-2008</td>
<td>Assistant Professor, Department of Biochemistry &amp; Biophysics, Texas A&amp;M University,</td>
</tr>
<tr>
<td></td>
<td>College Station, Texas, USA</td>
</tr>
<tr>
<td>2003-</td>
<td>Member, Faculty of Genetics, Texas A&amp;M University, College Station, TX</td>
</tr>
<tr>
<td>2008-2013</td>
<td>Chair, Graduate Recruitment and Admission Committee, Department of Biochemistry &amp;</td>
</tr>
<tr>
<td></td>
<td>Biophysics, Texas A&amp;M University, College Station, TX</td>
</tr>
<tr>
<td>2009-2012</td>
<td>Member, Faculty of Genetics Executive Committee, Texas A&amp;M University, College Station,</td>
</tr>
<tr>
<td></td>
<td>TX</td>
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<tr>
<td>2015-2016</td>
<td>-&quot;-</td>
</tr>
<tr>
<td>2008-</td>
<td>Member, Faculty of Neuroscience, Texas A&amp;M Institute for Neuroscience, College Station,</td>
</tr>
<tr>
<td>2008-2015</td>
<td>Associate Professor, Department of Biochemistry &amp; Biophysics, Texas A&amp;M University,</td>
</tr>
<tr>
<td></td>
<td>College Station, TX</td>
</tr>
<tr>
<td>2013-2014</td>
<td>Faculty Development Leave, Visiting Professor, Center for Integrative Genomics, University of Lausanne, Switzerland</td>
</tr>
<tr>
<td>2015-</td>
<td>Professor, Department of Biochemistry &amp; Biophysics, TAMU, College Station, TX</td>
</tr>
</tbody>
</table>

**Other Experience and Professional Memberships**

<table>
<thead>
<tr>
<th>Year</th>
<th>Experience</th>
</tr>
</thead>
<tbody>
<tr>
<td>1993</td>
<td>Summer School on Bioinformatics, International Center for Genetic Engineering &amp; Biotechnology, Trieste, Italy</td>
</tr>
<tr>
<td>1995</td>
<td>Invited Lecturer, Moscow State University, Moscow, Russia</td>
</tr>
<tr>
<td>1997 –</td>
<td>Member, The Genetics Society of America</td>
</tr>
<tr>
<td>2000 –</td>
<td>Member, The Society for Glycobiology</td>
</tr>
<tr>
<td>2009 –</td>
<td>Member, The Society for Neuroscience</td>
</tr>
<tr>
<td>2010 – 2014</td>
<td>Board of Directors, The Society for Glycobiology</td>
</tr>
<tr>
<td>2013 –</td>
<td>Editorial Board Member, Journal of Biological Chemistry</td>
</tr>
<tr>
<td>2013 –</td>
<td>Editorial Board Member, Glycobiology</td>
</tr>
</tbody>
</table>
Honors and Fellowships

1987 Diploma SummaCum Laude, Moscow State University, Moscow, Russia
1994 Honor State Fellowship for Outstanding Young Scientists from the President of Russia Boris Yeltsin, Moscow, Russia
1994 Fellowship from the International Science Foundation (est. by George Soros), New York, NY
1998 Charles and Johanna Busch Fellowship, Waksman Institute, NJ
2003 Basil O’Connor Starter Scholar Research Award, March of Dimes Foundation, NY
2013 Swiss National Science Foundation International Sabbatical Grant, University of Lausanne, Switzerland

C. Contribution to Science

1. In my previous research, I was focusing on genetic and developmental mechanisms that control Notch signaling pathway. My specific interests concentrated on Drosophila model of Notch regulation by Fringe, a glycosyltransferase that directly modifies O-fucose glycans on Notch, thus affecting interactions between the receptor and its ligands, Serrate and Delta. Together with my collaborators who shared my research interests while working on mammalian models of Notch signaling, I characterized the biochemical activity of Fringe and elucidated molecular, developmental and genetic mechanisms underlying this evolutionarily conserved pathway. My research also contributed to elucidation of conserved functional mechanisms of Notch regulation in mammalian organisms.


2. One of the central projects of my laboratory has been focused on protein O-mannosylation pathway. Our studies concentrate on evolutionarily conserved functions of protein O-mannosylation (POM) in the nervous system, neuromuscular development and muscle physiology. We elucidate genetic, cellular and molecular mechanisms that control O-mannosylation. Our research has established a Drosophila model of dystroglycanopathies. We demonstrated that Drosophila Dystroglycan is modified with O-mannosyl glycans by collaborative efforts of RT and TW, the two evolutionarily conserved homologues of mammalian protein O-mannosyltransferases 1 and 2 (POMT1/2), respectively. Working together with mass spectrometry experts, we pioneered identification of O-mannosylation sites on Dystroglycan and revealed interplay between protein O-mannosylation and O-linked GalNAc modification pathways. Our recent studies discovered that POMTs function in the peripheral nervous system to regulate muscle contractions (manuscript is under review).

3. Our strong interest in genetic and molecular mechanisms of evolutionarily conserved glycosylation pathways has shed light on functions of several novel glycosyltransferase genes and glycan structures that they produce. My early research in this direction contributed to revealing Fringe-related families of glycosyltransferases in Drosophila genome. In a collaborative research on protein O-fucosylation, we characterized the biochemical activity of a novel protein O-fucosyltransferases that modify thrombospondin type 1 repeats. We also shed light on genetics of β1,4-N-acetylgalactosaminyltransferase genes and their interactions with sialylation pathway. Together, these results elucidated several key O- and N-linked glycosylation pathways in Drosophila and revealed or suggested activities for evolutionarily conserved counterpart pathways in mammalian organisms.


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: PIETRANTONIO, PATRICIA V.

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

POSITION TITLE: Professor of Entomology and AgriLife Research Fellow - Insect Physiology and Toxicology

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.)

<table>
<thead>
<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
<th>Completion Date MM/YYYY</th>
<th>FIELD OF STUDY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Universidad de Buenos Aires, Bs. As., Argentina</td>
<td>Ing. Agr.</td>
<td>08/1982</td>
<td>Agronomy-(Fitotecnia)</td>
</tr>
<tr>
<td>National Institute of Agriculture and Cattle Tech. (INTA Castelar, Buenos Aires, Argentina)</td>
<td>INTA Fellow</td>
<td>08/1987</td>
<td>Insect Toxicology</td>
</tr>
<tr>
<td>University of California, Riverside, CA</td>
<td>M.S.</td>
<td>12/1990</td>
<td>Entomology- Tox</td>
</tr>
<tr>
<td>University of California, Riverside, CA</td>
<td>Ph.D.</td>
<td>09/1995</td>
<td>Entomology- Tox-Phys</td>
</tr>
</tbody>
</table>

A. Personal Statement

I have a consistent record as PI of federally funded research, teaching and service in Insect Toxicology and Physiology. My research is broad-based and includes: 1) applied pest control aspects in hematophagous and crop pests and 2) the discovery of novel pesticide targets in fire ants, mosquitoes and ticks. 1) On the applied aspect we determine the insecticide resistance status of populations and elucidate the molecular mechanisms of pesticide resistance in arthropods of public and/or animal health importance and those of production agriculture. My laboratory led, in collaboration with the Harris County Mosquito Control division, the first published study on insecticide resistance in *Cx. quinquefasciatus* mosquitoes in Harris County, TX (Houston) and the mechanism of resistance; this study supported changing control practices. 2. In the basic aspect we search for novel targets for feeding deterrents or pesticides in the yellow fever mosquito *Aedes aegypti*, and in ticks, the Lyme disease vector *Ix. scapularis* and the cattle fever tick *R. microplus*. We focus on G protein-coupled receptors of neuropeptides and biogenic amines involved in water balance. With potential for patent development we have just discovered the aversive response of *Aedes aegypti* females to a leucokinin peptidomimetic that shuts down the sugar neuron and triggers fly away, walk away or jump away behavior. We utilize bioassays, cellular biology tools, recombinant receptor bioluminescence assays, RNAi, physiological assays and other molecular techniques. I have served as Major Professor of 7 Ph.D. students, 3 M.S., 1 M. Ag., mentored 7 post-docs and many international visiting scientists, all receiving training in molecular insect sciences and toxicology. I have been recognized for mentoring students and faculty alike.

B. Positions and Honors

Positions
1987-1990 International Rotary Foundation Competitive Scholarship holder for MS at UCR, CA.
1990-1995 Research Assistant, Dept. of Entomology. UC Riverside, CA.
Honors

International Competitive Scholarship. Rotary International Foundation: Freedom From Hunger, for completion of the M.S. in Entomology at the University of California, Riverside. 1987-1990. Tuition, fees and board ($65,000).


Registration Award by Committee Selection by USDA/CREES. Fifth International Symposium in Molecular Insect Science. Tucson, Arizona, May 21, 2006.


TAMU Executive Vice President and Provost’s Diversity Award for “Faculty Individual Achievement” in contributing to diversity of students and faculty. Texas A&M University, April 5, 2004. One award per year.

Texas A&M Experiment Station (TAES) Fellow: “For significant scholarly accomplishments and meaningful contributions to science through exceptional research leadership and grantsmanship.” January 10, 2006. Plaque and $4,000 award. Eight awarded.


Environmental Health and Safety Award to P.V. Pietrantonio and Insect Toxicology Laboratory. August 2013. Safest lab among 400 laboratories inspected at TAMU.


C. Contributions to Science (chronological order)

Neurobiology and molecular reproductive physiology in the red imported fire ant Solenopsis invicta


Functional analyses of G protein-coupled receptor-ligand interactions in *Aedes aegypti* mosquitoes and ticks with peptidomimetics: toward rational pesticide design and lead identification.


Taneja-Bageshwar, S., Strey, A. R. E. Isaac; G. M Coast; Zubrzak, P., Pietrantonio, P.V.* and Nachman, R.J.* 2009. Biostable agonists that match and/or exceed the activity of insect kinins on recombinant arthropod GPCRs. General & Comparative Endocrinology 162: 122-128.


Molecular regulation of water balance in ticks: tick genomics, and GPCR signaling for neuropeptides and serotonin

We characterized the first neuropeptide receptor from the Acari and the first neuropeptides by MALDI-TOF. We identified, cloned and helped annotate GPCRs for the genome publication of the tick *Ix. scapularis*.


Molecular regulation of water balance in Aedes aegypti: GPCR signaling and aquaporin

We pioneered the identification and characterization of G protein-coupled receptors from mosquitoes and ticks before the availability of genomic resources. We also characterized the second aquaporin from insects from the mosquito Ae. aegypti and proposed a mechanism of function of this aquaporin that explains Sir. Wigglesworth’s theory of tracheolar physiology.


See my bibliography at NCBI:
http://www.ncbi.nlm.nih.gov/pubmed?term=pietrantonio%20p%5Bau%5D%20AND%20College%20Station%5Bad%5D&cmd=search

D. Research Support
ONGOING RESEARCH SUPPORT

C-REEMS No: 2015-07009
GRANT11884618 Pietrantonio (PI) Temeyer (co-PI) 03/2016 – 02/2019 USDA NIFA-AFRI
Title: Towards Novel Acaricide Development Against Cattle Fever Tick: GPCR Target Validation and Identification of Chemical Leads

Award Number: 1257837 Pietrantonio (PI) Tamborindegeuy (Co-PI) 3/2013-2/2017 NSF-IOS
Title: Neuropeptide Receptors and Identification of Genes in Signaling Networks Involved in Reproduction and Nutrition in the Red Imported Fire Ant

CDC Grant to UTMB-TAMU. Gulf Coast Center of Excellence for Vector-borne Diseases (Scott Weaver, PI) Subaward to Pietrantonio, co-PD. $100,000 yearly from Jan 2017-2021.

Grant No. 114188 Pietrantonio (PI) Shen (coPI) 09-2016/08-2017 TA&M AgriLife Research Insect Vector Disease program
Title: Discover new targets and develop novel chemicals to deter mosquito feeding (or other blood-sucking vectors feeding) using a GPCR1 pipeline and computational modelling

Texas Invasive Ant Research and Management Seed Grant Program. Pietrantonio (PI) FY 16-17
Title: Fire ant brain-gut-ovary signaling: neuropeptides and receptors as the key to reproduction interference.

Project No. TX 11-940 Pietrantonio (PI) 1/2016- 12/16 Cotton Incorporated
Title: Development of molecular diagnostic tools for detection of Cry1Ac/Cry1Ab (Bt) resistance in bollworm (H. zea).

COMPLETED RESEARCH (last 3 years)
Project No. TX 11-940 Pietrantonio, PV. PI. Cotton Incorporated
Development of molecular diagnostic tools for detection of Cry1Ac/Cry1Ab (Bt) resistance in bollworm (H. zea). Renewed annually since 2011-2016.

Texas Fire Ant Research and Management Project. Pietrantonio, PV. (PI) FY 2012-2013 TX AgriLife Research.
Title: Brain and ovary receptors as novel targets to manage the Red Imported Fire Ant, *Solenopsis invicta* Buren.


Title: Neuropeptides and their receptors controlling reproduction, nursing and foraging as targets to manage invasive ants.
CURRICULUM VITAE
(Michelle Pine, D.V.M., Ph.D.)
(8/19/10)

PRESENT POSITION AND ADDRESS:

Title: Clinical Assistant Professor
Office: 68 VMS     Phone: (979) 458-0594

Home: 1700 Lee, Brenham, TX     Phone: (979) 277-5140

EDUCATION:

<table>
<thead>
<tr>
<th>Degree/Training</th>
<th>Conferring Institution</th>
<th>Field</th>
<th>Year</th>
</tr>
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<tbody>
<tr>
<td>B.S.</td>
<td>University of Missouri-Columbia</td>
<td>Animal Science</td>
<td>1987</td>
</tr>
<tr>
<td>D.V.M.</td>
<td>University of Missouri-Columbia</td>
<td>Veterinary Medicine</td>
<td>1991</td>
</tr>
<tr>
<td>Ph.D.</td>
<td>Texas A&amp;M University</td>
<td>Toxicology</td>
<td>2002</td>
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</tbody>
</table>

PROFESSIONAL EXPERIENCE AND ACADEMIC APPOINTMENTS:

CLINICAL POSITIONS

Veterinary Practitioner, Phelps County Veterinary Clinic, Rolla, MO 1991-92
Manager/Staff Veterinarian, Central MO Humane Society, Columbia, MO 1992-93
Staff Veterinarian, Jefferson City Animal Shelter, Jefferson City, MO 1993-94
Veterinary Practitioner, Boonville Veterinary Clinic, Boonville, MO 1993-94

GOVERNMENT POSITIONS

Veterinary Medical Officer (GS12), USDA, Laurel, MS 1994-98

ACADEMIC APPOINTMENTS

Postdoctoral Research Fellow, Veterinary Integrative Biosciences 2003-04
Texas A&M University (Dr. W. Les Dees)
Research Assistant Professor, Veterinary Integrative Biosciences 2004-06
Texas A&M University
Clinical Assistant Professor, Veterinary Integrative Biosciences 2006-Present
Texas A&M University
Faculty of Neuroscience, Texas A&M University 2006-Present
Interdisciplinary Faculty of Toxicology, Texas A&M University 2007-Present
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AWARDS AND HONORS:
                Jesse Lyn Memorial Christian Leadership, Elizabeth H. Schell
Certificate of Merit, United States Department of Agriculture 1998
Regents Graduate Fellow, Texas A&M University 1998
Best Reproductive Toxicology Publication of the Year Award 2006 2007
                IGF-1 Administration to Prepubertal Female Rats Can Overcome Delayed
                Awarded by The European Teratology Society.

GRANTS AND FUNDING
2010-2011 Texas AgriLife Research Vector-Borne Disease Program
        “Vector Eradication: Delivery of Novel Pesticide Encapsulated Nanoparticles”
        $100,000 total award.
        PI: Michelle Pine
        Co-PIs: Christie Sayes, Patricia Pietrantonio, and Brian Porter

TEACHING EXPERIENCE:
        I have been involved in teaching gross anatomy to both undergraduate and professional
        students beginning in the fall of 1998 as a teaching assistant and continuing through my postdoctoral
        training. As a faculty member during the spring of 2007, I assisted in both lab sections of Gross
        Anatomy II (VIBS 912) this also included the live horse palpation. Because this class has two
        sections, I was physically in the classroom 15 hours/week. Preparation time for the dissections and
        live horse palpations required another 20-25 hours/week. Spring 2008 I coordinated both laboratory
        sections of Biomedical Anatomy 305. I was physically in the classroom 10 hours per week. I also
        wrote, set up, and graded lab exams. Fall 2008 I was course coordinator and oversaw the lecture
        portion of the course. This required preparing lecture slides on power point and writing and grading
        lecture exams. Spring 2009 I was the sole course coordinator for 305. This involved lecturing and
        overseeing both lab sections (10 hours/week) plus writing and grading exams and providing office
        hours for students (2 hrs/week).

Undergraduate:

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<th>% of Course</th>
<th>Credit Hr</th>
<th>Dates</th>
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<td>TAMU</td>
<td>4</td>
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<tr>
<td>Biomedical Anatomy 305</td>
<td>TAMU</td>
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<td>50%</td>
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<td>8/08-12/08</td>
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<tr>
<td>Biomedical Anatomy 305</td>
<td>TAMU</td>
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<td>50%</td>
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Curriculum Vitae
Michelle Pine
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Professional:

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<th>Institution</th>
<th>Credit</th>
<th>% of Course</th>
<th>Formal Contact Hr</th>
<th>Dates</th>
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<tr>
<td>Gross Anatomy II 912</td>
<td>TAMU</td>
<td>4</td>
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<td>1/07-5/07</td>
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Invited Lectures/Graduate and Undergraduate

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<th>Dates</th>
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<tr>
<td>Neuroendocrine Anatomy 603</td>
<td>TAMU</td>
<td>Guest lecture</td>
<td>Fall 2004</td>
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<tr>
<td>Biomedical Neuroendocrinology &amp; Endocrine Disorders 604/489</td>
<td>TAMU</td>
<td>Guest lecture</td>
<td>Fall 2007</td>
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<tr>
<td>Developmental Neurotoxicology 489</td>
<td>TAMU</td>
<td>Guest lecture</td>
<td>Fall 2009</td>
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<td>Basic Environmental Toxicology</td>
<td>TAMU</td>
<td>Guest lecture</td>
<td>Fall 2009</td>
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<tr>
<td>VIBS 670/PHEO 610</td>
<td>TAMU</td>
<td>Guest lecture</td>
<td>Fall 2009</td>
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Undergraduate Students Research:

<table>
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<th>Institution</th>
<th>Advisor or Committee Member</th>
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<tbody>
<tr>
<td>Marco Chavez</td>
<td>TAMU Undergrad Research Scholar</td>
<td>Advisor</td>
<td>2007-08</td>
</tr>
<tr>
<td>Kendra Hewitt</td>
<td>TAMU directed studies Research</td>
<td>Advisor</td>
<td>2009</td>
</tr>
</tbody>
</table>

Undergraduate Students Teaching:

<table>
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<th>Institution</th>
<th>Advisor or Committee Member</th>
<th>Dates</th>
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</thead>
<tbody>
<tr>
<td>Roy Wilmeth</td>
<td>TAMU BIMS 485 Anatomy TA</td>
<td>Advisor</td>
<td>2008</td>
</tr>
<tr>
<td>Ben Curtis</td>
<td>TAMU BIMS 485 Anatomy TA</td>
<td>Advisor</td>
<td>2009</td>
</tr>
<tr>
<td>Fara Flados</td>
<td>TAMU BIMS 485 Anatomy TA</td>
<td>Advisor</td>
<td>2009</td>
</tr>
<tr>
<td>Amber Woodin</td>
<td>TAMU BIMS 485 Anatomy TA</td>
<td>Advisor</td>
<td>2009</td>
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Undergraduate Student Worker:

<table>
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<th>Advisor or Committee Member</th>
<th>Dates</th>
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<tbody>
<tr>
<td>Ricky Volz</td>
<td>TAMU</td>
<td>Supervisor</td>
<td>2006</td>
</tr>
<tr>
<td>Taylor Wagner</td>
<td>TAMU</td>
<td>Supervisor</td>
<td>2009</td>
</tr>
<tr>
<td>Amy Blankenship</td>
<td>TAMU</td>
<td>Supervisor</td>
<td>2010</td>
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Graduate Students

<table>
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<tbody>
<tr>
<td>Stephanie Mattair</td>
<td>TAMU (BIMS Masters)</td>
<td>Advisor</td>
<td>2009-</td>
</tr>
<tr>
<td>Abraham Robinson</td>
<td>TAMU (TOXI, PhD)</td>
<td>Committee Member</td>
<td>2009-10</td>
</tr>
<tr>
<td>Michael Villanuevos</td>
<td>TAMU (BIMS Masters)</td>
<td>Advisor</td>
<td>2010</td>
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<tr>
<td>Xin Guo</td>
<td>TAMU (TOXI, Masters)</td>
<td>Committee Member</td>
<td>2010</td>
</tr>
<tr>
<td>Aishwarya Sooresh</td>
<td>TAMU (MSE, PhD)</td>
<td>Co Advisor</td>
<td>2010</td>
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Residents/Interns/Postdoctoral Fellows:

<table>
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<th>Name</th>
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</tr>
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<tbody>
<tr>
<td>Brian Laffin</td>
<td>Postdoctoral Fellow TAMU</td>
</tr>
</tbody>
</table>

BIBLIOGRAPHY:

Publications in Refereed Journals:


**Named the 2006 Best Reproductive Toxicology Publication of the Year by the European Society of Teratology.**

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Brian Laffin, Marco Chavez, and Michelle Pine. 2009. The pyrethroid metabolites 3-phenoxybenzoic acid and 3-phenoxybenzyl alcohol do not exhibit estrogenic activity in the MCF-7 human breast carcinoma cell line or Sprague-Dawley rats. Toxicology (accepted for publication).

Abstracts:


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Michelle D. Pine. The type II pyrethroid pesticide esfenvalerate lowers serum estradiol and delays the onset of puberty. 46th annual meeting of the Society of Toxicology, Charlotte, NC. March, 2007.

Michelle D. Pine and Jill K. Hiney. Esfenvalerate acts at the hypothalamus to suppress the afternoon rise of luteinizing hormone in prepubertal female rats. 47th annual meeting of the Society of Toxicology, Seattle, WA. March, 2008.


Michelle Pine, Brian Laffin, and Marco Chavez. In Vitro and In Vivo Assessment of Estrogenic Activity of the Pyrethroid Metabolites 3-Phenoxybenzoic Acid and 3-Phenoxybenzyl Alcohol. 48th annual meeting of the Society of Toxicology, Baltimore, MD. March, 2009.

Brian Laffin and Michelle Pine. Neurodevelopmental effects of in utero deltamethrin exposure. 48th annual meeting of the Society of Toxicology, Baltimore, MD. March, 2009.

Kendra Hewitt, Brian Laffin, and Michelle Pine. Perturbation of DARPP-32 phosphorylation following in utero Deltamethrin Exposure. 1st Annual Texas A&M University and Health Science Center Faculty of Neuroscience Symposium, May, 2009.


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Michelle Pine
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**PATENTS AND RELATED DISCOVERY ACTIVITIES:**

**SERVICE ACTIVITIES:**
**Professional Organizations and Service:**

American Association of Veterinary Anatomists
Society of Toxicology
Gulf Coast Society of Toxicology
Society for Neuroscience
Gamma Sigma Delta
Phi Kappa Phi Honor Society
International Association of Aquatic Animal Medicine
Deputy State Veterinarian (Missouri)
Missouri Veterinary Licensee (current status inactive)
Veterinary Accreditation, USDA, APHIS
Manuscript Review of Journals:
Neurotoxicology
Pesticide Biochemistry and Physiology
Journal of Applied Toxicology

Major Committee Assignments:

Departmental: Teaching Committee for Undergraduate and Professional Education
Member, internal faculty evaluation committee for Dr. Anton Hoffman

College: Faculty Interviewer for selection of the Veterinary Classes of 2011-13
Selection Committee for the Veterinary Class of 2014 (replacement for Dr. Melanie Landis)
Biographical Sketch

Provide the following information for the key personnel in the order listed for Form Page 2. Follow this format for each person. DO NOT EXCEED FOUR PAGES.

<table>
<thead>
<tr>
<th>NAME</th>
<th>POSITION TITLE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brian F. Porter</td>
<td>Clinical Associate Professor, Veterinary Pathobiology, College of Veterinary Medicine, Texas A&amp;M University</td>
</tr>
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EDUCATION/TRAINING (Begin with baccalaureate or other initial professional education, such as nursing, and include postdoctoral training.)

<table>
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<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE</th>
<th>YEAR(s)</th>
<th>FIELD OF STUDY</th>
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<tbody>
<tr>
<td>Texas A&amp;M University</td>
<td>B. S.</td>
<td>1989</td>
<td>Veterinary Science</td>
</tr>
<tr>
<td>Texas A&amp;M University</td>
<td>D. V. M.</td>
<td>1992</td>
<td>Veterinary Medicine</td>
</tr>
</tbody>
</table>

NOTE: The Biographical Sketch may not exceed four pages. Items A and B may not exceed two of the four-page limit.

A. Positions and Honors. List in chronological order previous positions, concluding with your present position. List any honors. Include present membership on any Federal Government public advisory committee.

ACADEMIC APPOINTMENTS:
2000-2003 Resident, Veterinary Pathology, Cornell University, Ithaca, NY
2003-2004 Veterinary Pathologist, Texas Veterinary Medical Diagnostic Laboratory, College Station, TX
2004-2011 Clinical Assistant Professor, Department of Veterinary Pathobiology, College of Veterinary Medicine, Texas A&M University, College Station, TX
2011- Clinical Associate Professor, Department of Veterinary Pathobiology, College of Veterinary Medicine, Texas A&M University, College Station, TX

HONORS AND AWARDS:
2012 Pfizer Carl J. Norden Distinguished Teacher Award
2009 Veterinary Medical Teaching Hospital Clinical Service Award
2003 Diplomate, American College of Veterinary Pathologists
2002 ACVP Young Investigator Award, Natural Disease Poster, Third Place, American College of Veterinary Pathologists Meeting, New Orleans

B. Selected (>180) peer-reviewed publications (in reverse chronological order). Do not include publications submitted or in preparation.

13. Ajilothoss Dk, Porter BF, Calise DV, Libal MC, Edwards JF: Septicemia in a neonatal calf associated with...
52. Plumlee QC, Meason-Smith C, Dieterly AM, Gomez G, **Porter BF**: Chaetomiaceae fungi, novel pathogens of equine neurotropic phaeohyphomycosis. Vet Pathol (in press)

C. Research Support. List selected ongoing or completed (during the last three years) research projects (federal and non-federal support). Begin with the projects that are most relevant to the research proposed in this application. Briefly indicate the overall goals of the projects and responsibilities of principal investigator identified above.

American Kennel Club- Canine Health Foundation Grant No. 640, 2006-2008. Linkage disequilibrium analysis of markers associated with pug dog encephalitis. (P.I.- Kimberly Greer); $60,000

NAME: Jayanth Ramadoss                Title: Assistant Professor

<table>
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<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
<th>Date MM/YYYY</th>
<th>FIELD OF STUDY</th>
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<tbody>
<tr>
<td>BITS, Pilani, India</td>
<td>B.E. (Hons.)</td>
<td>1997-2002</td>
<td>Electronics and Instrumentation</td>
</tr>
<tr>
<td>BITS, Pilani, India</td>
<td>M.Sc. (Hons.)</td>
<td>1997-2002</td>
<td>Chemistry</td>
</tr>
<tr>
<td>Texas A&amp;M University, College Station, USA</td>
<td>Ph.D.</td>
<td>2004-2008</td>
<td>Biomedical Sciences</td>
</tr>
<tr>
<td>University Of Wisconsin, Madison, USA</td>
<td>Post-Doctoral</td>
<td>2008-2011</td>
<td>Obstetrics and Gynecology</td>
</tr>
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</table>

### A. Personal Statement

The overall long term goals of my lab are to investigate the role of maternal-fetal interaction in the etiology of fetal alcohol spectrum disorders, to develop state of the art non-invasive biomarkers for maternal alcohol consumption, to investigate fetal alcohol programing of adult-onset vascular and endocrine disease states, and to discover means to enhance intrauterine environment that may have enduring and life-long health benefits for the offspring. I am committed to promote collaborative interactions with clinician scientists and veterinarians, and build strategic multi-investigator projects. My current teaching focus is on physiology training of bioengineering students. My goal is to provide training to develop engineering solutions for medical problems. In my teaching, I strive to bridge gaps between basic science and clinical perspectives, and thus foster an appreciation of the significance of integrative physiology in real life scenarios. Utilizing state of the art teaching approaches, I aspire to actively engage students and facilitate problem solving. My overall goal is to establish best practices in teaching integrative physiology.

### B. Positions and Honors.

#### Positions and Employment

<table>
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<tr>
<th>Date</th>
<th>Position Description</th>
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<tr>
<td>01/2004-12/2007</td>
<td>Research assistant</td>
<td>Texas A&amp;M University, College Station, TX</td>
</tr>
<tr>
<td>01/2008-06/2010</td>
<td>Research associate</td>
<td>University of Wisconsin, Madison, WI</td>
</tr>
<tr>
<td>07/2010-09/2011</td>
<td>Assistant Scientist</td>
<td>University of Wisconsin, Madison, WI</td>
</tr>
<tr>
<td>10/2011-Present</td>
<td>Assistant Professor-Tenure Track</td>
<td>University of Texas Medical Branch, Galveston, TX</td>
</tr>
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</table>

#### Honors & Awards (SELECT)

Research Grant Award, Office of Graduate Studies, Texas A&M University (2005)
Outstanding Graduate Student Award, CVM Texas A&M University (2007)
NIH Young Investigator award, Perinatal Research Society, Santa Fe, New Mexico (2008)
Podcast interview, American Journal of Physiology-Integrative and Regulatory (2008)
FASDSG Merit Award, Research Society on Alcoholism, Washington DC (2008)
RSA Junior Investigator Award, Research Society on Alcoholism, (2009)
USDA Sponsored Aspen Perinatal Biology New Investigator Award, Aspen CO (2010)
Enoch Gordis Award Winner, Research Society on Alcoholism, San Antonio, TX (2010)
Larry Ewing Travel Award, Society for Study of Reproduction, Milwaukee, WI (2010)
Pfizer President’s Presenter Award, Society for Gynecological Investigation (SGI), (2010)
University of Wisconsin Hildale Award to E. Schuler; Mentors, J. Ramadoss/R. Magness, (2011)
Podcast Interview, American Journal of Physiology Heart and Circulation, (2012)
Speaker, NIH/NIAAA, PASS Network Study Group Talk, Rockville, MD (2012)
Speaker, NIH/NICHD, PASS Network Study Group Talk, Bethesda, Maryland (2013)
Symposium Co-Chair and Organizer, Research Society on Alcoholism, Orlando, FL (2013)
Pfizer Award from SGI awarded to Mr. Naik; Mentor, J. Ramadoss (2013)
Ad hoc Reviewer, NIH AA-1 Biomedical Research Review Subcommittee (June, October 2014)
Ad hoc Reviewer, NIH AA-1 Biomedical Research Review Subcommittee (March, June 2015)
Member, NIH ZRG1 EMNR D 55 R (July, 2015).
Ad hoc Reviewer, NIH AA-1 Biomedical Research Review Subcommittee (June, October 2015)
Member, NIH ZAA1 GG Special Emphasis Panel (November, 2015)
Ad hoc Reviewer, NIH PN Pregnancy and Neonatology Study Section (February, 2016)
Reviewer, NIH ZRG1 EMNR-A (55) R, (April 2016)
Outstanding Young Faculty Research Award, CVM, Texas A&M University (2016)
Academic Program Review, May 2017

Juan Carlos Robles Emanuelli Teaching Award, CVM, Texas A&M University (2016)
Reviewer, NIH NIGMS ZGM1 RCB-9(CI) (July, 2016)
Reviewer, NIH 2016/10 ZAA1 JJ (01) 1 Special Emphasis Panel (July, 2016) Outstanding Young Faculty Research Award, College of Veterinary Medicine and Biomedical Sciences, Texas A&M University 2016 Juan Carlos Robles Emanuelli Teaching Award, College of Veterinary Medicine and Biomedical Sciences, Texas A&M University
RSA Junior Investigator Award to Katie Davis (Postdoctoral fellow), Mentor: Dr. Ramadoss (2016)
RSA Student Merit Award to Raine Lunde (Graduate Student), Mentor: Dr. Ramadoss (2016)
RSA Student Merit Award to Marcus Orzabal (Undergraduate Student), Mentor: Dr. Ramadoss (2016)
CVM Outstanding Masters Non-Thesis Award to Raine Lunde (Grad Student), Mentor: Dr. Ramadoss (2016)
Texas A&M University Walter W. Lechner Estate Scholarship awarded to Raine Lunde (Graduate Student), Mentor: Dr. Ramadoss (2016)
Reviewer, NIH ZAA1 JJ (01) – Council 2017/05 Special Emphasis Panel (April, 2017)

TEACHING RESPONSIBILITIES

OBGYN (AY 2013-14): Renal, Fluids, and Electrolytes, University of Texas Medical Branch (PBL Facilitator, Fall 2013).

PBL global assessment of tutor score for Fall 2013: 5.00 out of 5.00

OBGYN (AY 2013-14): Renal, Fluids, and Electrolytes, University of Texas Medical Branch (PBL Facilitator, Fall 2014; lecture for Fall 2014).

PBL global assessment of tutor score for Fall 2014: 5.00 out of 5.00

VTPP 434 (section 501): Physiology for Bioengineers I, Texas A&M University (Fall 2015); Course coordinator; Instructor for endocrinology, neurophysiology, muscle physiology, laboratory coordinator. (Class size: ~66) (total of ~21 lecture contact hours, ~13 lab contact hours, course coordination)

Student Evaluation rating: 4.90 out of 5.00

VTPP 434 (section 502): Physiology for Bioengineers I, Texas A&M University (Fall 2015); Course coordinator; Instructor for endocrinology, neurophysiology, muscle physiology, laboratory coordinator. (Class size: ~69) (total of ~21 lecture contact hours, ~13 lab contact hours, course coordination)

Student Evaluation rating: 4.90 out of 5.00

VTPP 435 (section 501): Physiology for Bioengineers II, Texas A&M University (Spring 2016); Course coordinator; Instructor for Vascular, respiratory, renal physiology. (Class size: ~58) (total of ~26 lecture contact hours and course coordination)

Student Evaluation rating: 4.97 out of 5.0

VTPP 435 (section 502): Physiology for Bioengineers II, Texas A&M University (Spring 2016); Course coordinator; Instructor for Vascular, respiratory, renal physiology. (Class size: ~55) (total of ~26 lecture contact hours and course coordination)

Student Evaluation rating: 5.00 out of 5.0

VTPP 434 (section 501 and 503): Physiology for Bioengineers I, Texas A&M University (Fall 2016); Course coordinator; Instructor for fluid balance, endocrinology, neurophysiology, muscle physiology. (Class size: ~75) (total of ~26 lecture contact hours and course coordination)

Student Evaluation rating: 4.96 out of 5.0

VTPP 434 (section 502, 504): Physiology for Bioengineers I, Texas A&M University (Fall 2016); Course coordinator; Instructor for fluid balance, endocrinology, neurophysiology, muscle physiology. (Class size: ~75) (total of ~26 lecture contact hours and course coordination)

Student Evaluation rating: 4.98 out of 5.0

PUBLICATIONS:

C. RESEARCH SUPPORT

R01AA23520 (Ramadoss, PI) 04/01/2015 – 03/31/2020 Total Award Amount: $1,636,875 NIH NIAAA; Alcohol and maternal uterine vascular adaptations in pregnancy.

R21AA23035 (Ramadoss, PI) 09/05/2015-08/31/2017 Total Award Amount: $421,169 NIH NIAAA; A novel platform for maternal alcohol consumption screening.

R00AA19446 (Ramadoss, PI) 09/27/2011–08/31/2016 Total Award Amount: $729,570 NIH/NIAAA; Maternal Uterine Vascular Origins of FASD

Tier One Program Grant (Ramadoss, Contact PI) 09/01/2016-08/31/2019 Direct Cost: $288,000 Texas A&M University; Collaborative Learning Initiatives in Maternal, Perinatal, and Infant Health Research

K99AA19446 (Ramadoss, PI) 04/01/2010–09/26/2011 Total Award Amount: $267,072 NIH/NIAAA; Maternal Uterine Vascular Origins of FASD

Interdisciplinary Research in Women’s health (Ramadoss, PI) 03/04/2014-08/31/2014 Direct Cost: $15,000 University of Texas Medical Branch; Protein Signature Profile for Chronic Pelvic Pain in Women
BIOGRAPHICAL SKETCH

Provide the following information for the Senior/key personnel and significant contributors. Follow this format for each person. DO NOT EXCEED 5 PAGES.

NAME: Doodipala Samba Reddy, Ph.D., R.Ph., FAAPS, FAAAS, FAES

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

POSITION TITLE: Professor, Neuroscience & Experimental Therapeutics, and NIH CounterACT Investigator

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.)

<table>
<thead>
<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE</th>
<th>Completion Date MM/YY</th>
<th>FIELD OF STUDY</th>
</tr>
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<tr>
<td>Kakatiya University, Warangal, India</td>
<td>B.S.</td>
<td>1992</td>
<td>Pharmaceutical Sci.</td>
</tr>
<tr>
<td>Panjab University, Chandigarh, India</td>
<td>M.S.</td>
<td>1994</td>
<td>Pharmacology</td>
</tr>
<tr>
<td>Panjab University, Chandigarh, India</td>
<td>Ph.D.</td>
<td>1998</td>
<td>Neuropharmacology</td>
</tr>
<tr>
<td>NINDS, National Institutes of Health, USA</td>
<td>Post-doc</td>
<td>1998-2001</td>
<td>Epilepsy Neuroscience</td>
</tr>
<tr>
<td>Texas Board of Pharmacy, Austin, TX, USA</td>
<td>R.Ph.</td>
<td>2009</td>
<td>Regd. Pharmacist</td>
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</table>

A. Personal statement

I am a NIH CounterACT investigator working on novel therapeutics for OP pesticides and nerve agents. My research interests are centered to understand the molecular pathophysiology and develop novel translational therapeutic strategies for epilepsy, brain injury, and chemical neurotoxicity. We have proposed neurosteroids as highly effective anticonvulsants against organophosphate and nerve agent-induced seizures and brain injury.

I have the expertise, leadership and motivation necessary to carry out the studies proposed in this project. Over the last 20 years, our studies in preclinical models have shown that GABAergic agents and neurosteroids are robust anticonvulsants and there is new evidence that they exert neuroprotective effects. I have a broad background in neuropharmacology, with specific training and expertise in epilepsy research. My development as a scientist results directly from my professional education and training in pharmacy. I received extensive training in neuropharmacology, electrophysiology and neuroscience from excellent mentors. I worked as a postdoctoral fellow at the intramural NINDS Epilepsy Research Branch, and then served as a faculty member at NC State University for 6 years prior to joining Texas A&M. My lab is primarily interested in epilepsy research, with special emphasis on drug development, identifying molecular mechanisms of neurosteroids, and testing the efficacy of mechanism-based drugs for epileptogenesis and status epilepticus. Neurosteroids are steroids synthesized locally within the brain that rapidly change neural excitability by non-genomic mechanisms, principally via postsynaptic GABA-A receptors that play critical role in controlling excessive neuronal excitability. Recently, we began establishing a new translational project on epigenetic therapy. Our lab is utilizing multidisciplinary (pharmacological, molecular, immunohistochemical, and electrophysiological) approaches in research projects. Over the past many years, we have developed animal models of catamenial epilepsy, determined the mechanisms of neurosteroids actions, and their clinical potentials in brain disorders. My lab has been among the first to propose ‘neurosteroid replacement’ as a specific treatment for epilepsy and catamenial epilepsy. Recently, we showed a crucial role of PRs in epileptogenesis, and also obligatory role for tonic inhibition in limbic epileptogenesis. During the past 10 years, I have become involved in the NIH study sections as a Chartered Member within in the BDCN IRG, and participated as member of the DOD MRP panel, and the NIH CounterACT special emphasis panels.

   **The impact of this work was high as evident of over 135 citations**

   **The innovation of this work was recognized by expert editorial commentary in Epilepsy Current 2002; 2(5):146-148**

   **The impact of this work was high as evident of over 125 citations**
**The innovativeness and impact of this work was evident from its selection as top article in biology and medicine and was cited as a must read article by the Faculty of 1000 Prime [http://f1000.com/prime/8343975]**

**The innovation of this work was recognized by expert editorial commentary in Epilepsy Currents 2015; 15(2): 80–82**

B. Positions and Honors

**Positions and Employment**

1992-1993  Junior Research Fellow in Pharmacology, Panjab University, Chandigarh (India)
1994-1996  Senior Research Fellow in Pharmacology, Panjab University, Chandigarh (India)
1997-1998  Assistant Professor (Lecturer) of Pharmacology, Panjab University, Chandigarh
1998-2001  Postdoctoral Fellow, Epilepsy Research Branch, NINDS, NIH, Bethesda, MD
2002-2007  Assistant Professor of Pharmacology, North Carolina State University, Raleigh, NC
2008-2013  Associate Professor, Department of Neuroscience and Experimental Therapeutics, Texas A&M University Health Science Center, College of Medicine, Bryan, Texas.
2008-present  Faculty Member, Texas A&M Institute of Neuroscience (TAMIN), College Station, Texas.
2013-present  Professor, Department of Neuroscience and Experimental Therapeutics, Texas A&M University Health Science Center, College of Medicine, Bryan, Texas.

**NIH Study Sections and Editorial Activity**

2010-present  Scientific Reviewer, US Department of Defense Medical Research Program grant review panel.
2010-present  Member NIH Special emphasis panel – ZRG1-MDCN-50(J)– CounterACT program.
2010-2012  Chartered Member, NIH Study Section – CNNT– Clinical Neuroplasticity and Neurotransmitters.
2011-2011  Member, NIH Special emphasis panel – ZNS1 SRB-B-32 – EUREKA epilepsy grants.
2008-2010  Chartered Member, NIH Study Section – ANIE – Acute Neuronal Injury and Epilepsy.
2007-2008  Ad hoc Member NIH Study Section – CND– Clinical Neuroscience and Disease.
2007-2009  Ad hoc Member NIH Study Section – CNNT– Clinical Neuroplasticity and Neurotransmitters.
2009-present  Ad hoc Member NIH Study Section – ICP1– International and Cooperative Projects.
2009-present  Member Grant Review Panel– New Zealand Neuroscience Foundation.
2012-present  Grant Reviewer, Texas A&M—Weizmann Institute Israel Collaborative Program.
2008-2010  Executive Editor, International Journal of Pharmaceutical Sciences and Nanotechnology
2011-present  Editor-in-Chief, International Journal of Pharmaceutical Sciences and Nanotechnology
2010-present  Review Editor, Frontiers in Aging Neuroscience; Frontiers in Pharmacology
2003-present  Editorial boards/reviewer: over 20 pharmacology/neuroscience journals.

**Professional Activity and Membership**

1999-present  Member: Society for Neuroscience; International Brain Research Organization
1999-present  Member: American Epilepsy Society & Coordinator – AES’s SIG on Neuroendocrinology
1999-present  Member: American Society for Pharmacology and Experimental Therapeutics
1999-present  Member: American Association of Pharmaceutical Scientists; AAAA
2001-2002  Member, NIH Scientific Committee, Fellows Award for Research Excellence
2008-present  Scientific program/ abstract committee, American Association of Pharma Scientists
2010-present  Coordinator, American Epilepsy Society Neuroendocrinology SIG session.
2012-present  Member, The United States Pharmacopoeia (USP).
2016-present  Member, The United States Environmental Protection Agency (EPA) Science Advisory Board.

**Honors and Awards**

1992  Gold Medalist (six medals for academic excellence), Kakatiya University, Warangal, India
1992  Master’s Fellowship, University Grants Commission, New Delhi, India
1993  GP Nair Award, Indian Drug Manufacturers’ Association, Bombay, India
1994  Doctorate Fellowship, Council of Scientific and Industrial Research, New Delhi
1996  CL Malhotra Prize in Pharmacology, Physiologists and Pharmacologists of India
1997  Uvnas Prize in Pharmacology & AVTP Devi Prize in Neuroendocrinology
1998  Biographical Citation, Marque’s Who’s Who in the World, USA
C. Contribution to Science  (total publications = 160; h-index =41)

1. My initial investigations uncovered the role of neurosteroids in seizure disorders and the promise of neurosteroid therapy for epilepsy. Neurosteroids such as allopregnanolone potentiate synaptic GABA-A receptor function and also activate extrasynaptic receptors that mediate tonic currents in the brain. My publications over the past decade have shown that neurosteroids are broad-spectrum anticonvulsants and confer seizure protection in various animal models. I found key role for neurosteroids in the pathophysiology of epilepsy, especially in catamenial epilepsy. Based on this knowledge, I developed the first animal model of catamenial epilepsy and advanced it further. These models were utilized successfully for developing therapies for catamenial epilepsy. I have identified neurosteroids and their synthetic analogs as rational treatments for this condition. In 2009, we proposed a “neurosteroid replacement therapy” for treating catamenial epilepsy. A neurosteroid could be administered in a “pulse” prior to menstruation and then withdrawn or continuously administered throughout the month. The neurosteroid would be administered at low doses to avoid side effects. Ganaxolone was identified as lead neurosteroid and has been actually tested in women with catamenial epilepsy (CoCensys, Inc.). I served as the primary investigator or senior author in all these pharmacological studies.


2. In the early 2000s, I made a major discovery that neurosteroids are a better treatment option than benzodiazepines for catamenial epilepsy. However, the molecular mechanism was unknown at that time. As faculty member, I continued my scholarly innovations in this field, which are published in a set of 12 publications, with an emphasis on disease model characterization, optimization of neurosteroid treatment strategy, and molecular & electrophysiological mechanisms. As evident from the body of papers from 2001 to 2014, my team has successfully cracked the mechanism responsible for the superior neurosteroid therapeutics. This work was published recently in the prestigious *Journal of Neuroscience* in 2014, and highlighted in an editorial in *Epilepsy Currents* journal. This exciting discovery of “extrasynaptic molecular mechanism” defies the dogma of synaptic basis of neurotransmission in epilepsy. This extrasynaptic system is providing the molecular rationale for clinical studies of neurosteroid replacement therapy in women with epilepsy and catamenial epilepsy. This has opened new frontiers in the field as extrasynaptic receptors that could play key roles in other CNS conditions such migraine, neuropathic pain, sleep disorders and movement disorders.

3. A body of my translational publications reveals my contributions to diverse areas of brain research, including status epilepticus, stress, PR signaling mechanisms in the brain, and chemical neurotoxicity. The main emphasis was on the understanding of the causes of the epilepsies, epileptogenic processes with acquired forms of epilepsies, including those associated with status epilepticus and neurodegeneration. I was the first to characterize that stress induced seizure protection could be due to the adrenal-derived neurosteroid THDOC, which increases GABA-A receptor function by allosteric potentiation and direct activation. Through patch-clamp studies, I characterized how THDOC acts at the single-channel level and how these physiologic changes can mediate the effects of stress on seizure susceptibility. Of clinical significance, stress is known to trigger seizures and can exacerbate a variety of neuropsychiatric conditions. I advanced preclinical therapy development for epilepsy prevention and disease modification by identifying new strategies and interventions to prevent or reduce acute status epilepticus and brain injury. My recent studies provide compelling evidence that neurosteroids may have antiepileptogenic properties. This is a particularly significant finding in the face of current challenges in identifying new targets for developing interventions to prevent or modify epileptogenesis.


4. My recent studies are primarily focused on developing a novel anticonvulsant drug. I was among the first to demonstrate the therapeutic utility of tonic inhibition in epilepsy and related brain disorders. We targeted extrasynaptic GABA-A receptors, which generate “tonic” inhibition and do not internalize during prolonged seizures or chemical exposures, with neurosteroids and found them to be more effective treatments for status epilepticus (SE), an emergency neurological condition with prolonged seizures. Novel therapies are needed for SE resistant to current medications. Our work eventually led to developing newer compounds offering proof-of- concepts in distinct SE models. Allopregnanolone has been selected for clinical trials for refractory status epilepticus (Sage Pharma Inc.). My recent work proves tonic inhibition as a novel strategy for preventing or retarding epileptogenesis in subjects at risk. This work is actually opening new horizons outside of the epilepsy field. I was among the first academic scientists to discover the therapeutic potential of neurosteroids for organophosphate and nerve agent (‘nerve gas’) neurotoxicity— a truly colossal discovery. This work is centered on identification of effective medical therapies against chemical threat agents. The increased risk of a terrorist attack in the United States involving chemical agents has created new challenges for federal government. This work on life-saving anticonvulsants is of national importance within the biodefense field. I am testing a dual-acting neurosteroid as an effective antidote (anticonvulsant) for nerve agent intoxication. This strategy involves combating such intoxication by targeting synaptic and extrasynaptic GABA-A receptor targets using the synthetic analog ganaxolone, which has been selected as lead compound for clinical trials.


A complete list of published work can be found in PubMed:
D. Research Support

**Ongoing Support**

NIH/OD U01 NS083460-01 Reddy DS (PI) 9/1/2013 – 8/30/2018  
**Neurosteroid Treatment for Organophosphate-Intoxication**  
The long-term goals of this project are to develop a novel neurosteroid treatment for organophosphate intoxication seizures and neurotoxicity in the brain. This is part of the NIH CounterACT program.

DOD/CDMRP # EP150062 Reddy DS (PI) 9/1/2016 – 8/30/2019  
**Epigenetic Mechanisms of Posttraumatic Epilepsy**  
The major goal of this project is to investigate the alterations of the epigenetic HDAC signaling pathway as a critical pathophysiological mechanism underlying the posttraumatic epilepsy.

**Completed Support**

NIH/OD 3R21 NS076426-02S1 Reddy DS (PI) 9/1/2012 – 8/30/2013  
**Efficacy of Neurosteroid Therapy in the Soman Model (nerve agent)**  
The main goal of this Supplement award was to determine the efficacy of the neurosteroid ganaxolone against soman-induced seizures, status epilepticus and neurotoxicity.  
*Role: PI*

NIH/OD R21 NS076426-02 Reddy DS (PI) 10/1/2011 – 9/30/2014  
**A Neurosteroid-Based Novel Treatment for OP-Intoxication**  
The main goal of this CounterACT project was to test the efficacy of neurosteroid therapy for organophosphate pesticide poisoning and its chronic neurotoxic manifestations.  
*Role: PI*

NIH/NINDS R01 NS051398-05 Reddy DS (PI) 8/1/2007 – 7/31/2013  
**Progesterone Receptors and Seizure Susceptibility**  
The specific aims of the project were: (1) to investigate the role of PRs in seizure susceptibility in the hippocampus kindling model of epilepsy, and (2) to investigate the role of PR pathway in GABA-A receptor subunit expression and function in the hippocampus.  
*Role: PI*

**Tonic Inhibition Therapy for Refractory Status Epilepticus**  
The main goal of this application was to investigate the efficacy of tonic inhibition therapy for status epilepticus using neurosteroid-based mechanistic strategies.  
*Role: PI*
NAME
Cynthia Ann Riccio

POSITION TITLE
Professor

EDUCATION/TRAINING  
(Begin with baccalaureate or other initial professional education, such as nursing, and include postdoctoral training.)

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<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
<th>YEAR(s)</th>
<th>FIELD OF STUDY</th>
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<tr>
<td>University of Connecticut, Storrs, CT</td>
<td>B.A.</td>
<td>1970-1974</td>
<td>Psychology</td>
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<tr>
<td>University of Hartford, Hartford, CT</td>
<td>M.S.Ed.</td>
<td>1979-1980</td>
<td>School Psychology</td>
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<tr>
<td>University of Hartford, Hartford, CT</td>
<td>Specialist</td>
<td>1980-1982</td>
<td>School Psychology</td>
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<tr>
<td>University of Georgia, Athens, GA</td>
<td>Ph.D.</td>
<td>1990-1993</td>
<td>Educational Psychology</td>
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<tr>
<td>University of Georgia, Athens, GA</td>
<td>Postdoctoral</td>
<td>1993-1994</td>
<td>Pediatric Neuropsychology</td>
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A. Positions and Honors

Professional Experience
August 1993 - July 1994: Post-Doctoral Fellow, Center for Clinical and Developmental Neuropsychology, University of Georgia, Athens, GA
January - August 1994: Instructor, Special Education, University of Georgia.
August 1994 – Aug 1997: Assistant Professor, Educational Psychology/School Psychology Program, Professional Studies, The University of Alabama
August 1997 – present: Assistant (1997-1999) and Associate (2000- present) Professor, Department of Educational Psychology and Neuroscience Faculty, College of Education, Texas A & M University, College Station, TX.

Professional Memberships
Member of Review Boards for:
School Psychology Review, January 1996 – present
Journal of Psychoeducational Assessment, June 1996 - present
Developmental Neuropsychology, September 1997 – present
School Psychology Quarterly, January 2002- present

Member of:
American Psychological Association:
Division 16 (School Psychology): Treasurer, 2017-2020; Division 16 Fellow Nomination Committee 2015; 2016; 2017
Member: Division 40 (Neuropsychology), Division 53 (Clinical Child), Division 54 (Pediatric Psychology)
National Academy of Neuropsychology
National Association of School Psychologists
CDSPP Liaison for Futures Program – Future of School Psychology 2011-2013
SSSP Liaison for Graduate Education Workgroup 2011 - present
Society for the Study of School Psychology (President-Elect, President, Past- President (2012-2015)
Honors
2013 Outstanding Service Recognition, College of Education and Human Development, Development Council
2012 Admitted to Academy of Board Certified School Psychologists (American Board of Professional Psychology)
    Diplomate Status, American Board of Pediatric Neuropsychology (ABPdN)
2010 Elected to Fellow, American Psychological Association, Division 16
2009 Elected to Fellow, National Academy of Neuropsychology, New Orleans, LA

B. Selected Peer-Reviewed Publications (last 5 years)


C. Research Support
**BIOGRAPHICL SKETCH**

Dr. Bruce B. Riley 

Professor, Biology Department 
Texas A&M University

**EDUCATION**

University of Colorado-Boulder  B. A.  1982  Biology  
University of Wisconsin-Madison  Ph.D.  1990  Mol/Dev Biology  
University of Wisconsin-Madison  postdoc  1990-1992  Chick Development  

A. Personal Statement

I have worked in the field of zebrafish developmental genetics for over 25 years, with 21 years as a PI running my own lab. Research in my lab has focused primarily on development of the zebrafish inner ear, with a number of seminal contributions to the field: We were the first group to demonstrate that Fgf is the primary factor responsible of otic placode induction; we were amongst the first to directly test the role of Delta-Notch signaling in patterning hair cells and support cells; we demonstrated that Atoh1 acts akin to proneural genes, establishing the entire pro-sensory equivalence group rather than simply promoting hair cell differentiation; we were the first to demonstrate that Sox2 potentiates the pro-sensory activity of Atoh1; we showed that Sox2 is essential for hair cell regeneration (to our knowledge the first such gene identified in the inner ear); and we identified for the first time the molecular mechanism by which SAG neuroblasts delaminate from the otic vesicle.

B. Positions and Honors

**Positions**

**Sept. 2007 to present**: Professor, Biology Department, Texas A&M University.  
**Sept. 2000 to Aug. 2007**: Associate professor, Biology Department, Texas A&M University.  
**Aug. 1995 to Aug. 2000**: Assistant professor, Biology Department, Texas A&M University.  
**May 1992 to Aug. 1995**: Postdoctoral research with David Grunwald, University of Utah.  
**Sept. 1990 to Apr. 1992**: Postdoctoral research with John Fallon and Brad Olwin, University of Wisconsin-Madison.  
**Jan. 1985 to Aug. 1990**: Graduate research with Steve Barclay, University of Wisconsin-Madison.

**Honors**

B.A. with distinction, University of Colorado, 1982.  
Postdoctoral Fellowship, ACS, University of Utah, 1994-1996.  
NIH awardee, 1998-present.
C. Selected Peer-Reviewed Publications


D. Research Support

Current Support

National Institutes of Health, NIDCD R01 DC003806 03/01/13-02/28/18
“Genetic Analysis of Inner Ear Development in Zebrafish”. Role: PI

Completed

National Institutes of Health, NIDCD R01 DC003806 03/01/08-02/28/13
“Genetic Analysis of Inner Ear Development in Zebrafish”. Role: PI

National Institutes of Health, NIDCD R01 DC003806 03/01/03-02/28/08
“Genetic Analysis of Inner Ear Development in Zebrafish”. Role: PI

National Institutes of Health, NIDCD R01 DC003806 05/01/98-02/28/03
“Genetic Analysis of Inner Ear Development in Zebrafish”. Role: PI
NAME: Rimer, Mendell

POSITION TITLE: Associate Professor

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

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.)

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<th>DEGREE (if applicable)</th>
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<th>FIELD OF STUDY</th>
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<td>Universidad de Los Andes, Mérida, Venezuela</td>
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<td>12/86</td>
<td>Biology</td>
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<td>University of Maryland at Baltimore, MD</td>
<td>Ph.D.</td>
<td>05/93</td>
<td>Molecular &amp; Cell Biology</td>
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<td>Stanford University, Stanford, CA</td>
<td>Postdoctoral</td>
<td>07/97</td>
<td>Neurobiology</td>
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<td>New Mexico State Univ., Las Cruces, NM</td>
<td>Postdoctoral</td>
<td>12/98</td>
<td>Bioinformatics</td>
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<tr>
<td>New York University, New York, NY</td>
<td>Postdoctoral</td>
<td>07/00</td>
<td>Genetics, Neurobiology</td>
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A. Personal Statement

The long-term goal of my research program is to contribute to the understanding of molecular and cellular mechanisms of synapse formation, normal maintenance and pathology. Because of its simplicity and experimental accessibility, I have used the vertebrate neuromuscular junction (NMJ) as model synapse. Throughout my career, I have made important and valued contributions to the field, specifically to the molecular and cell biology of acetylcholinesterase, agrin and the neuregulins. More recently, my lab has been studying the modulation of agrin signaling by the ERK1/2 MAP kinases (Wang et al., 2016; Seaberg et al., 2015) and murine models of spinal muscular atrophy (SMA) (Paez-Colasante et al., 2013; Lee et al., 2011), largely attracted by the NMJ abnormalities characteristic of this disease.


B. Positions and Honors

Positions and Employment
1998 Instructor, Department of Biology, New Mexico State University.
C. Contribution to Science

1. Role of agrin in vivo.

I joined the lab of U.J. McMahan at Stanford as a postdoctoral fellow following my PhD work on acetylcholinesterase under Bill Randall at the University of Maryland at Baltimore. The McMahan lab had purified agrin as an extracellular matrix protein capable of inducing AChR clusters on cultured myotubes and had cloned its gene in Torpedo and chick. The important question then was to determine the role of agrin in NMJ formation in vivo. While others took the loss-of-function approach, Terje Lømo, Ilana Cohen and I in the McMahan lab injected cDNA for neural agrin into the extrajunctional region of rat soleus muscles and showed that agrin could induce in vivo AChRs clusters that accumulated many –if not all– of the components of the native postsynaptic apparatus (Cohen et al., 1997). In addition, at the time the growth factor neuregulin 1 (NRG1), but not agrin, was thought as the nerve-derived factor responsible for synapse-specific expression of postsynaptic genes. NRG1 was particularly active in stimulating transcription of Chrne, the gene encoding the AChRε subunit and was believed to be expressed by motoneurons but not by muscle fibers. We showed that ectopic agrin was sufficient to induce AChR clusters containing AChRε (Rimer et al., 1997) and that these clusters also had NRG1 and its receptors, ErbB2 and ErbB3 (Rimer et al., 1998). Thus agrin also appeared sufficient in vivo to induce Chrne expression and seemed to be doing so by aggregating muscle NRG1 and its receptors. Hans Brenner and colleagues at the University of Basel, in experiments that were carried out almost
simultaneously and independently, reached similar results and conclusions. This work had a lot of impact at the time and has influenced the work of many others in the field since.


2. Role of neuregulin in vivo.

Once on my own, I decided to investigate the role of NRGs at the NMJ and to follow on the results of my postdoctoral work. It was important then to test the role of NRG1 genetically. While others used a conditional loss-of-function approach, I used a gain-of-function strategy by inducibly expressing in muscle fibers a constitutively active form of ErbB2 (CAErbB2) to ask in vivo what NRG signaling could do in muscle. To accomplish this, my lab generated a novel mouse line, in which the reverse-tetracycline transactivator (rtTA) was driven by a muscle-fiber specific promoter (MDAF-rtTA). Crossing this line to mice harboring a CAErbB2 whose expression could be induced by rtTA, allowed us spatial and temporal control of CAErbB2 expression. Unexpectedly we found that muscle expression of CAErbB2 during embryonic development led to synaptic disassembly, extensive axonal sprouting and perinatal lethality. Further experiments suggested that activation of NRG signaling in muscle by CAErbB2 interfered with agrin signaling (Ponomareva et al., 2006). Our results, together with data from Hans Brenner’s and Steve Burden’s groups showing that conditional deletion of the NRG receptors in muscle, or of *Nrg1* in motoneurons, muscle or both failed to alter synapse-specific *AChR* expression or synaptic morphology, changed the then prevalent view of NRG1 in the field from an essential factor in synaptogenesis to a modulator of the process. Our approach in muscle inspired Chris Hayworth in Wes Thompson’s lab to express inducible CAErbB2 in adult Schwann cells and demonstrate that turning on NRG signaling mimicked the effects of denervation in the synaptic glia (Hayworth et al., 2006).


3. Applications of a unique line of mice generated in my lab.

In the early-to-mid 2000’s a myogenic model of neuromuscular synapse formation arose from results in zebrafish and mouse that showed that AChR clusters could be found in the future endplate region in vivo prior to nerve-muscle contact, forming what is now known as the prepatter. At the time, these results were controversial and challenged the classical neurocentric model in which the nerve induced, via agrin, the de novo formation of the postsynaptic apparatus. We took advantage of the reversible nature of the rtTA-mediated CAErbB2 expression in our mice to test these models by transient induction of CAErbB2 at midgestation to eliminate the central prepatter and probing if and where synapses reformed after birth. Our results seemed to support the myogenic model in which the muscle fiber instructs the nerve where along its length to engage in synaptogenesis (Vock et al., 2008).

The MDAF-rtTA mice produced in my lab made it possible to generate a mouse model for the neuromuscular disease myotonic dystrophy type I (DM1), by inducible and selective overexpression of CUG-binding protein 1 (CUGBP1) in skeletal muscle fibers (Ward et al., 2010). DM1 patients harbor a mutant allele of the DM protein kinase gene with a CTG repeat expansion in the 3'-untranslated region. RNA transcribed from the expanded allele has the expanded CUG repeats and leads to the nuclear removal of Muscleblind-like 1 protein and to increased levels of CUGBP1. The specific contribution of the increased CUGBP1 to the DM1 skeletal muscle pathology was unknown before this work. Adult mouse skeletal muscle overexpressing...
CUGBP1 recapitulated molecular and physiological defects of DM1 tissue, suggesting that CUGBP1 has a major role in DM1 skeletal muscle pathogenesis.


4. Major contribution outside my field.

In 2002, Kari Steffansson and colleagues published a seminal study linking NRG1 to susceptibility to schizophrenia based on human gene association studies and on the phenotypes of mice hypomorph for all Nrg1 isoforms. Because of our work with NRG1 at the NMJ, we had acquired Nrg1 mice hypomorph for a set of variants known as the Ig-domain isoforms. We collaborated with a well-known behavioral neuroscience lab and showed that these mice had phenotypes consistent with NRG1 conferring susceptibility to schizophrenia (Rimer et al., 2005). This work was one of the earliest studies using Nrg1/ErbB receptor mutant mice that further examined a possible link to schizophrenia and has become widely cited in that field.


Complete List of Published Work in My Bibliography:

D. Research Support

Ongoing

R21NS101477 M. Rimer (PI) 03/01/17-02/28/19
NIH
Isolation of Terminal Schwann Cells by Fluorescence-Activated Cell Sorting
The goal of this project is to identify genes specifically expressed by terminal Schwann cells that can be used as tools to selectively manipulate these cells genetically.
Role: PI

PO#100935375 C-P. Ko (PI) 11/03/15-11/02/16
University of Southern California / SMA Foundation
Sprouting Capacity Upon Partial/Complete Denervation in an Intermediate SMA Mouse Model
The goal of the project is to evaluate axonal sprouting ability in a SMA mouse model whose lifespan is extended pharmacologically.
Role: Sub-award PI

Completed

R21NS077177 M. Rimer (PI) 09/01/12-07/31/15
NIH
Role of ERK1/2 in Neuromuscular Synapses and Myofiber Development In Vivo
The goal of this project was to study in vivo the role of myofiber–derived ERK1/2 MAP kinases in the formation and maintenance of neuromuscular synapses and their attending skeletal muscle fibers.
Role: PI
Motoneuron-selective Rescue of SMA Model Mice
The goal of this project was to establish how much of the disease phenotype in mouse models of spinal muscular atrophy (SMA) is contributed by motoneurons.
Role: PI
BIOGRAPHICAL SKETCH

GIL G. ROSENTHAL (PI)

(a) Professional preparation

Harvard University  Cambridge, MA  Biology  A. B. magna cum laude, 1993
University of Texas  Austin, TX  Zoology  Ph.D., 2000
University of California  San Diego, CA  Ecology, Behavior, & Evolution  2000-2002

(b) Appointments

2013-date  Professor, Department of Biology, Texas A&M University
2014-date  Past chair and graduate admissions chair, Faculty of Ecology & Evolutionary Biology
2014-date  President, CICHAZ, A. C.
2006-date  Faculty of Genetics

(c) Publications

i. Five publications most closely related to the proposed project


ii. Other significant publications


(d) Synergistic activities
- Developing the Centro de Investigaciones Científicas de las Huastecas (CICHAZ) as a regional field station for multidisciplinary basic and applied research;
- organizing annual Calnali Day of Science and Sustainable Development in 2014, 2015, 2016 and developing K-12 outreach program;
- chairing TAMU’s Faculty of Ecology and Evolutionary Biology and developing a new PhD program;
- co-supervising three Ph.D. students, a Master’s student and two undergraduate students in Latin America;
Gül A. Russell, Ph.D.

Professor
Department of Humanities in Medicine
8441 State Hwy 47, Suite 1400
Bryan, TX 77807

Phone: 979-436-0523
russell@medicine.tamhsc.edu | mailto:russell@medicine.tamhsc.edu

Education and Post-Graduate Training

Education
B.A. (1958) English and Art History, Lindenwood College, St. Charles, Missouri
Ph.D. (1962), John H. Edwards Fellow, Comparative Studies (History of Ideas under H.J. Muller; History and Philosophy of Science under N.R. Hanson), Indiana University, Bloomington, Indiana.

Postdoctoral Training

Selected Publications


Invited Research Presentations

C.N.R.S., Paris; University of Cambridge (Corpus Christi); Delft (Trinity College), Edinburgh; King’s College, London; Erasmus University Medical Center, Rotterdam; Free University, Berlin; Dibner Institute/Harvard; Herzog August Bibliothek, Wolfenbüttel, Germany; Oud-Turnhout, Belgium; Royal College of Physicians, Dublin; University of Padua, Italy; University of Istanbul Medical School, Turkey; University of Brunei, Bandar Seri Begawan, Brunei; National Institute of Science, Technology and Development Studies, Delhi, India; The Academy of Science, Kuala Lumpur, Malaysia; Carthage, Tunisia; Baylor College of Medicine, Houston; University of Oklahoma; Kluge Center, Library of Congress, Washington, D.C. Keynote: University of Calgary, Canada; Clifford Rose Memorial Lecture, Brussels, Belgium. On teaching: history of science at higher institutions: UNESCO Headquarters, Paris, France; on education physicians, Ohio University Medical School, Columbus, Ohio; Harvard Medical School and Museum of Art.

International Service

- She is also an affiliate of the Dibner Institute at the Massachusetts Institute of Technology (MIT), College of Liberal Arts (TAMU).
- President of the Phi Kappa Phi Honor Society, TAMU Chapter (2015-2016).
- President of the International Society for the History of the Neurosciences (2017-).


NAME: Erin M. Scott, VMD, DACVO

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

POSITION TITLE: Clinical 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.)

<table>
<thead>
<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
<th>Completion Date MM/YYYY</th>
<th>FIELD OF STUDY</th>
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</thead>
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<tr>
<td>Georgetown University, Washington, DC</td>
<td>BS</td>
<td>05/2004</td>
<td>Biology</td>
</tr>
<tr>
<td>University of Pennsylvania, Philadelphia, PA</td>
<td>VMD</td>
<td>05/2010</td>
<td>Veterinary Medicine</td>
</tr>
<tr>
<td>Louisiana State University, Baton Rouge, LA</td>
<td>Internship</td>
<td>06/2011</td>
<td>Small Animal Medicine and Surgery</td>
</tr>
<tr>
<td>University of Wisconsin-Madison, Madison, WI</td>
<td>Fellowship</td>
<td>07/2012</td>
<td>Comparative Ocular Pathology</td>
</tr>
<tr>
<td>University of Wisconsin-Madison, Madison WI</td>
<td>Residency</td>
<td>07/2015</td>
<td>Comparative Ophthalmology</td>
</tr>
<tr>
<td>American College of Veterinary Ophthalmologists</td>
<td>Diplomate</td>
<td>08/2015</td>
<td>Ophthalmology</td>
</tr>
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</table>

A. Personal Statement

I am most interested in developing a career path in translational research, allowing me to bridge basic science findings to practical applications in the clinic in order to enhance the care and well being of veterinary patients. My professional interests include diseases of the retina and pathogenesis of glaucoma, with a strong emphasis in ocular pathology. By linking the clinical features of ocular disease with their associated histopathologic changes, I can provide a unique perspective in the interpretation and understanding of ocular disease processes in veterinary patients and their relevance to comparable human disorders.

B. Positions and Honors

Positions and Employment

09-2015 – present  Department of Small Animal Clinical Sciences  Clinical Assistant Professor
Texas A&M University, College Station, TX

Honors

2015  Dr. Paul Dice Memorial Award for Best Case Report
American College of Veterinary Ophthalmologists

2015  Small Animal Resident of the Year Award
University of Wisconsin-Madison, Madison, WI

2014  Zoetis Award for Research Excellence by a House Officer, First Place
University of Wisconsin-Madison, Madison, WI

2014  Vision for Animals Foundation (VAF) Grant Award
American College of Veterinary Ophthalmologists
C. Contributions to Science

1. As a veterinary student, I studied a canine model of retinal degeneration that was later discovered to be the genetic homologue of a severe childhood-onset blinding disease in people, known as Leber congenital amaurosis (LCA). I was influential in characterizing the retinal pathology associated with this disease in dogs. This allowed the identification of critical time points in the pathogenesis of the disease, which will help to define a potential time window for testing novel therapies for translation to human patients. Incidentally, while studying this retinal disease, I noticed a discreet region within the temporal canine retina that contained multiple rows of cones. This observation transitioned into an interesting study where we discovered a fovea-like area in the canine retina that has not been previously described in any non-primate mammalian species.


2. As a fellow in comparative ocular pathology, I investigated the early pathological changes to the retina and optic nerve in dogs with primary angle-closure glaucoma (PACG). Canine PACG is the most common form of spontaneous primary glaucoma in dogs, and a leading cause of irreversible blindness. We found the pathogenesis of optic nerve and retinal degeneration in dogs with PACG varies considerably from other forms of glaucoma in other species. Characterizing these pathologic changes may enable the eventual direction of neuroprotective therapy to help prevent the rapid and irreversible cascade of events that ultimately lead to blindness.


3. As a resident in comparative ophthalmology, I diagnosed several highly malignant orbital tumors in juvenile dogs with an uncommon neoplasm called rhabdomyosarcoma. I performed a retrospective study in order to learn more about this disease, and found it closely parallels what is described in people. We discovered a duality in biologic behavior that may reflect differences in tissue of origin between juvenile onset tumors (which are highly malignant) and adult onset tumors (which are treatable). We also found a potential treatment approach that may prolong survival and quality of life in our veterinary patients with juvenile-onset tumors.


Complete List of Published Work in My Bibliography:

D. Research Support

Ongoing Research Support:
Effectiveness of a retrobulbar injection of 0.75% ropivacaine for postoperative analgesia following eye enucleation in dogs. Scott, EM (PI). GINN Research Fund ($8252.25), funded August 2016
Goal: To assess the additive pain-relieving effect of a local anesthetic, ropivacaine, in dogs undergoing an enucleation procedure.
Role: PI

Completed Research Support in the Last Three Years:

Active, latent and total TGF-β2 concentrations in the aqueous humor of dogs with open angle glaucoma. Scott, EM (PI). Vision For Animals Foundation Resident Research Fund ($4145.00), funded February 2013
Goal: To determine the effect of the ADAMTS10 mutation, age, and intraocular pressure on TGF-β2 aqueous humor concentrations in a canine model of inherited open angle glaucoma.
Role: PI
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: Lee A. Shapiro, Ph.D.

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

POSITION TITLE: Associate 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.)

<table>
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<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
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<th>FIELD OF STUDY</th>
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<tr>
<td>University of Colorado, Boulder</td>
<td>B.A.</td>
<td>12/1995</td>
<td>Psychology</td>
</tr>
<tr>
<td>State University of New York, Stony Brook</td>
<td>M.S.</td>
<td>06/2000</td>
<td>Biopsychology</td>
</tr>
<tr>
<td>State University of New York, Stony Brook</td>
<td>Ph.D.</td>
<td>12/2004</td>
<td>Neuroscience</td>
</tr>
<tr>
<td>University of California, Irvine</td>
<td>Post-Doc</td>
<td>12/2007</td>
<td>Anatomy &amp; Neurobiology</td>
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A. Personal Statement

The goal of the proposed research is elucidate the vagal control of glucose metabolism, and the circuits involved in this function. This project was born as a result of my collaboration with Dr. Harald Stauss on my currently funded DoD grant. In this grant, Dr. Stauss manufactures the vagus nerve stimulators that we implant into mice, in order to determine if this will improve gulf war illness symptomology. As part of this grant, Dr. Stauss flew to my lab in Temple, TX, where he instructed us on the proper implantation and operation of the vagus nerve stimulators. While I had a number of discussions with Dr. Stauss on the phone, this was my first time meeting him in person. During this visit, Dr. Stauss presented a seminar, and had discussions with a number of investigators. He and I had extensive conversations on each other’s work, and we quickly realized that a collaboration could greatly facilitate his immediate line of research, as well as a growing area of interest in my lab; the role of the vagus nerve in linking neuronal and peripheral responses. From these initial discussions, we have had countless more, which have ultimately evolved into this proposal.

My work is increasingly recognized in the fields of neuroanatomy, neuropathology, neuroinflammation and systemic pathology observed in different animal models. One focus of my lab is on seizures, epilepsy, and brain injury models, the studies of which have yielded a number of neuroanatomical contributions. Moreover, I have published several articles that are exclusively neuroanatomical, some of which involved using a number of neuroanatomical tracing techniques. I have performed pioneering work examining the aberrant integration of newly generated neurons that contribute to dysfunctional hippocampal circuits. A series of experiments showed that in a chemotoxic epilepsy model, basal dendrites from newborn neurons sprout along the processes of hypertrophied radial-glial-like astrocytes. These basal dendrites are targeted for aberrant synaptogenesis, forming a circuit that enhances excitability in the hippocampus and may contribute to hippocampal dysfunction. My work also includes a focus on peripheral contributions to traumatic brain injury (TBI) and other neurological disorders. Most recently, I have published an important paper, with our Co-Investigator, Sharon DeMorrow, highlighting changes in the liver following a traumatic insult to the brain (Nizamutdinov et al. Scientific Reports, 2017). Dr. DeMorrow and I have a number of other collaborations ongoing, all of which focus on liver/nervous system interactions. Moreover, I also teach the histology labs, and some of the lectures to the medical students. These labs use human tissue sections, and I have extensive experience in working with human liver samples, in addition to my experience working with rodent livers.
through my collaboration with Dr. DeMorrow. It should further be noted that as a member of the Department of Surgery, my Colleague, Dr. Lairmore will consult on this proposal. Dr. Lairmore has indicated his intentions to not only provide human specimens for the grant, but to also work with me in analyzing and interpreting the samples. The work described in the current research plan builds upon the methodological and conceptual foundations of previous and current work in my lab examining vagal contributions to neurological dysfunction, and the possibility of modulating the vagus nerve for therapeutic effect.

B. Positions and Honors

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<tr>
<td>01/2001-05/2003</td>
<td>Lecturer, Department of Biology, SUNY Stony Brook, Stony Brook, NY</td>
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<td>12/2007- 08/2015</td>
<td>Assistant Professor, Department of Surgery, Texas A&amp;M Univ. Temple, TX, Department of Neurosurgery, Scott and White Hospital and the Central Texas Veterans Health Care System (CTVHCS).</td>
<td></td>
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Effective 09/2015 Associate Professor, Department of Surgery, Texas A&M Univ. Temple, TX, Department of Neurosurgery, Scott and White Hospital and the Central Texas Veterans Health Care System (CTVHCS).

C. Contribution to Science

My early publications directly addressed adult hippocampal neurogenesis in normal rats and rats exposed to the chemoconvulsant, pilocarpine. The results reported in these publications showed that seizures alter hippocampal neurogenesis. Two consequences of these alterations that we reported were the appearance of hilar basal dendrites extending deep into the dentate gyrus hilar region, and the targeting of these hilar basal dendrites for aberrant synaptogenesis. We revealed these synapses on these hilar basal dendrites to be mossy fiber synapses, indicating that they emanated from dentate gyrus granule cells. As such, newly born granule cells were receiving axonal connections from granule cells, constituting a recurrent excitatory circuitry. These findings spawned an entire field of neurogenesis research in the epilepsy area designed to ameliorate the changes to seizure-induced aberrant neurogenesis. I served as the primary investigator in all of these studies, of which I have provided a sampling of 3 highly-cited manuscripts.


The role of GFAP-labeled astrocytes in neurogenesis and contributions to post-brain insult to the aberrant growth of hilar basal dendrites, neuroinflammation and epileptogenic progression. As an extension of my Ph.D work that was focused on neuroinflammation, I incorporated this “gliocentric” context into a parallel series of experiments designed to understand the roles of GFAP-expressing astrocytes in the pathogenesis linked to seizures, epilepsy and brain injury. Initially, these studies were focused on understanding the relationship with the newborn dentate granule cells, because the radial-glial like astrocytes in the dentate gyrus are the precursors for many of these newly born granule cells. We subsequently discovered that the relationship between the glial mother and the newborn neurons were altered following pilocarpine-induced seizures and that this alteration provided an anatomical and molecular (Via CCR2 overexpression) substrate for the aberrant growth of basal dendrites from newborn neurons into the hilus. I served as the primary investigator or senior investigator in all of these studies, of which I have provided a sampling of 3 highly-cited manuscripts and a very recent publication.


Foresti ML, Arisi GM, Katki K, Montañez A, Sanchez RM, Shapiro LA. Chemokine CCL2 and its receptor CCR2 are increased in the hippocampus following pilocarpine-induced status epilepticus. J Neuroinflammation. 2009 Dec 24;6:40. PMCID: PMC2804573.


My most recent work has been involved in exploring the role of peripheral infiltration, immune cell activation and neuroinflammation in traumatic brain injury, post-traumatic epilepsy, and other types of seizure-inducing injury to the brain. We initially demonstrated, using a novel multiplex technique that we perfected in brain homogenates, TBI-induced alterations that have unique temporal and spatial alterations. This work involved establishing a novel mouse model of TBI in my lab. These studies led to an examination of systemic factors that might contribute to post-traumatic syndromes, including post-traumatic epilepsy. At the end of 2014, I was the senior author on a manuscript that has already achieved the “highly-viewed” designation from the prestigious on-line portion of Acta Neuropathologica. In this manuscript, we demonstrate that blocking components of the peripheral and/or adaptive immune response is anti-inflammatory and neuroprotective after a TBI. I served as the primary investigator or senior investigator in all of these studies, of which I have provided a sampling of 3 highly-cited manuscripts, and the aforementioned manuscript with Dr. DeMorrow.


D. Current Research Support

**Wounded Warriors**: Shapiro (CO-I) 01/2013 – 12/2017; TDC: $473,765. This project is aimed at examining immunological (CNS and peripheral) contributions to blast- and blast-related traumatic brain injuries. We are examining neuroinflammation in the molecular, neuroanatomical, and cellular context. We are also examining neuronal structure, using immuhistochemical analysis

**Department of Defense GWI grant** Shapiro (PI) 10/2015 – 09/2018; TDC: $647,900. Vagus nerve stimulation as a treatment for Gulf War Illness. The goal of this proposal is to assess the efficacy and mechanisms of vagus nerve stimulation to reduce central nervous system inflammation, astrocyte and microglial activation, and to assess long-term behavioral outcomes in response to vagus nerve treatment.

**Citizens United for Research in Epilepsy (CURE)**: Shapiro (Co-PI) 10/2015 – 09/2017; TDC: $250,000.00 “Influence of antigen processing and presentation on the development of post-traumatic epilepsy.” The goal of this grant is to assess the influence of antigen processing and presentation on post-traumatic epilepsy. It is important to note that there is no overlap between the CURE grant and the current proposal.
BIOGRAPHICAL SKETCH

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

NAME
Charles H. Shea

POSITION TITLE
Professor

eRA COMMONS USER NAME

EDUCATION/TRAINING  (Begin with baccalaureate or other initial professional education, such as nursing, and include postdoctoral training.)

<table>
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<th>INSTITUTION AND LOCATION</th>
<th>DEGREE</th>
<th>YEAR(s)</th>
<th>FIELD OF STUDY</th>
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<tr>
<td>Virginia Polytechnic Institute and State U.</td>
<td>B.S.</td>
<td>1970</td>
<td>Hlth and PE</td>
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<td>Virginia Polytechnic Institute and State U.</td>
<td>M.S.</td>
<td>1975</td>
<td>Hlth and PE</td>
</tr>
<tr>
<td>Virginia Polytechnic Institute and State U.</td>
<td>Ph.D.</td>
<td>1978</td>
<td>Ed. Research and Eval</td>
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A. Positions and Honors.

Other Experience (Titles, Professional Memberships, and Offices Held)
- Associate Dean for Graduate Studies and Faculty Development, College of Education, Texas A&M University, (1993-1995).

B. Courses Taught (Last 5-Years)
- KINE 406 Motor learning and skill performance
- KINE 690 Theory of Research

C. Selected peer-reviewed publications and performances (Selected from last 5 years).

Book Chapters

Peer-Reviewed Articles


BIOGRAPHICAL SKETCH

NAME: Sitcheran, Raquel

POSITION TITLE: Assistant Professor of Molecular & Cellular Medicine

EDUCATION/TRAINING:

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<th>FIELD OF STUDY</th>
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<tr>
<td>Columbia University, New York, NY</td>
<td>A.B.</td>
<td>05/1992</td>
<td>Spanish, Biology</td>
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<tr>
<td>University of California, San Francisco, CA</td>
<td>Ph.D.</td>
<td>06/2000</td>
<td>Physiology</td>
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<tr>
<td>University of North Carolina, Chapel Hill, CA</td>
<td>Postdoctoral</td>
<td>08/2009</td>
<td>Molecular/Cell Biology</td>
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A. Personal Statement

My research program investigates the molecular underpinnings of brain tumor cell invasion and pathogenesis. We have established a fundamental role for noncanonical NF-κB signaling, driven by NF-κB-inducing kinase (NIK), in promoting the aggressive invasiveness of high-grade gliomas, and other cancers. Using interdisciplinary approaches in quantitative live cell imaging, fluorescence microscopy, ex-vivo assays, and orthotopic mouse tumor models, we have established novel roles for NIK in the regulation of mitochondrial dynamics, tumor cell motility/invasion and cancer cell metabolism. For example, we have discovered that a discrete pool of NIK is localized to mitochondria where it regulates mitochondria dynamics independently of downstream IKK/NF-κB pathways. Our findings have elucidated novel paradigms for NIK function beyond the immune system. Our research efforts employ proteomic and metabolomic approaches to study NIK regulation of mitochondrial functions in cancer cells, as well as normal, pluripotent cells. Lastly, we are investigating roles for NIK in development using several model systems, including mouse, drosophila and zebrafish.

My experience as a parent who has lost a young child to an aggressive brainstem glioma has left me with a profound personal motivation to decipher the molecular underpinnings of brain tumor cell invasion and pathogenesis. The strong foundation from my training as a graduate student and postdoctoral fellow in genetics, signaling and transcription regulation, has given me the necessary expertise to be successful as a group leader in an academic setting. Indeed, I have successfully secured funding from a number of sources, including an NIH R01 award, a High Risk/High Impact award from the Cancer Prevention and Research Institute of Texas (CPRIT), and a Seed Grant from the Texas Brain & Spine Institute, and published four significant research papers as corresponding author (see “Contributions to Science” below). I am training, or have trained, five graduate students and two postdoctoral fellows, and several undergraduate students, overseeing their research, as well as conducting my own experiments. My motivation to lead my research program and advance our understanding of aggressive, invasive brain tumors has never been higher.

B. Positions and Honors

Positions and Employment

1994-2000 Graduate Student, Biomedical Sciences Graduate Program, University of California, San Francisco, CA
2000-2006 Postdoctoral Fellow, Lineberger Comprehensive Cancer Center, University of North Carolina, Chapel Hill, NC
2006-2009 Research Associate, Lineberger Comprehensive Cancer Center and Department of Cell Biology, University of North Carolina, Chapel Hill, NC
Assistant Professor, Department of Molecular & Cellular Medicine, Texas A&M University Health Science Center, College Station, TX

Other Professional Experience, Service and Memberships

2000 - 2002  Co-founder and Chair, UNC Postdoc Association, UNC Chapel Hill
2008  Member, American Society for Cell Biology
2011-  Founder and Faculty Advisor, Student-Postdoc Seminar Series, Department of Molecular & Cellular Medicine, Texas A&M Health Science Center (TAMHSC)
2012 - 2015  Member, Admissions Committee, Interdisciplinary Graduate Program in Genetics, Texas A&M University
2012-  Member, MD/PhD Steering Committee, College of Medicine, TAMHSC
2014-  Research Faculty Member, Texas Brain and Spine Institute, Bryan TX
2014-  Member, Society for Neuro-Oncology
2014-  Member, Texas Brain and Spine Institute (TBSI), Bryan TX
2015-  Professional Member, American Heart Association
2015-  Member, Genetics Interdisciplinary Program Executive Committee, Texas A&M University
2015-  Early Career Reviewer, Center for Scientific Review, National Institutes of Health
2015-  Faculty Member, Texas A&M Institute for Neuroscience, Texas A&M University
2016-  Chair, Admissions Committee, Interdisciplinary Graduate Program in Genetics, Texas A&M University

Honors & Awards

1995 - 1996  Eugene Cota Robles Graduate Student Award, University of California at San Francisco, CA
2000 - 2001  Postdoctoral Training Fellow, UNC Lineberger Comprehensive Cancer Center, University of North Carolina, Chapel Hill, NC
2002  Scholarship Award for “NF-κB: Bench to Bedside” Keystone Symposia
2002 - 2004  Postdoctoral Fellowship, Cancer Research Institute
2004  Scholarship Award for “NF-κB: Biology and Pathology” Keystone Symposia
2004  Minority Affairs Committee Travel and Poster Presentation Award, American Society for Cell Biology Annual Meeting, Washington, DC
2005  Joseph S. Pagano Award, Lineberger Comprehensive Cancer Center, University of North Carolina, Chapel Hill, NC

C. Contributions to Science

a. Establishing a key role for the noncanonical NF-κB regulatory pathway in glioma pathogenesis:
   The noncanonical NF-κB signaling pathway controls activation of RelB-mediated transcription and is typically associated with specific immune responses. A significant area of my research program has been to establish the importance of noncanonical NF-κB proteins in high-grade glioma, the most common and deadliest of adult central nervous system (CNS) malignancies. We have shown that RelB activity predominates in mesenchymal glioma, an aggressive, invasive subtype where it promotes epithelial-to-mesenchymal transition (EMT), cell motility and invasion. Consistently, high RelB expression is a strong prognostic indicator for shorter time to disease progression and decreased patient survival. Notably, activation of NIK (NF-κB-inducing kinase/MAP3K14), a key upstream regulator of noncanonical NF-κB-signaling, and RelB-mediated invasion is induced by extracellular signals in glioma cells, regardless of their RelB expression levels, suggesting that the tumor microenvironment...
plays a key role in increasing RelB activation in non-mesenchymal tumors. These findings are significant because oncogenic, pro-invasive roles for RelB and NIK in tumors of the CNS were previously not appreciated, and they suggest that targeting noncanonical NF-κB activation, alone or in combination with inhibition of canonical NF-κB signaling, will be more efficacious for attenuation of tumor cell invasion in a broad range of glioma subtypes.


b. **Elucidating the molecular mechanism underlying the ability of NIK to promote cell invasion:** My lab has demonstrated that NIK plays an important role in promoting glioma cell invasion through both NF-κB-dependent and NF-κB-independent functions. We found that NIK can increase cell invasion by regulating the activity of the basement membrane degrading enzyme MT1-MMP/MMP14 in fibroblasts and glioma cells. NIK increases cell membrane localization and enzymatic activity of the basement membrane degrading enzyme MT1-MMP, which does not require transcriptional regulation of MT1-MMP gene expression and occurs in the absence of NF-κB proteins (canonical RelA/p65, cRel, or noncanonical RelB), highlighting a novel function for NIK to control the activity of a critical enzyme in an NF-κB-independent manner. Indeed, *NF-κB-independent functions for NIK in tumor pathogenesis have not been previously described*. These studies establish a critical need to identify novel NIK substrates, interacting proteins and downstream target genes, which my lab is actively pursuing.

We have also discovered a distinct mode of NIK regulation at the mRNA level. Specifically, we have found that NIK mRNA is induced by pro-invasive signals (e.g. TWEAK), and is also upregulated in cells that are actively undergoing invasion. We have identified a TWEAK-induced c-IAP1/E2F regulatory complex at the NIK promoter, which mediates upregulation of NIK. To our knowledge, signal-specific and invasion-induced control of NIK mRNA expression is a novel finding that challenges existing dogma that NIK is primarily regulated at the posttranslational level. We hypothesize that dynamic transcriptional upregulation of NIK is critical to propagate noncanonical NF-κB signaling in a subpopulation of pioneer cells that initiate and propagate invasion. The ability to block upregulation of NIK in these pioneer invading cells may offer a therapeutic strategy that will attenuate acquisition of invasive potential during tumor progression.


c. **NIK regulates mitochondria dynamics:** Although NIK is expressed in the adult brain, functions for NIK in the central nervous system (CNS) are poorly understood. We have made the novel finding that NIK is localized to mitochondria in high-grade gliomas, as well as normal primary astrocytes. Moreover, loss of NIK significantly impacts mitochondrial abundance and morphology in glioma cells. These findings have led us to investigate the hypothesis that NIK regulates mitochondrial dynamics and mitochondrial subcellular distribution to supply the energy requirements for the altered cell morphology associated with glioma cell migration and invasion. Notably, NIK regulates mitochondria dynamics and promotes invasion in the absence of its known downstream IKK/NF-κB targets. Additionally, the presence of NIK in the mitochondria of primary glial cells suggests that NIK may regulate mitochondrial functions during development and/or in normal, untransformed cells. Our findings establish a new paradigm for NIK function and may also have important implications beyond cancer in CNS disorders associated with mitochondrial dysfunction, such as Alzheimer’s and Parkinson’s disease.


**Complete List of Published Work:** https://www.ncbi.nlm.nih.gov/pubmed/?term=Sitcheran

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**D. Research Support**

**Ongoing Research Support**

1R01NS082554-01A1  Sitcheran (PI)  03/15/2014 – 02/28/2018  
NIH/NINDS  
**Title:** Pathway-Specific NF-kappaB Regulatory Networks in Glioma  
This project seeks to investigate the role of NF-κB signaling in regulating EMT and cancer stem cell survival in different glioma subtypes.  
**Role:** Principal Investigator

RP160842  Sitcheran (PI)  06/05/2016 - 06/04/2018  
Cancer Prevention & Research Institute of Texas (CPRIT) High-Impact/High Risk Award  
**Title:** Novel Roles for NIK/MAP3K14 in High-Grade Glioma: Regulation of Mitochondrial Dynamics to Control Cell Migration and Invasion  
Description: This proposal investigates the molecular underpinnings of finding that NIK regulates mitochondrial dynamics, subcellular localization and invasion.  
**Role:** Principal Investigator

1R21NS101394-01  Bayless (PI)  02/01/2017 – 01/31/2019  
NIH/NINDS  
**Title:** NIK Promotes a Leader Cell Phenotype in Glioma  
Description: This proposal investigates the molecular mechanisms by which NIK regulates expression of Integrin-α11 to initiate cancer cell invasion and promote tumor pathogenesis.  
**Role:** Co-Investigator

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**Completed Research Support**

K01CA118274  Sitcheran (PI)  08/03/2006 - 07/31/2012  
NIH/NCI, Diversity Training Branch  
**Title:** Investigating the role of NF-κB and N-myc in oncogenic pathways of the CNS  
This proposal investigated the role of NF-κB in signal-dependent regulation of the glutamate transporter EAAT2 and glial cell survival in CNS tumors.  
**Role:** Principal investigator

K01CA118274-04S1  Sitcheran (PI)  09/30/2009 - 09/29/2011  
NIH/NCI  
**Title:** Investigating the role of NF-κB and N-myc in oncogenic pathways of the CNS  
This ARRA supplement for K01CA118274 supported the salary of new personnel to carry out the aims of the parent award in the independent phase at the PI’s new institution.  
**Role:** Principal investigator

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**Departmental Start-Up Grant**  Sitcheran (PI)  09/01/2009 - 08/31/2013  
Texas A&M University Health Science Center  
These funds were used to set up the PI’s laboratory and provide research and salary support to generate preliminary results for extramural grant applications.  
**Role:** Principal Investigator
2016 Seed Grant
Sitcheran (PI), Toussaint (Co-I) 02/01/2016 – 01/31/2017
Texas Brain and Spine Institute
**Title: Novel functions for NIK/MAP3K14 in high-grade glioma: regulation of mitochondrial dynamics to control cell migration and invasion**
This seed grant investigates our novel finding that NIK is localized to mitochondria and regulates mitochondrial morphology in glioma cells
*Role: Principal Investigator*

Pending Research Support

2R01NS082554-04 Sitcheran (PI) 07/01/2017 – 06/30/2022
NIH/NINDS
**Title: Investigating Novel Functions for NIK/MAP3K14 in High-Grade Glioma**
This proposal builds on results from the parent grant (1R01NS082554-01A1) to investigate NF-κB-dependent and -independent mechanisms by which NF-kappaB-inducing kinase (NIK) regulates mitochondrial dynamics to control cell invasion
*Role: Principal Investigator*
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: Laura N. Smith

eRA COMMONS USER NAME (credential, e.g., agency login): LSMI20

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.)

<table>
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<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
<th>Completion Date MM/YYYY</th>
<th>FIELD OF STUDY</th>
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<tbody>
<tr>
<td>Hendrix College (Conway, AR)</td>
<td>B.A.</td>
<td>05/2000</td>
<td>Psychology (Biol. minor)</td>
</tr>
<tr>
<td>George Mason University (Fairfax, VA)</td>
<td>M.A.</td>
<td>05/2004</td>
<td>Biopsychology</td>
</tr>
<tr>
<td>George Mason University (Fairfax, VA)</td>
<td>Ph.D.</td>
<td>06/2008</td>
<td>Biopsychology</td>
</tr>
<tr>
<td>University of Texas Southwestern Med Ctr, Dallas</td>
<td>Postdoctoral</td>
<td>07/08-08/12</td>
<td>Molecular Neuroscience</td>
</tr>
<tr>
<td>Harvard Medical School, McLean Hospital (Belmont, MA)</td>
<td>Postdoctoral</td>
<td>08/12-06/16</td>
<td>Molecular Neuroscience</td>
</tr>
</tbody>
</table>

A. My former training, education and experience make me well-qualified to conduct the proposed research. My educational background began with a broad training in psychology which narrowed to neuroscience with additional molecular neuroscience-based investigations of neuropsychiatric disorders, including addiction and autism. As an undergraduate, I was employed as an in-home aide to children with neurodevelopmental disorders, including several with autism, in some cases conducting daily behavior-based therapy. This experience still influences my research interests. My graduate training provided me with an excellent base in dendritic neuronal morphological, statistical, and rodent behavioral analyses, with a concentration in drug- and age-related effects on brain plasticity. These skills are demonstrated by my authorship on several publications during this time, two of which are mentioned here (1, 2). In my postdoctoral training, I continued to study drugs of abuse, investigating the role of developmental proteins in their effect on the brain and behavior, while adding molecular, cellular and biochemical techniques to my skill set. The study of developmental proteins in rodent models lacking their expression also offered opportunities to investigate normal brain plasticity, reward and motor function in autism-related neurodevelopmental disorders. During this time, I co-authored several manuscripts, including an investigation of the role of the developmental protein FMRP, which is the focus of the current proposal, in the regulation of synapse elimination (3). I continued studying FMRP, first-authoring a publication in Neuron (4) on its role in cocaine-related plasticity. The current application logically extends my prior research, in part, addressing the role of FMRP in understudied brain regions with high potential relevance to autism, and in part, continuing to address its role in addiction using a behavioral model with face validity.


B. Positions and Honors

Positions and Employment
2016-present Assistant Professor, Department of Neuroscience & Experimental Therapeutics, Texas A&M University Health Science Center, Bryan, TX

Other Experience and Professional Memberships
2005-2010 American Psychological Association (APA)
2006-2008 APA Division 6: Behavioral Neuroscience & Comparative Psychology
2006-2007 Sigma-Xi, The Scientific Research Society
2002-present Society for Neuroscience

Honors
2003 Nomination for Outstanding Teaching Assistant, George Mason University
2004 Research Fellowship Award, George Mason University, DBS Program
2004 Grant-in-Aid of Research from Sigma Xi, The Scientific Research Society
2005 Outstanding DBS Program Doctoral Student Award, George Mason University
2006 Outstanding Graduate Student Instructor Award, George Mason University
2007 Dissertation Research Fellowship Award, George Mason University
2007 Grant-in-Aid of Research from Sigma Xi, The Scientific Research Society
2007 Dissertation Research Award, American Psychological Association
2008 Invited presentation, Virginia Graduate Research Forum, Richmond, Virginia
2008 Institutional NRSA (T32), National Institutes of Health (NIDA)
2009, 2010 Individual NRSA (F32), National Institutes of Health (NIDA)
2011, 2012 Postdoctoral Fellowship, FRAXA Research Foundation
2014 Eleanor and Miles Shore HMS Fellowship, Harvard Medical School, McLean Hospital
2015 Alfred Pope Award for impact of a publication, McLean Hospital
2015 Phyllis and Jerome Lyle Rappaport Mental Health Research Fellowship, McLean Hospital
2016 American College of Neuropsychopharmacology Travel Awardee

B. Contribution to Science

1. My graduate training publications addressed the problem of early drug use, primarily during the period of adolescence. Adolescent drug use is particularly concerning given that the brain is still undergoing active development during this time, putting it at greater risk for lasting consequences of drug and alcohol use. Furthermore, increased risk-taking and peer-pressure at this age can increase the likelihood of exposure to abused substances. Using a rat model, we treated adolescent and adult rats with drugs of abuse, primarily nicotine, or vehicle control and examined behavioral and neuronal morphological outcomes after lengthy withdrawal periods, when all animals were adults. This work indicates that adolescents are uniquely vulnerable to lasting changes in brain and behavior following drug exposure. Specifically, adult animals that received nicotine as adolescents showed increased dendritic elaboration in a reward-related brain region and less flexibility in learned behavior. Our work also implicates the dopamine D3 receptor pathway in the development of nicotine sensitization specifically in adolescents.

2. Addiction to drugs of abuse, such as cocaine, is thought to be mediated by lasting changes in the brain that support and promote maladaptive behaviors associated with continued use. Given that changes in the brain following drug exposure are reminiscent of changes seen both during learning and memory and during earlier brain development, such as dendritic spine formation and elimination, we investigated the potential roles of developmental proteins. In earlier work, my postdoctoral lab showed that a transcription factor called MEF2, known to regulate synapse number, also played a role in cocaine-induce spine increases. Changes in spine density and shape can also be observed in neuropsychiatric and neurodevelopmental disorders, such as the autism-related fragile X syndrome. In collaboration with Dr. Kimberly Huber's laboratory at the University of TX Southwestern Medical Center, which studies fragile X mental retardation protein, or FMRP, we observed that indeed FMRP is required for MEF2-dependent synapse elimination. Following this finding, I then led a project that showed FMRP is required for the normal development of cocaine-associated behaviors, including behavioral sensitization and conditioned place preference. At the same time, we observed that cocaine caused significant increases, likely precocious, in synapse number and strength in the fragile X mouse compared to normal, wild-type mice.


   c. Invited video associated with our publication in the journal Neuron: https://www.youtube.com/watch?v=fpzwdg4Zc5A


3. As a postdoc, I led another major project investigating the role of cocaine-induced plasticity in the development of reward and drug sensitivity. The work stemmed from my original observation that mice lacking a learning and memory-related protein called Arc (the activity-regulated cytoskeleton-associated protein) show sensitivity to cocaine in certain assays. Additional work suggests that experience-dependent plasticity in Arc KO mice is altered, such that mice lacking Arc develop reward-related sensitivity to cocaine after prior exposure compared to wild-type controls. Thus it appears that Arc normally plays a role in limiting the development of sensitivity to cocaine. However, this lower sensitivity is associated with greater intake. We are now writing this manuscript for submission to the journal Biological Psychiatry.


Complete List of Published Works in MyBibliography:
https://www.ncbi.nlm.nih.gov/sites/myncbi/1fuAc7kwQj-Qg/bibliography/51091044/public/?sort=date&direction=descending

D. Additional Information: Research Support and/or Scholastic Performance

Completed Research Support
Phyllis and Jerome Lyle Rappaport Mental Health Research Fellowship 07/15-06/16
The goal of this project is to study the role of Arc in drug-induced reward, as well as associated synaptic alterations that occur in reward-related brain circuitry.
Role: PI

Eleanor and Miles Shore Harvard Medical School Fellowship 07/14-06/15
The goal of this project is to study the role of FMRP in drug-induced reward and associated morphological correlates.
Role: PI
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:  Smith, Rachel J.

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

POSITION TITLE:  Assistant Professor, Department of Psychology

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.)

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<th>FIELD OF STUDY</th>
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<tr>
<td>University of California, Santa Barbara</td>
<td>B.S.</td>
<td>06/2002</td>
<td>Biopsychology</td>
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<tr>
<td>University of Pennsylvania</td>
<td>Ph.D.</td>
<td>12/2008</td>
<td>Neuroscience</td>
</tr>
<tr>
<td>Medical University of South Carolina</td>
<td>Postdoctoral</td>
<td>03/2014</td>
<td>Neuroscience</td>
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A. Personal Statement

My role as principal investigator on this project is to organize and oversee all aspects of the proposed studies, including the design, execution, and analysis of experiments. I have the experience and technical expertise required to successfully conduct this research proposal. My undergraduate, graduate, and postdoctoral experience has provided excellent training in behavioral neuroscience and addiction research. I have extensive experience with the drug self-administration paradigm and other rodent addiction models. I also have extensive experience with a variety of techniques used to study the neural mechanisms underlying behavior, including the use of optogenetics to activate or inhibit specific neuronal subpopulations or pathways with temporal precision. My lab currently works with several viral delivery vectors and is conducting optogenetic experiments related to an ongoing project in the lab funded by an R21 grant awarded by NIH. My previous research experiences have equipped me with the skills and knowledge necessary to carry out the current proposed research plan.

B. Positions and Honors

Positions and Employment

2015 -  Assistant Professor, Texas A&M University, Department of Psychology, Institute for Neuroscience
2014 - 2015 Research Assistant Professor, Medical University of South Carolina, Department of Neurosciences

Other Experience and Professional Memberships

2015 -  Member, International Society for Neurochemistry
2010 -  Member, International Behavioral Neuroscience Society
2003 -  Member, Society for Neuroscience

Honors

2014  R21 Cutting-Edge Basic Research Award (CEBRA), NIDA
2011  F32 Ruth L. Kirschstein National Research Service Award (NRSA), NIDA
2009  Travel Award, Gordon Research Seminar on Catecholamines
2008  Travel Award, International Narcotics Research Conference
2007  Travel Award, NIDA Mini-Convention at Society for Neuroscience
2005  F31 Ruth L. Kirschstein National Research Service Award (NRSA), NIDA
2004  Travel Award, NIDA Mini-Convention at Society for Neuroscience
C. Contributions to Science

1. Role for orexin in cue-elicited drug seeking. My graduate work in the laboratory of Gary Aston-Jones was focused on the role of orexin in cocaine seeking using self-administration and reinstatement paradigms in rats. Previous studies indicated that this neuropeptide may be involved in reward and addiction, in addition to its role in wakefulness and narcolepsy. The experiments I conducted for my PhD showed that orexin is universally involved in cue-induced drug seeking for both cocaine and heroin, but plays a complex role in drug and food reward behaviors. I speculated that orexin signaling was only involved in certain behaviors because these behaviors involved increased glutamatergic signaling in ventral tegmental area, a key site for orexin actions. I collaborated with lab members to write several highly-cited reviews on orexin's involvement in addiction, in addition to a recent perspective article discussing a possible unifying theory for orexin function in the brain.


2. Common neural mechanisms for stress- and cue-induced relapse. Based on the involvement of orexin in cocaine seeking elicited by stress or drug-associated cues, I hypothesized that stress- and cue-induced reinstatement (relapse) might share common neural mechanisms. Norepinephrine and CRF play a well-established role in drug seeking and reinstatement elicited by stressors, but it was somewhat assumed that they did not play a role in reinstatement elicited by cues. However, I found that cue-induced reinstatement of cocaine seeking was blocked by noradrenergic and CRF antagonists. Further, I found that drugs acting at imidazoline receptors (hypothesized to be closely associated with the noradrenergic system) were also successful at blocking reinstatement without any effect on locomotor activity, in contrast to adrenergic drugs. Altogether, these results indicate that neural pathways typically associated with stress also play a role in relapse triggered by drug cues.


Complete List of Published Work in My Bibliography:

D. Research Support

Ongoing Research Support
R21 DA037744-02 Smith, Rachel J (PI) 04/01/14-03/31/17 (NCE)
Opposing Roles of Distinct Output Projections from Prefrontal Cortex
Role: PI
### Completed Research Support

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<td>PI</td>
<td>04/15/11</td>
<td>01/24/13</td>
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<td>Molecular Mechanisms of Cocaine-Induced Alterations in Accumbens AMPA Receptors</td>
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<td>08/31/07</td>
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<td>Involvement of protracted withdrawal in morphine relapse</td>
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Role: PI
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: Michael Steven Smotherman

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

POSITION TITLE: Associate 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.)

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<td>Occidental College</td>
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<td>1989</td>
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<td>University of Maine</td>
<td>M.S.</td>
<td>1992</td>
<td>Zoology</td>
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<tr>
<td>University of California, Los Angeles</td>
<td>Ph.D.</td>
<td>1998</td>
<td>Physiology</td>
</tr>
<tr>
<td>University of California, Los Angeles</td>
<td>Postdoc</td>
<td>1999-2004</td>
<td>Neurosci</td>
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A. Personal Statement

My lab at Texas A&M investigates the functional neurocircuits of the brain that control voice. We use an integrative approach, employing behavior, physiology, and cellular/molecular tools to better understand how specialized sensorimotor circuits are adapted to support extraordinary behaviors such as bat biosonar or human speech. My technical expertise lies primarily in using electrophysiological and pharmacological tools in animal models, but I have also received specialized training in bioacoustics, computational modeling, electronics and robotics. I have published research on ion channel kinetics, synaptic physiology, neuroanatomy, bioacoustics and behavioral research. My lab focuses on how brain controls the timing and acoustic properties of vocalizations, and to that end we focus on biosonar behaviors because of their unique adaptations for controlling pulse emissions. We use echolocating bats because they display the most temporally precise vocal behaviors of any mammal other than human and have a hypertrophied sensorimotor control network for connecting hearing to voice. My lab has developed several unique behavioral assays of vocal production, including assays of pitch control, loudness control, vocal-respiratory interactions, temporal patterning, and the production of complex vocal sequences. Regarding this proposal, I have recently published a series of papers describing unique features of the free-tailed bat’s sonar behavior that allow them to hunt and navigate in noisy, cluttered environments. At Texas A&M University we have in place all the facilities, tools and expertise necessary to train students to become successful independent researchers and conduct novel experiments to advance our understanding of biosonar behavior. My research has received funding from the NIH, DOE, NSF and several private foundations. I am chair of the graduate program for Texas A&M’s Institute for Neuroscience. I also serve as director of the Biology Department’s Animal facilities, and I am the current chair of the Texas A&M IACUC.
Selected publications highlighting my experience and qualifications relevant to this project:


B. Positions and Honors

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<th>Year</th>
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<tbody>
<tr>
<td>2010-</td>
<td>Associate Professor, Department of Biology, Texas A&amp;M University</td>
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<tr>
<td>2004-2009</td>
<td>Assistant Professor, Department of Biology, Texas A&amp;M University</td>
</tr>
<tr>
<td>2000-2004</td>
<td>Postdoctoral Researcher, Department of Physiological Science, UCLA.</td>
</tr>
<tr>
<td>2002</td>
<td>Grass Fellow, Woods Hole Marine Biological Laboratory</td>
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Other Relevant Professional Experience

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<td>1989-1990</td>
<td>Environmental Surveyor, Diagnostic Engineeings Inc, Arcadia CA.</td>
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<tr>
<td>2011-</td>
<td>Chair, Graduate Program Texas A&amp;M Institute for Neuroscience</td>
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<tr>
<td>2011-</td>
<td>Graduate Advisor, Texas A&amp;M Institute for Neuroscience</td>
</tr>
<tr>
<td>2012-</td>
<td>Environmental Consultant, City of San Antonio (Bat Expert)</td>
</tr>
<tr>
<td>2016-</td>
<td>Chair, Institutional Animal Care and Use Committee, Texas A&amp;M</td>
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Honors

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<td>1992</td>
<td>University of Maine, Outstanding Graduate Student Research Fellowship</td>
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<td>1997</td>
<td>Edith Hyde Memorial Scholarship in Physiology (UCLA)</td>
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<td>1998</td>
<td>UCLA Dissertation Year Fellowship</td>
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<td>2000-2003</td>
<td>NIH-NRSA F32 Post-doctoral Fellowship (NIDCD)</td>
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<td>2000</td>
<td>Capranica Foundation Award in Neuroethology (Honorable Mention)</td>
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<tr>
<td>2002</td>
<td>Woods Hole Marine Biological Laboratory Young Investigator Award</td>
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</table>

C. Contribution to Science

1. **Auditory Neuroscience**: For my PhD thesis research I used single cell electrophysiology (patch-clamp recordings) to identify and characterize the ion channels in vertebrate hair cells and used modeling to explain how ion channel functions contributed to the spectral and temporal response properties of the auditory system. This research showed for the first time that ion channel kinetics could be used as an efficient mechanism for auditory spectral filtering at low frequencies, but imposed significant constraints at high frequencies. An important element of this research was that it provided a biological basis for constraints and trade-offs between temporal and spectral resolution that shaped the evolution of vertebrate auditory systems. This research was funded by the NIDCD (PI-Narins).

2. **Neural Circuits Controlling Biosonar Pulse Emissions**: As a postdoc I investigated how auditory feedback was used to adjust vocal pitch in mammals. We used horseshoe bats because these animals adjust the pitch of their outgoing sonar vocalizations to compensate for
flight-induced Doppler-shifts in the pitch of the returning echoes. This sensorimotor process occurs in less than 15 ms and is one of the most precise examples of sensory feedback known in any animal. I located a midbrain region where excitatory and inhibitory inputs from the auditory system directly manipulated neural activity in the descending motor pathways to finely tune the spectro-temporal features of the outgoing pulse emissions in real time. This remains the only published description of the cellular and synaptic mechanisms by which the mammalian brain uses auditory feedback to control vocal pitch. This work is particularly relevant to this proposal because it highlights the tools and methodology for identifying midbrain circuits used to control biosonar emissions, and illustrates how these circuits may be separate from the ones involved in scene analyses, target selection and attention. This research was funded by the NIDCD (PI-Metzner). Representative publications:


3. Mechanisms for Mitigating Acoustic Interference In Biosonar: My lab at Texas A&M has successfully used echolocating free-tailed bats for the last decade to investigate how pulse acoustics and timing of pulse emissions were manipulated to minimize mutual interference between bats. Free-tailed bats are highly gregarious, living in dense colonies of millions, and therefore provide a uniquely compelling model for how animal biosonar systems are adapted to function in noisy, cluttered environments. We have developed a variety of different behavioral assays to directly measure how pitch, amplitude and timing are influenced by auditory feedback in bats, and we have used electrophysiology, pharmacology, neuroanatomy and molecular tools to map the neurocircuits involved in specific behaviors. Relevant to this proposal, we were the first group to demonstrate a role for the mammalian basal ganglia circuitry in biosonar performance, which may be important because in mammals generally the BG circuits are essential for learning and plasticity and provide a major substrate for sensorimotor integration. We were also the first to show that striatal dopamine is important for controlling vocal pitch and timing in bats. My lab also discovered a novel cooperative behavior exhibited by bats that allows them to optimize pulse emission rates relative to group sizes, thereby optimizing sonar performance in social conditions. This research was funded by NIH and NSF (PI- Smotherman). Representative publications:


4. Vocal Communication in bats: Bats display a prominent repertoire of complex vocal communications that have only recently come to light. My lab has been at the forefront of investigating the behavioral ecology and bioacoustics of these singing behaviors. We have focused on singing by the free-tailed bat, and currently maintain the only captive colony of singing bats in the world. These studies have focused on building probabilistic models of song composition, investigating the special role of song syntax on bat behavior, and what special ecological factors may have promoted singing as a signal in bats. Singing in bats may play a social cohesion role similar to singing by humpback whales or the signature whistles used by dolphins.
Complete List of Published Works can be found here:
http://www.bio.tamu.edu/FACMENU/FACULTY/SmothermanM.php

D. Research Support

**Ongoing Research Support**

1. NSF IOS-1354381 Smotherman (PI) 8/01/14-7/31/2017
   “Network strategies used by bats to improve social sonar”.
   This goal of this study is to develop novel algorithms for explaining how groups of bats coordinate their sonar pulse emissions to reduce mutual interference. Free-tailed bats adjust the temporal patterns of their pulse emissions to optimize information throughput in different social and behavioral contexts. The project fuses biology with communications and information theory by comparing algorithms found in bat sonar networks with algorithms developed for artificial wireless communication networks, and uses a combination of empirical behavioral studies and computational modeling to identify and validate a novel mechanism for coordinating sonar pulse emissions by groups of animals. This project currently receives 16% effort, but will be completed before the start of the proposed MURI research project.

2. DOE DE-EE0007032 Seivert (PI)/Smotherman(Collaborator) 9/19/2015-8/31/2017
   “A Biomemetic Ultrasonic Whistle for Use as a Bat Deterrent on Wind Turbines”
   This goal of this study is to use 3D printed models of the bat larynx to provide a cost-effective method for generating loud ultrasonic pulses to deter bats from flying into windmills. This is a collaborative project with U Mass- Amherst, and Texas A&M’s role is to conduct bat behavioral tests of acoustic responses to artificial whistles manufactured at U Mass. It takes 2% of the PIs time and will be completed before the start of the proposed MURI research project.

**Completed Research Support**

3. R03 DC007962 Smotherman (PI) 8/01/06-8/01/10
   “Coordination of speech and breathing in mammals”.
   This goal of this study was to characterize midbrain pathways and cellular/synaptic mechanisms required for coordinating normal breathing movements during vocalizing in mammals.
NAME: Farida Sohrabji

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

POSITION TITLE: Joseph Shelton Professor of Neuroscience

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.)

<table>
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<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
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<th>FIELD OF STUDY</th>
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<tbody>
<tr>
<td>St. Xaviers College, Bombay, India</td>
<td>B.A.</td>
<td>1982</td>
<td>Psychology</td>
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<tr>
<td>Bombay University, Bombay, India</td>
<td>M.A.</td>
<td>1984</td>
<td>Clinical Psychology</td>
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<tr>
<td>University of Rochester, Rochester, NY</td>
<td>M.S.</td>
<td>1989</td>
<td>Neurobiology</td>
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<tr>
<td>University of Rochester, Rochester, NY</td>
<td>Ph.D.</td>
<td>1991</td>
<td>Biopsychology/Neurobiology</td>
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<tr>
<td>Columbia University Medical Center, NY, NY</td>
<td>Postdoctoral</td>
<td>1991-1994</td>
<td>Neurobiology/MolecularBiology/Tissue Culture</td>
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NOTE: The Biographical Sketch may not exceed five pages. Follow the formats and instructions below.

A. Personal Statement

My research program focuses on brain-immune interactions regulated by age and sex hormones and its implications for neuro-inflammatory diseases such as stroke in women. Stroke is one of the leading causes of disability and mortality in the US, and, with age, disproportionately affects women. Few stroke therapies are available and developing stroke neuroprotectants is the focus of considerable research, although most have failed to show translational effectiveness. Increased risk for stroke after menopause has led to the hypothesis that estrogen therapy may improve stroke outcomes in older females, and this was shown to be effective using ovariectomized young female rats or mice as a model. However, using acyclic middle aged female rats, we were the first to show that estrogen treatment to this group, paradoxically, exacerbates stroke induced infarction. We further showed that it is the age-related loss of IGF-1 that reduces the effectiveness of estrogen as a neuroprotectant, and can be reversed by IGF-1 infusions post-stroke. These data suggest the exciting possibility that IGF-1 may be an effective stroke neuroprotectant for aging females. This renewal application will therefore focus on the translational potential of IGF-1, by determining the cellular/molecular mechanism underlying the effects of IGF-1 and more importantly, to determine whether IGF-1 improves stroke outcomes in the long term. Additionally, this application will also test the prediction that the aging astrocyte is the critical mediator of the outcomes of ischemic stroke. Below are recent invited book chapters that I have authored that focus on the theme of sex and sex differences in stroke and the role of IGF-1 as a stroke neuroprotectant.

B. Positions and Honors

1990-1994 Postdoctoral Fellow/Associate Research Scientist, Columbia University College of Physicians and Surgeons.
1995-1998 Associate Research Scientist, Human Anatomy and Neurobiology, Texas A&M HSC
1998-2003 Assistant Professor, Human Anatomy and Neurobiology, Texas A&M Health Science Center
2003-2009 Associate Professor, Human Anatomy and Neurobiology (reorganized as Neuroscience and Experimental Therapeutics) Texas A&M Health Science Center
2009-present Professor, Neuroscience and Experimental Therapeutics Texas A&M Health Science Center

Other positions:
2016- Fellow of the American Heart Association (Stroke Council)
2012- Joseph H. Shelton Professor of Neuroscience
2011-present Vice-Chair, Texas A&M Institute of Neuroscience
2007-present Director, Women’s Health in Neuroscience Program
2007-present Associate Department Chair, Neuroscience and Experimental Therapeutics
2006-present Adjunct Faculty, Department of Psychology, TAMU
2005-present Texas Brain and Spine Institute (Research Director, 2010-present)
1997-present Faculty of Neuroscience/Texas A&M Institute of Neuroscience/Faculty of Reproductive Biology

Professional Service:
NNRS study section: 2007-2012 (Chartered member 2008)
ICER study section: 2014-2018 (Chartered member 2015)
Special Emphasis Panels: MDCN2, 2002; BDCN 2009
AHA 1A Study Section Brain/Renal 2005-2009; Co-Chair 2008-2009
Ad hoc review: NSF (Endocrinology), Alzheimer’s Association 1997, 1999-2009
Member, Advisory Committee on Research on Women’s Health (NIH/OD) 2009-2013
External Advisory Committee, Oklahoma Reynolds Center on Aging, 2009
Editorial Board, Endocrinology 2010-2013
Frontiers in Aging Neuroscience, Editorial Board, 2009-present
Organization for the Study of Sex Differences (OSSD), Treasurer, 2012-2015
Texas Alzheimer’s Research Consortium and Care (TARCC): Steering committee member 2013-2016

C. Contribution to Science

1. Development of a female reproductive aging model:
My research program over the last 20 years has centered on the effects of estrogen on neuroinflammation and stroke. Our earlier studies were performed in young ovariectomized females, which mimics a surgical menopause. About 15 years ago, I made a critical decision to study a more clinically valid animal model to study hormone replacement. We selected 10-12 month old (Sprague Dawley) female rats. These rats are acyclic (as determined by daily vaginal smears), have undetectable levels of estrogen, low levels of progesterone and elevated levels of FSH. This hormonal profile more closely mimics menopausal females. We reported that this middle aged acyclic female differs dramatically from normally cycling adult females in its response to inflammatory stimuli and, more importantly, the effects of estrogen are dependent on the reproductive age of the animal. Thus while estrogen is neuroprotective and anti-inflammatory in young females, it is diametrically opposite in middle-aged acyclic females.

2. Stroke and reproductive aging: Stroke occurs more often in the elderly, and within that demographic, stroke occurs more often, and is more severe, in women. Our preclinical studies have shown that stroke is more severe in middle-aged females as compared to younger females and that estrogen treatment is not neuroprotective in this older population. Our studies have focused aggressively on identifying new therapeutics for this older group. We have reported an age-related decline in circulating and parenchymal levels of the peptide hormone, IGF-1 and further shown that post stroke IGF-1 treatment is neuroprotective in this older female group. We are currently focused on IGF-1 dependent mechanisms (inflammation, maintenance of the blood brain barrier), as well as epigenetic regulators that mediate the effects of IGF-1.


3. Blood brain barrier in aging and ischemia: We were among the first lab to show that the blood brain barrier is more permeable in middle-aged female rats as compared to younger females. Furthermore, while estrogen treatment improves barrier function in young females, hormone treatment, paradoxically, increases barrier permeability in middle aged females. This observation provides a mechanistic clue as to why older animals have worse stroke outcomes.


4. Astrocytes as a critical target of aging: At a mechanistic level, our studies have led us to consider the possibility that cellular components of the blood brain barrier (astrocytes and endothelial cells) may be critical mediators of the stroke response. Post stroke, astrocytes provide trophic support for ischemic neurons and clearance of cytotoxic compounds. Our studies have shown that in the aging astrocyte, these repair mechanisms are inefficient, and may be associated with epigenetic alterations in this cell with aging.


5. In vivo experiments with miRNA therapeutics: Our recent work focuses on epigenetic changes in aging and innovative therapeutic strategies involving small non-coding RNA and histone modifying agents for stroke neuroprotection. Our first strategy was a ‘targeted’ approach, based on miRNA that would elevate endogenous levels of IGF-1. Thus, miRNA with consensus sites on the IGF-1 UTR were targeted with antagonirs in a stroke model. Although this
The approach proved successful in young females but was not effective in middle-aged females. In order to identify a neuroprotectant for older groups, we are profiling age and sex differences in circulating miRNA and age differences in histone methylation, to identify novel epigenetic modifiers.


Complete List of Published Work in MyBibliography:

D. Research Support
Ongoing:

1R01NS074895 Role: PI 9/01/11- 05/30/17
NIH/NINDS
Neuroprotection in the Aging Female Brain
Synopsis: The overall goal of this application is to determine the interaction of estrogen and IGF-1 in the context of stroke and neuroprotection in middle age females, using an animal model. The current application is a renewal of this application. (No cost extension)

1R01ES020276 (F. Sohrabji) Role: PI 9/15/11-5/31/17
NIH (NIA/NINDS/ORWH)
Epigenetics of the Aging Astrocyte: Implications for Stroke
Major goals: The overall goal of this application is to identify aging- and stroke-related epigenomic changes in astrocytes (in response to RFA ES 10-002). No overlap with present proposal.

R01AA024659 (Miranda) Role: Co-I 10/03/16-28/2/21
NIH/NIAAA
Prenatal microRNA neuro-therapeutics for fetal alcohol exposure.
Synopsis: The overall goal of this application is to develop epigenetic therapies for individuals exposed to fetal alcohol exposure.

Discovery Foundation, Dallas, TX Role: PI 1/1/15-12/31/17
The impact of IGF-1 on post-stroke depression and neuroinflammation in a preclinical model
Synopsis: This application examines the neuroinflammatory response to stroke and longterm consequences of stroke on depression in an animal model. There is no budgetary overlap with this application.

SCIRP160225 Role Co-PI 04/01/17-03/31/20
Department of Defense
Derivation of the Mechanisms Mediating the Adverse Effects of Morphine in a Rodent Model of SCI: Functional Recovery and Neuron Loss
Synopsis: This application examines the effects of morphine on neuroinflammatory response after spinal cord injury and its impact on cell survival and behavioral recovery. No overlap with present proposal

State Contract:
Development of TARCC Investigator Grant Program Role: PI 9/26/14-9/25/2018
Texas Council on Alzheimer’s Disease and Related Disorders

Completed (in the last 3 years):
Texas A&M Institute for Neuroscience 568
Estrogens, Ovarian Aging and Calcium Channel Modulation

Synopsis: The overall goal of this project is to examine sex differences and the effect of estrogen on calcium currents in basal forebrain cholinergic neurons in young and middle aged rats. No overlap with present proposal.

Circadian Clocks and Neuroprotection in Response to Stroke during Reproductive Aging

Synopsis: The overall goal is to examine whether alterations in circadian patterns will impact the severity of stroke in middle-aged females. No overlap with present proposal.
BIOGRAPHICAL SKETCH

NAME: Rahul Srinivasan

eRA COMMONS USER NAME: SRAHUL

POSITION TITLE: Assistant Professor

EDUCATION/TRAINING

<table>
<thead>
<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
<th>Completion Date MM/YYYY</th>
<th>FIELD OF STUDY</th>
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<tbody>
<tr>
<td>University of Mumbai, India</td>
<td>MBBS</td>
<td>01/2000</td>
<td>Medicine</td>
</tr>
<tr>
<td>University of Pittsburgh, Pittsburgh, PA</td>
<td>PhD</td>
<td>06/2006</td>
<td>Human Genetics</td>
</tr>
<tr>
<td>University of Pittsburgh, Pittsburgh, PA</td>
<td>Postdoctoral</td>
<td>07/2007</td>
<td>Molecular Genetics</td>
</tr>
<tr>
<td>California Institute of Technology, Pasadena, CA</td>
<td>Postdoctoral</td>
<td>07/2013</td>
<td>Neuroscience</td>
</tr>
</tbody>
</table>

A. Personal Statement

I have expertise in advanced microscopy, including live imaging of calcium signals in astrocytes in brain slices, advanced molecular biology, tissue culture and transgenic mouse generation. I published several papers on neuroprotection by nicotine in Parkinson’s disease as well as high impact papers to develop cutting edge tools for studying astrocyte biology. Based on my extensive training, technical experience and a track record of consistent publications in the fields of neurodegeneration and astroglial biology, I believe that I will be able to provide all of the required expertise and support for Jianrong Li to successfully execute the proposed project.

The following recent publications attest to my expertise in glial biology and Parkinson’s disease:


B. Positions and Honors

Positions Held:

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<tbody>
<tr>
<td>Internship; Residencies – University of Mumbai, India</td>
<td>General Physician, Mumbai, India</td>
<td>Doctorate, Human Genetics, University of Pittsburgh</td>
<td>Postdoctoral Associate, Molecular Genetics &amp; Biochemistry, University of Pittsburgh</td>
<td>Postdoctoral Research Scholar, Division of Biology, California Institute of Technology</td>
<td>Assistant Research Physiologist, Dept. of Physiology, UCLA</td>
<td>Assistant Professor, Dept. of Neuroscience and Experimental Therapeutics Texas A&amp;M Health Science Center</td>
<td></td>
</tr>
</tbody>
</table>

Honors and awards:

2004 American Society for Gene Therapy (ASGT) Excellence in Research Award
2008-2009 Michael J Fox Foundation (MJFF) Rapid Response Innovation Award

Texas A&M Institute for Neuroscience
C. Contribution to Science

I consider my recent work from 2013 to 2016 in the field of astrocyte biology and my postdoctoral work showing that nicotine is neuroprotective at nanomolar concentrations and that neuroprotection occurs via pharmacological chaperoning of neuronal nicotinic acetylcholine receptors to be two of my most important contributions to science.

I recently published two high impact papers in the field of astrocyte biology. Both these papers contribute new tools and transgenic mice for astrocyte research and are cited below:


I published several papers on nicotine neuroprotection in Parkinson’s disease from 2007 to 2016 and some of these papers are cited below:


A full list of my publications can be found at the following URL: https://www.ncbi.nlm.nih.gov/pubmed/?term=rahul+srinivasan

D. Additional Information: Research Support

2008-2009 Michael J Fox Foundation (MJFF) Rapid Response Innovation Award
2009-2012 Tobacco Related Disease Research Program (TRDRP) Postdoctoral Research Award
2013 American Parkinson Disease Association Research Grant
BIOGRAPHICAL SKETCH

Provide the following information for the key personnel and other significant contributors in the order listed on Form Page 2.
Follow this format for each person. DO NOT EXCEED FOUR PAGES.

NAME  George Stoica

POSITION TITLE
Professor

eRA COMMONS USER NAME
George

EDUCATION/TRAINING (Begin with baccalaureate or other initial professional education, such as nursing, and include postdoctoral training.)

<table>
<thead>
<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
<th>YEAR(s)</th>
<th>FIELD OF STUDY</th>
</tr>
</thead>
<tbody>
<tr>
<td>College of Vet. Medicine, Yassy-Romania</td>
<td>DVM</td>
<td>1966</td>
<td>Veterinary Medicine</td>
</tr>
<tr>
<td>Ohio State University, Columbus, OH</td>
<td>MSc</td>
<td>1982</td>
<td>Experimental Pathology</td>
</tr>
<tr>
<td>Michigan State University, East Lansing</td>
<td>Ph.D</td>
<td>1984</td>
<td>Experimental Pathology</td>
</tr>
</tbody>
</table>

A. Personal Statement

I am an experimental pathologist with more than 30 years of experience in conducting, as a PI, and as a Co-PI in many projects using spontaneous animal diseases and laboratory animals. The lab animals used in our previous projects were mainly dogs, rabbits, mice and rats. The projects were primarily funded by NIH and also private foundations. My major research interest is in spontaneous neurological disorders (e.g., cancer, degenerative disease) as animal models for human diseases.

B. Positions and Honors. List in chronological order previous positions, concluding with your present position. List any honors. Include present membership on any Federal Government public advisory committee.

1984-1990        Assistant Professor, Department of Veterinary Pathobiology, Texas A&M University
1990-1996        Associate Professor, Department of Veterinary Pathobiology, Texas A&M University. Phi Beta Delta, Soc. for International Scholars
1996-present    Full Professor, Department of Veterinary Pathobiology, Texas A&M University
1993-present    Adjunct Professor, University of Texas M.D. Anderson Cancer Center, Department of Carcinogenesis, Science Park-Research Division
1982             Phi Zeta, National Veterinary Medical Honor Society
1982             Upjohn Biology Education Award
1990             Tenure and Promotion to Associate Professor
1992             Joint Appointment with Department of Cardiology, Texas A&M University, College of Medicine, Scott & White Hospital, Temple, Texas.
1992             European Society of Pathology member
1993             Adjunct Associate Professor, University of Texas, M.D. Anderson, Cancer Center Department of Carcinogenesis, "Smithville Science Park."
1993             Elected Member of International Institute of Comparative Oncology
1993             PHI BETA DELTA Society. Honor Society for International Scholars
1996             Member of the organization committee of the 5th International Congress of Pathology, Greece, Promoted to Full Professor
1997             Developmental Leave (May to November), IARC-Lyon, France
1998             President of Phi Zeta Honor Society of Veterinary Medicine
2001             Elected Active Member of Texas A&M University Senate
2001             Honorary Member of the University of Yassy-Romania
2003  European Conference on Biomedical Optics, 22-25 June, Munich, Germany. Chairman of Novel Biomedical Instrumentation Session.

2005  Journal Scholarship Award from CL Davis DVM Foundation for the Advancement of Veterinary and Comparative Pathology

2008  Pfizer Award for Research Excellence

2011  European Neurological Society, member

2015  Certificate of Recognition, OMICS International

2015  Keynote Speaker, International Conference on Parkinson’s Disease & Movement Disorders, Frankfurt, Germany, Aug. 11-13, 2015

C. Selected peer-reviewed publications (in chronological order from 104 total). Do not include publications submitted or in preparation.


C. Research Support

Ongoing Research Support

2011 – 2012  The role of alpha synuclein in neurodegeneration, Rat model. MJ Fox Foundation for Parkinson’s disease. G.Stoica, PI. $73,199.94

2012 -2013  Role of dopamine and alpha synuclein in Parkinson’s disease. A rat animal model. G.Stoica PI. $150,000

### 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:** Sun, Yuxiang

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

**POSITION TITLE:** Assistant Professor (tenure-track)

**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.)

<table>
<thead>
<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
<th>Completion Date MM/YYYY</th>
<th>FIELD OF STUDY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beijing Medical University, Beijing, China</td>
<td>M.D.</td>
<td>07/1988</td>
<td>Medicine</td>
</tr>
<tr>
<td>University of Manitoba, Winnipeg, Manitoba, Canada</td>
<td>M.Sc.</td>
<td>02/1996</td>
<td>Physiology</td>
</tr>
<tr>
<td>University of Manitoba, Winnipeg, Manitoba, Canada</td>
<td>Ph.D.</td>
<td>10/2000</td>
<td>Molecular Endocrinology</td>
</tr>
</tbody>
</table>

**A. Personal Statement**

My research interests are nutritional regulation, glucose- and energy-homeostasis, and the pathophysiology of obesity and diabetes. I received outstanding Ph.D training at the University of Manitoba in Canada. My Ph.D thesis work was awarded the most prestigious research award for graduate students in the university (only one is awarded each academic year 1). I subsequently received a 3-year Postdoctoral Fellowship from Canada and joined Dr. Roy Smith’s laboratory at Baylor College of Medicine. Ghrelin is the only known circulating orexigenic hormone. My postdoctoral work was focused on understanding the regulatory effects of ghrelin and its receptor (GHS-R) on the growth hormone signaling pathway during aging. I performed extensive structural and functional analysis of ghrelin and GHS-R. I was the first to generate and characterize ghrelin (MCB, 2003) and GHS-R (PNAS, 2004) knockout mice, and demonstrated that ghrelin’s effects on GH release and appetite are mediated through GHS-R. I subsequently discovered that ghrelin deletion increases insulin secretion and mitigates diabetes by regulating UCP2 in the pancreatic islets (Cell Metab. 2006). My work was the first to establish that ghrelin is a new player in diabetes.

Directly relevant to the current proposal, my lab has discovered that GHS-R regulates thermogenesis (Aging Cell 2011; PLoS ONE 2011, Methods Enzymol. 2012, Aging 2014, Diabetes 2016). We reported that old Ghsr−/− mice are lean and insulin-sensitive due to increased energy expenditure, but not energy intake. Global GHS-R ablation activates thermogenesis in brown and beige fat by enhancing mitochondrial biogenesis and improving mitochondrial dynamics. Our most recent study (in press in Diabetes) has further demonstrated that GHS-R mediated thermogenic is primarily centrally regulated; GHS-R deletion in neurons enhances thermogenesis and has robust protective effect on diet-induced obesity. These findings strongly suggest that the ghrelin receptor is a novel thermogenic regulator and its site of action is in the brain.

GHS-R is highly expressed in hypothalamus, including the “hunger center” arcuate nucleus (ARC) and “satiety center” ventromedial hypothalamus (VMH), both of which are known to be involved in thermoregulation. In this proposal, we hypothesize that GHS-R regulates thermogenesis in hypothalamic ARC and VMH neurons, and hypothalamic GHS-R deletion protects against diet-induced obesity and insulin resistance by activating thermogenesis. We will use our newly generated hypothalamic-specific GHS-R knockout mice to unravel the roles and pertinent mechanisms of GHS-R in thermoregulation. The successful completion of this proposal will provide proof-of-concept evidence on whether targeting GHS-R in hypothalamus to burn fat might be a paradigm-shifting anti-obesity strategy.

My publications are among the highest referenced literature in ghrelin research. I served as PI for completed NIH R03 and USDA grants. I currently have research support from the AHA and ADA. As demonstrated in our publications and preliminary data, my lab has extensive experience in characterization of metabolic phenotypes of in vivo mouse models, ex vivo and in vitro cell systems, histological, biochemical, and cellular/molecular techniques for mechanistic studies. To ensure the complete success of the project, I have recruited a well-trained neuroscientist, Dr. Chia-Shan Wu, to serve as a primary investigator for
this proposal. Dr. Wu has extensive experience in neuroanatomy, in situ hybridization, icv injection, neuronal culture, immunohistochemistry/immunofluorescence, and behavioral neurology. To ensure complete success, the PI has also brought together a team of experts to assist in the project. Dr. Yong Xu will conduct electrophysiology studies for us, and assist targeted neuronal injections. Dr. Fukuda has extensive experience in neuronal culture and neuronal pathway identification. Dr. Yu-Hua Tseng, a renowned expert in BAT thermogenesis & mitochondrial function. They will all make their expertise and resources available to us. I believe that my expertise and experience, plus the support of my collaborators, will be sufficient for ensuring the complete success of this project. The publications below are closely relevant to this proposal:


B. Positions and Honors

Positions and Employment
1988-1991 Physician, First Teaching Hospital, Beijing Medical University, P.R.China
1993-1996 M.Sc. Student, Molecular Endocrinology Laboratory, University of Manitoba, Canada
1996-2000 Ph.D. Student, Gene Technology Laboratory, University of Manitoba, Canada
2000-2003 Postdoctoral Fellow, Huffington Center on Aging, Baylor College of Medicine, Houston, TX
2003-2005 Research Associate, Huffington Center on Aging, Baylor College of Medicine, Houston
2006-2007 Instructor, Department of Molecular and Cellular Biology, Baylor College of Medicine.
2007 –Sep. 2008 Assistant Professor, Dept. of Molecular and Cellular Biology, Baylor College of Medicine
Oct. 2008- Nov. 2015, Assistant Professor (tenure-track), USDA ARS Children’s Nutrition Research Center (CNRC), Huffington Center on Aging (HCOA), Departments of Pediatrics & Molecular and Cellular Biology, Baylor College of Medicine. Houston, TX
Oct. 2008-present. Adjunct Assistant Professor, USDA ARS Children’s Nutrition Research Center (CNRC), Departments of Pediatrics, Huffington Center on Aging (HCOA), Baylor College of Medicine. Houston, TX
Dec. 2015- present Assistant Professor (tenure-track), Dept. of Nutrition and Food Science, Texas A&M University (TAMU)
Jan, 2016-present Full Member, Center for Translational Environmental Health Research, TAMU

Honors
1999 - Apotex Major Award for Excellence of Doctoral Research in Molecular Biology, Faculty of Medicine, University of Manitoba, Canada (highest research award for graduate students; only one is honored each academic year)
- Women in Endocrinology (WE) Travel Award (20 awardees internationally), 81st Annual Meeting of Endocrine Society, San Diego, CA
- Alumni Conference Travel Award, Alumni Association, University of Manitoba, Canada
- Graduate Student Poster Award (1st place in the department), University of Manitoba, Canada
2000 - MRC Postdoctoral Fellowship, Medical Research Council of Canada (3-year stipend plus research travel)
2003 - ENDO 2003 Travel Grant Award (116 out of 3000 abstract submissions), Endocrine Society 85th Annual Meeting, Philadelphia, PA
- Women in Endocrinology (WE) Aventis Metabolism Award (total 10 Recipients)
2005 - ENDO 2005 Travel Grant Award, Endocrine Society’s 87th Annual Meeting, San Diego, CA
2007 - Finalist, Rulanette and Berdon Lawrence Bone Research Award, Bone Disease Program of Texas. Endocrine Society Travel Award, 89th Annual Meeting, Toronto, Canada
2010 - Young Investigator Award, the Fat Disorders Research Society, Endocrine Society’s 92th Annual Meeting in San Diego, CA
Abstract selected as one of 10 “Novel & Newsworthy Top Picks” from among 1100 mini-symposium submissions. 50th Annual Meeting of American Society for Cell Biology.


2016 - Selected for moderated poster presentation, 76th Scientific Sessions | American Diabetes Association, June 10 - 14, 2016, New Orleans, LA

Other Professional Experience

Review panels

2010 Grant reviewer (Ad Hoc) for “Diabetes UK”
2012 Grant reviewer (Ad Hoc) “AHA innovative research groups” (Oct. 2012)
2013 Grant reviewer (Ad Hoc) for “AFAR’s Biology of Aging Grant Programs”
Member on AFAR’s National Scientific Advisory Council (NSAC)
National Natural Science Foundation of China
2014 National Natural Science Foundation of China
Grant reviewer (Ad Hoc) for “NIH study section IPOD” (June 2014)
External Reviewer for “Diabetes Research Center P/FGrants of University of Michigan”
2015 Council Member of AFAR’s National Scientific Advisory Council (NSAC)
2015-2017 Research Grant Review Committee (RGRC) for American Diabetes Association
National Scientific Advisory Council of AFAR

Journals: 2013 “Regulatory Peptides”, “Molecular Endocrinology”, “Nature Communications”
2016 “International Journal of Endocrinology”, “Molecular Metabolism”

C. Contribution to Science

1. I started my postdoctoral training at Dr. Roy Smith’s lab shortly after ghrelin was discovered. Prior to ghrelin’s discovery, Dr. Smith’s lab at Merck cloned the growth hormone secretagogue receptor (GHS-R), which later became known as the ghrelin receptor. Trained as molecular biologist, I decided to take on the challenge of making knockout mice for ghrelin and GHS-R. I was the first to generate knockout mice for ghrelin (MCB, 2003) and GHS-R (PNAS, 2004). Now these mouse models are extensively used by academia around the world, and by major pharmaceutical companies such as Merck and Eli Lilly. These mouse models have also generated many high impact collaborative works (JCI, PNAS, JBC, MCB), and inspired several major reviews (Endo. Reviews, Trends Endocrinol. Metab.).


2. The most recognized function of ghrelin is its orexigenic effect as an appetite stimulant, so it was highly anticipated that ghrelin ablation would reduce obesity. To test that hypothesis, I bred ghrelin-null mice to leptin-deficient obese and diabetic ob/ob mice. Surprisingly, the ghrelin ablation did not reduce obesity at all. The finding was contradictory to the pharmacological effect of ghrelin, it indicates that ghrelin deletion can be developmentally compensated in vivo. Remarkably, I found that ghrelin ablation rescues the diabetic phenotype, and ghrelin ablation increases insulin secretion by reducing the expression of UCP2 in pancreatic islets (Cell Metab. 2006). This work was the first evidence that links ghrelin to diabetes, that has led to an entirely new line of research. Interestingly, we later found GHS-R deletion in ob/ob mice...
worsens the diabetes (Am J Phy. Endo. Metab 2012). This suggests that ghrelin system is complex; ghrelin’s effect on pancreatic islets may not be mediated by GHS-R.


c. Ma X#, Lin Y#, Lin L, Qin G, Pereira FA, Haymond MW, Butte NF, Sun Y (2012). Ablation of ghrelin receptor in leptin-deficient ob/ob mice has paradoxical effects on glucose homeostasis when compared with ablation of ghrelin in ob/ob mice. Am J Physiol Endocrinol Metab 303: E422-E431. PMID:22669248 (PMCID is N/A)


3. We have observed that the body weight of GHS-R null mice becomes significantly lower during aging. Our metabolic analysis indicates that the difference is not due to reduced food intake but rather is due to increased energy expenditure. During our characterization of the mice, brown fat was discovered in adult humans. Thermogenesis has been thought to be a promising strategy to combat obesity. Interestingly, we found that GHS-R expression in thermogenic brown fat is increased during aging and correlated with obesity. Our studies further reveal that the GHS-R ablation alleviates age-associated thermogenic impairment, leading to reduced obesity and improve insulin sensitivity (Aging Cell 2011; PLoS ONE 2011). This new finding shows for the first time that the ghrelin receptor is an important thermogenic regulator, and suggests that GHS-R antagonists might be a novel anti-obesity agent shifting the body’s energy balance from obesogenesis to thermogenesis. GHS-R regulates energy homeostasis by regulating thermogenesis but not energy intake, which challenges the dogmatic view that ghrelin regulates energy homeostasis primarily by its orexigenic property. Our follow-up studies show that GHS-R regulates thermogenic signaling by modulating both mitochondrial biogenesis and mitochondrial dynamics (Aging 2014). Our most recent study has further revealed that GHS-R deletion in neurons robustly abolishes diet-induced obesity (DIO), showing enhanced thermogenic thermogenesis (Diabetes 2016).


4. High fructose corn syrup (HFCS) has been heavily used in soft drinks and processed food in US. The safety of HFCS has been a subject of ongoing debate. We found that mice fed HFCS exhibit more severe insulin resistance than even the mice fed the higher calorie-containing high fat diet (HFD), but have no effect on obesity. Moreover, we found that GHS-R ablation attenuates HFCS-induced insulin resistance by attenuating adipose inflammation and hepatic steatosis (Nutrition & Diabetes 2013). So even though HFCS does not cause obesity per se, it is more hazardous, because it causes adipose and hepatic inflammation, which leads to severe insulin resistance. Thus, HFCS is not safe, and the dietary guideline of HFCS usage should be revisited. In addition, the data suggest for the first time that GHS-R antagonism has anti-inflammatory effects in fat and liver. We also found that GHS-R ablation attenuates HFCS-induced adipose inflammation and nonalcoholic steatohepatitis (NASH) by reducing pro-inflammatory M1 macrophage infiltration into these tissues and shifting macrophages toward anti-inflammatory M2. Recently, it has been shown that aalternatively-activated anti-inflammatory M2 macrophages are known to produce norepinephrine, which stimulates thermogenesis in BAT and promotes lipolysis in WAT (Aging 2016). Our new observation suggests that GHS-R may regulate thermogenesis and lipid mobilization via its effect on macrophage polarization.

5. Even though ghrelin is recognized as an orexigenic hormone, we found that deletion of neither ghrelin nor GHS-R gene changes long-term total energy intake. It has been suggested that both meal quantity and meal pattern affect body weight and body composition. Indeed, we found that GHS-R deletion increases both meal size and meal frequency, and alters appetite-regulating hypothalamic peptides (Journal of Nutrition 2014). This observation suggests that ghrelin signaling regulates energy homeostasis not by altering total energy intake per se, but rather by modulating feeding patterns. Because this is a novel concept, we have been invited by several nutrition journals to write follow-up papers on the subject. We have discovered cannabinoid receptor GPR55 regulates energy homeostasis (IJO 2015).


Complete List of Published Work in My Bibliography (55 peer-reviewed publications):
http://www.ncbi.nlm.nih.gov/sites/myncbi/1lCHesdf6kzAg/bibliography/48697663/public/?sort=date&direction=ascending

D. Research Support (for the past three years)
Ongoing Research Support
ADA: Basic Science Award #1-15-BS-177 Sun (PI) 1/1/2015-12/31/2017
“Ghrelin receptor in macrophages: A key mediator of both non-shivering thermogenesis in brown fat and adipose inflammation in white fat?”
To determine whether global GHS-R deletion regulates macrophage phenotypical shift in aging, and attenuates age-associated obesity and insulin resistance.

USDA/ARS CRIS grant: 6250-51000-059 Sun (Lead Scientist) 10/01/2014- 09/31/2019
“Metabolic Effects of Ghrelin and Glucagon-Like Peptide Hormones”
The aims of this project are to study gut-derived hormones, ghrelin and glucagon-like peptide 2(GLP-2), in the control of energy- and glucose-homeostasis.

Completed Research Support
Texas Medical Center Digestive Diseases Center (DDC) pilot/feasibility grant P30 DK56338
“The role of ghrelin receptor in dietary-induced steatohepatitis” Sun (PI) 3/1/15-2/28/17
To determine whether macrophage GHS-R plays a crucial role in pathogenesis of fructose-induced nonalcoholic steatohepatitis (NASH). We will study co-cultures of Kupffer Cells and hepatocytes.

AHA: Grant-in-Aid award 14GRNT18990019 Sun (PI) 1/1/2014-12/31/2016
“GHS-R promotes obesity and insulin resistance by regulating macrophage-mediated thermogenesis and adipose inflammation”
To elucidate the roles of GHS-R in thermogenesis and inflammation. The GHS-R global null mice and the primary cells from the null mice will be used for phenotypical characterization.

USDA/ARS CRIS grant: 6250-51000-055 Sun (PI) 10/01/2009-09/31/2014
“The role of ghrelin and its receptor in nutritional regulation of energy and glucose homeostasis”
To characterize the role of ghrelin in nutritional regulation of energy and glucose homeostasis. The study may provide new insight for dietary guidelines, and may lead to novel therapies.

Interim Bridge Fund of Baylor College of Medicine Sun (PI) 10/1/2013-3/31/2014
“The Role of Ghrelin Receptor (GHS-R) in Thermogenic Regulation”
To study the role of GHS-R in non-shivering thermogenesis during aging. The thermogenic phenotype of GHS-R null mice will be investigated.
A. Personal Statement

I have more than 35 years of academic experience and have trained graduate students and postdocs who subsequently have gone on to successful scientific careers. I have been a faculty member at summer courses taught to graduate students and postdoctoral fellows at Woods Hole and Cold Spring Harbor. I have served on the dissertation committees for more than 70 students. I have been successful in obtaining research funds for my own work, including 28 continuous years with an NIH R01, a Javits award, and smaller grants from foundations.

My scientific career has been concentrated on the topic of development and maintenance of synapses, particularly the neuromuscular junction. I study this synapse in health and disease. My own major contributions (and the ones I list below that came from work with my students) have been largely to understanding the role of glial (Schwann) cells at this synapse. I list 4 recent studies that meet these criteria, bolding the name of the graduate student associated with the work.

1. Li, Y., Lee, Y. I., and Thompson, W. J. 2011. Changes in aging mouse neuromuscular junctions are explained by degeneration and regeneration of muscle fiber segments at the synapse. *J. Neurosci.* 31: 14910-14919 PMCID 3213690. This study uses repeated vital imaging to show that NMJs undergo remarkable changes during the process of aging but that these changes occur suddenly and are linked to injury to muscle fibers and their degeneration and regeneration.

2. Smith, I. W., Mikesh, M., Lee, Y -I., and Thompson, W. J. 2013. Terminal Schwann cells participate in the competition underlying neuromuscular synapse elimination. *J. Neurosci.* 33:17724-17736 PMCID 3818548. This study shows that during synapse elimination in early rodent development, Schwann cells compete with nerve terminals for contact with the synaptic surface of the muscle and actively phagocytose terminals in synaptic contact with the muscle.

3. Kang, H., Tian, L., Mikesh, M., Lichtman, J.W., and Thompson, W.J. 2014. Terminal Schwann cells participate in neuromuscular synapse remodeling during reinnervation following nerve injury. *J. Neurosci.* 34: 6323-33 PMCID 4004816. This study shows that during reinnervation following nerve injury there are changes to NMJs that are due to the loss of association of Schwann cells with the old synaptic site.

4. Li, Y. and Thompson, W. J. 2011. Nerve terminal growth remodels neuromuscular synapses in mice following regeneration of the postsynaptic muscle fiber. *J. Neurosci.* 31: 13128-13136 PMCID 3181159. This study shows that deliberate damage to muscle fibers in the living animal with a laser microbeam causes synaptic changes that resemble those in aging and in neuromuscular disease.

B. Positions and Honors.
Jan. 2013-present, Professor of Biology, Texas A&M University
June 2014-present, Adjunct Professor, Department of Neuroscience, University of Texas at Austin
Sept. 1999-Dec. 2012, Professor of Biological Science, University of Texas at Austin
Sept. 1979-Aug. 1999 Assistant Professor, Associate Professor and Professor of Zoology, University of Texas at Austin
Nov. 1977-Dec. 1979 postdoctoral fellow, Department of Physiology and Biophysics, Washington University School of Medicine, St. Louis with Dale Purves; supported by USPHS traineeship
Aug. 1971--Jun. 1972; Sept. 1972--Sept. 1975 full-time graduate student, Department of Molecular Biology, U.C. Berkeley; major advisor: Gunther Stent; supported by NIH traineeship
Graduation with high honors, North Texas State University
MDA, NATO, and NIH (declined) postdoctoral fellowships
Selection as a "Searle Scholar" by Searle Scholars Program of the Chicago Community Trust, 1981
NIH Research Career Development Award, 1984-1989
member, NSF panel to evaluate applicants for NATO postdoctoral fellowships, 1987
College of Natural Sciences, University of Texas, Teaching Excellence Award, 2000
member NIH Molecular, Cellular and Developmental Neuroscience Study Section(MCDN-7), 1999-2003
NIH Javits Neuroscience Investigator Award, 2001-2007
Co-editor, special edition of the Journal of Neurocytology featuring articles from prominent scientists working on the neuromuscular junction.
member, Scientific Advisory Board, Spinal Muscular Atrophy Foundation, 2005-
member, editorial board, Neuron Glia Biology, Cambridge University Press, 2003-2010
member, NIH NST (NINDS) Study Section, Oct 2003-Jun 2007

C. Contributions to Science
My contributions to neuroscience and to understanding of the NMJ are described below:

1. **Activity and synapse elimination.** When I arrived as a postdoctoral fellow in the laboratory of Jan Jansen in 1975, experiments had just been completed that analyzed the change from polyneuronal innervation of muscle fibers at birth in rats to single innervation some two weeks later. The process was named “synapse elimination.” I had been taught by my doctoral mentor and the experiments of Terje Lømo the important role activity plays in the nervous system in general and in the control of muscle fiber and synaptic differentiation, in particular. I therefore was interested in the influence synaptic activity played in the process. First, with Jan Jansen, and later in my own laboratory, I explored the role of activity in this process, first by blocking impulse conduction along nerves and later by external stimulation of activity in the muscle itself. These experiments demonstrated the critical role that activity plays in the process. Subsequent studies have used additional techniques to demonstrate a change from coordinated to uncoordinated neural activity among competing motor neurons is crucial for the outcome of synapse elimination.


2. **Sprouting.** During my postdoctoral training I used muscle partial denervation in attempt to understand if the removal of some of the motor neurons, reducing the number of competing inputs, would affect synapse elimination. In the process, I became interested in the process of sprouting itself. First with Jan Jansen, I explored just how extensively individual motor neurons expand their terminal arbors in muscle. In the adult animal, there was a clear limitation: motor neurons could expand to innervate about 5 times the normal...
number of fibers they innervated, but no more, even at the expense of leaving muscle fibers denervated. This observation has been repeated several times since the initial publication. The observation sets a lower limit on how many motor neurons can be lost and muscle function maintained. It also implies the ability of motor neurons to grow is limited. When I established my own lab, I found that the sprouting phenomenon was not limited to motor axons but also involved sprouting (or process extension) of the Schwann cells that normally covered the NMJ, I became intrigued about the relationship between the two growth phenomena. By use of immunostaining, my graduate student, Young-jin Son, and I first demonstrated that the glial cell growth stimulated the nerve growth and guided its course through the muscle. Desiring a closer look at this process I learned vital imaging and made a transgenic mouse in which Schwann cells expressed GFP (with my graduate student Hyuno Kang and a postdoc Yi Zuo. Using this mouse bred to a mouse from Jeff Lichtman/Josh Sanes that labeled motor axons with a blue fluorescent protein, we were able to demonstrate that the things we observed in the static images with antibodies occurred in vivo, that the phenomenon was quite dynamic, and that the glial cells actually led the nerve growth (with graduate students Li Tian and Hyuno Kang). We have also clarified the role of glial cells in the reinnervation of synaptic sites (with graduate student Hyuno Kang), demonstrating that the glial cell coverage of the synaptic site that remains following denervation greatly influences what sites are reinnervated during nerve regrowth.

Son, Y.-J. and Thompson, W.J. 1995. Schwann cell processes guide regeneration of peripheral axons. Neuron 14: 125-132 PMID 7826630

Son, Y.-J. and Thompson, W.J. 1995. Nerve sprouting in muscle is induced and guided by processes extended by Schwann cells. Neuron 14: 133-141 PMID 7826631


3. Trophic mechanisms affecting developing NMJs. During denervation experiments in neonatal rat muscles we observed that the terminal Schwann cells disappeared from junctions (with graduate student Josh Trachtenberg). We showed that the mechanism here was apoptosis. We demonstrated that the dying cells could be rescued by exogenous application of the tropic factor neuregulin (with Josh Trachtenberg). Thus, neuregulin supplied by motor axons normally prevents neonatal Schwann cells from undergoing apoptosis. This finding has been confirmed not just for terminal Schwann cells, but also for Schwann cells associated with sensory receptors and peripheral nerves. We have since explored the role of neuregulin signaling in a variety of Schwann cell activities (with graduate student Chris Hayworth, see below).


4. Mechanisms of synapse elimination. I have been intrigued by the question of whether tSCs play any role in synapse elimination at the NMJ. Since NMJs appear to be altered during sprouting and reinnervation and SCs play a role in these phenomena, it seemed a distinct possibility. Our efforts at fluorescent imaging of junctions undergoing synapse elimination produced little evidence for an involvement of tSCs. We therefore undertook a study at the ultrastructural level making serial sections through entire junctions during synapse elimination, segmenting objects in the electron micrographs, and reconstructing 3-D views (with then undergraduate student and now graduate student Ian Smith and with postdoc Matt Lee). To our surprise, we found several unexpected results that profoundly alter the view of synapse elimination. First of all, at the time of birth and shortly before nerve terminals of several individual motor neurons are adjacent to each other on the muscle surface at the developing NMJ. SCs arrive at the muscle after the motor axons and begin to
intercalate between the nerve terminal endings. SCs assume a position above the muscle surface that is as close to the muscle synaptic surface filled with AChR as are the terminals (ca. 50 nm). As development proceeds after birth the SCs come to occupy a progressively larger portion of the postsynaptic surface and the portion contacted by nerves declines. Thus, it appears that tSCs compete with the nerve terminals for contact with the muscle. The SC’s apparent affinity for the postsynaptic surface declines with time and consequently the SCs assume a position where they cover the surface of the nerve terminal and contact the muscle fiber only at the edges of the nerve terminal. Interestingly, the AChR in the muscle change from a plaque to a pretzel during this period, and SC occupation of sites in the muscle with high density AChR may result in the presence of nerve activity in the loss of regions so contacted. Even more interestingly, the tSCs in the neonate are engaged in two activities we have not seen after the period of synapse elimination: (1) the tSCs send processes that intervene between the nerve terminals and muscle fibers and (2) the tSCs are engaged in phagocytic activity directed against the nerve terminals. It is our hypothesis that tSCs are part of the competitive processes leading to conversion to single innervation. tSCs clearly do not select the winner; their activities are directed against all the nerve terminals on the muscle fibers. Rather, we favor a model in which tSCs can accelerate the process by causing random removal of terminal boutons and the competition that drives synapse elimination is one that comes from the random reoccupation of synaptic sites by a nearby nerve terminal. This is a model proposed by Turney and Lichtman. Thus we believe tSCs are a mechanism for remodeling nerve terminals during synapse elimination. We are intrigued by the possibility that tSCs could play a similar role in remodeling junctions in aging (this proposal) and disease.


5. **Mechanisms of aging of NMJs.** NMJs are mostly stable following the period of synapse elimination in early development. That is, the position and shape of nerve terminals, the AChR plaque and the Schwann cells change only by intercalary growth to match the growth of the muscle fiber. However, in aging rodents and in humans as well, the NMJs undergo dramatic changes: NMJs become fragmented into varicose nerve endings that appose small islands of AChR (see figures in this application). Schwann cells proliferate and extend processes. These changes are occurring as the muscles themselves become weaker, although the relationship between the weakness and synaptic changes is controversial (and is an aim of this proposal). We have explored the events during aging by conducting repeated vital imaging of the same NMJs during aging (with graduate student Yue Li). To our surprise we found that NMJs do not change gradually as the animal ages but rather suddenly over the course of a week or so. The change is stochastic, so that only a small percentage of junctions change over any imaging period, but the changes accumulate with age. The change is apparently associated with the degeneration of the muscle fibers followed by their regeneration. We hypothesize that a cause of the changed morphology is proliferation and activity of the tSCs at the junction and we propose here to investigate this hypothesis. Our hypothesis is supported by several observations made by us and others. Our experiments have shown that almost identical synaptic changes follow deliberate laser ablation of muscle fibers. Similar, rapid changes in morphology result from the damage to muscle fibers resulting from Duchenne muscular dystrophy or administration of a myotoxin (cardiotxin) that causes rapid degeneration and regeneration of muscle fibers. Our experiments therefore suggest muscle fiber damage as a mechanism underlying aging-related change and further suggest that possible treatments involve addressing muscle fiber fragility in aging and/or the events underlying synaptic change.


Complete List of Published Work in MyBibliography:

D. Research Support
  Ongoing Research Support:
    R01 NS-20480-29 Thompson (PI) 06/01/2015—5/31/2020
    Formation and maintenance of neuromuscular synapses
    The goal of this study is to understand the role of Schwann cells at the neuromuscular junction.
    Role: PI

    Startup funds from Texas A&M University
    Upon joining the university I was given a generous startup package which I have used to establish my new lab and support the ongoing work. I will use these funds to bridge any gap in NIH funding and to provide for completion of the research project of Robert Louis Hastings.

Pending Research Support
none outstanding although an application on aging is planned for the next NIH deadline
BIOGRAPHICAL SKETCH

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

NAME: Evelyn Tiffany-Castiglioni
Assoc. Dean for Undergraduate Education
eRA COMMONS USER NAME: ecastiglioni

EDUCATION/TRAINING: (Begin with baccalaureate or other initial professional education, such as nursing, and include postdoctoral training.)

<table>
<thead>
<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE</th>
<th>YEAR(s)</th>
<th>FIELD OF STUDY</th>
</tr>
</thead>
<tbody>
<tr>
<td>University of Texas -El Paso</td>
<td>B.S.</td>
<td>1975</td>
<td>Biology</td>
</tr>
</tbody>
</table>

Personal Statement. My laboratory carries out research on mechanisms of neurotoxicity of environmental contaminants, including metals and pesticides. I am the editor and co-author of In Vitro Toxicology: Principles and Challenges (Humana Press, 2004), which is being revised and updated currently for release in 2018 (Springer). I have served as major professor for 18 graduate students in toxicology and neuroscience, most of whom are now productive scientists in academia, government, or industry.

A. Positions and Honors

Positions and Employment:
1982-1987 Assistant Professor, Texas A&M University
1987-1994 Associate Professor, Texas A&M University
1989-1990 Visiting Associate, Professor University of Texas Health Science Center, San Antonio
1990-present Faculty of Neuroscience and Faculty of Toxicology, Texas A&M University
1994-present Professor, Texas A&M University
1996-1998 Asst. Dean for Undergraduate Education, Texas A&M University
1998-present Assoc. Dean for Undergraduate Education, Texas A&M University
1998-1999 Interim Department Head, Texas A&M University, Vet. Anatomy & Public Health
1999-present Department Head, Texas A&M University, Vet. Integrative Biosciences (dept. renamed 2004)

Other Experience and Professional Service (last 6 years):
Editorial Board of International Journal of Developmental Neuroscience, 2000-present
Associate Editor, Neurotoxicology, 2004-present
National Science Foundation review panel for Graduate Research Fellowships Program, 2014
NIOSH Disease, Disability and Injury Prevention and Control Special Emphasis Panel ZOH1 EEO (50), teleconference meeting, March 5, 2013.
NIOSH SOH Study Section ad hoc member, 1 or 2 times per year, 2012-2017.
NORA Study Section ad hoc member, 1 time per year, 2012-2017.

B. 15 Selected peer-reviewed publications (from 84 peer-reviewed publications and 17 book chapters)


Tiffany-Castiglioni, E. et al., Kohl and surma eye cosmetics as significant sources of lead (Pb) exposure. Journal of Local & Global Health Science vol. 1. 2012. DOI: 10.5339/jlghs.2012.1


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: Leonide Gerard Toussaint III, M.D.

POSITION TITLE: Associate Professor

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

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.)

<table>
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<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
<th>Completion Date</th>
<th>FIELD OF STUDY</th>
</tr>
</thead>
<tbody>
<tr>
<td>University of North Carolina at Chapel Hill</td>
<td>BS</td>
<td>1994</td>
<td>Mathematics</td>
</tr>
<tr>
<td>University of North Carolina School of Medicine</td>
<td>MD</td>
<td>2000</td>
<td>Medicine</td>
</tr>
<tr>
<td>Mayo Graduate School of Medical Education</td>
<td></td>
<td>2000-2007</td>
<td>Neurosurgery</td>
</tr>
</tbody>
</table>

A. PERSONAL STATEMENT

This research proposal takes advantage of the collaboration of experts in epileptogenesis and brain tumor biology. As a researcher and practicing neurosurgeon, I have been curious about why some of my patients with brain tumors develop seizures, while others do not. This study will give us an opportunity to address this unanswered question. My colleagues, Drs. Wu and Reddy, will provide the needed expertise in epilepsy. My access to human glioma samples, experience with xenograft brain tumor models, and clinically-driven research focus are likely to make this project successful.

More significantly, I look forward to enhancing the lives of my future patients.

B. POSITIONS AND HONORS

July 2004 – June 2006: Clinician Investigator Training Program participant
July 2004 – June 2006: Fellow, Division of Neuro-oncology, Mayo Clinic, Rochester, MN
June 2006 – June 2007: Chief Resident, Dept of Neurological Surgery, Mayo Clinic, Rochester, MN
July 2007 – June 2015: Assistant Professor, Department of Neuroscience and Experimental Therapeutics, Texas A&M Health Science Center College of Medicine.
July 2007 – Current: Neurosurgeon, The Texas Brain and Spine Institute, Bryan/College Station, TX
June 2015 – Current: Associate Professor, Department of Neuroscience and Experimental Therapeutics, Texas A&M Health Science Center College of Medicine.

Professional Memberships:

2000- Congress of Neurological Surgeons
2000- American Association of Neurological Surgeons
2002- AANS/CNS Section on Tumors
2004- Society for Neuro-Oncology
2005-07 American Association for Cancer Research

Texas A&M Institute for Neuroscience
Honors:
1996  Holderness Research Fellowship
1998  Distinguished Medical Scholars Program
1998-00  Associate Editor, Fax, UNC School of Medicine Student Research Journal
2003  Clinical Investigator Training Program Award Recipient, Mayo Foundation
2006  Mayo Clinic Department of Neurology Basic Science Research Award
2007  American Academy of Neurological Surgeons Paper Award Runner-Up

C. Contributions to Science

Contribution #1
In my work as a neurosurgeon, I have been disappointed by my inability to cure patients with high grade glioma. Invasion of the normal brain parenchyma by tumor cells moving away from the core of the lesion provides a major source of recurrence. These cells typically are not seen by the surgeon and are more resistant to adjuvant therapies than those that comprise the bulk of the cancer. Early in my residency, I aimed to discover the molecular signature of those invasive cells at the tumor periphery. In a paper published with our research team at Mayo Clinic, I helped to establish the benefit of using nude mouse xenograft models of glioblastoma – they provide an accurate molecular recapitulation of the human disease. In addition, we shared the usefulness of an anti-human vimentin antibody in staining U87 cells present anywhere in the nude mouse brain. Finally, and most importantly, we confirmed the role of galectin-1 in promoting the invasion and motility of high grade glioma.


Contribution #2
After contributing to the molecular definition of invasive glioblastoma cells using standard expression profiling techniques, I turned my attention to newly discovered noncoding RNAs. The role of micro-RNA in mediating the invasion of glioblastoma was not completely understood in 2007, when I joined the faculty at Texas A&M Health Science Center College of Medicine. Along with a strong laboratory group, I began an inquiry that led to identification of miR-143 and miR-145 as mediators of glioma invasion. Along the way, we designed and published a cheap, reproducible, and reliable method of selecting cells from glioma cell lines with the most invasive potential. Our identification of miR-143 and miR-145 as mediators of invasion is both supported by and refuted by the experience of other laboratories working in glioma and other solid tumors. The field of molecular oncology has not reached consensus on these molecules, and we contend that the effect of expression of this joint locus (miR-143/145 arise from the same transcript) is dependent on tumor type and possibly even tumor sub-type as well as prior therapy, and that this expression relates to patient outcome.


Contribution #3
During my residency training, I joined a research group interested in challenging dogma in ruptured aneurysm treatment. Through careful and voluminous data extraction, we generated a robust base from which we could answer important questions regarding our patients. Each investigator on the team championed his questions of interest, and the result was a series of highly-cited publications in our field. At the time, the general consensus on patients who suffered a cardiac arrest due to aneurysmal subarachnoid hemorrhage was that their outcome was universally horrible. In my review, we found survivors who not only made it out of the hospital, but some who returned to work.
Another unsettled question in cerebral aneurysm patients was whether to continue or to stop aspirin use after the discovery of a cerebral aneurysm. By examining the outcomes of patients who used aspirin prior to their subarachnoid hemorrhage and comparing their results to patients not on aspirin, we discovered two balancing trends. First, there was a slightly increased risk of re-hemorrhage for those on aspirin. Yet, the patients who used aspirin prior to subarachnoid hemorrhage suffered fewer permanent deficits/strokes from delayed vasospasm. This result bolstered investigators in Europe who were beginning a randomized trial of aspirin and magnesium after aneurysmal subarachnoid hemorrhage.

Other papers from our group 1) defined rates of pulmonary complications in these patients, 2) supported the low risk of early ventriculostomy, 3) offered a new classification system for vasospasm risk, 4) examined the efficacy and complication rates in coil embolization, among others.

Contribution #4

My collaboration with basic scientists at Texas A&M University College of Medicine proved fruitful in unraveling a new signaling pathway mediating glioblastoma invasion. The role of NF-kB transcription factors has been established in many tumor types, including glioblastoma, but the prevailing thought was that their canonical signaling pathway was responsible for their downstream effects. Our research team described the importance of NF-kB inducing kinase (NIK) signaling through a novel pathway in promoting glioblastoma invasion. This pathway is likely dependent on a pool of NIK protein in the mitochondria that was heretofore uncharacterized. In a series of two articles, we were able to establish NIK as an emerging target of interest for future research and novel therapies.


Complete List of Published Work in MyBibliography:


(sic.)
D. RESEARCH SUPPORT

The role of perlecan domain V in the angiogenic and invasive biology of glioblastoma  
Principal Investigators: Gregory Bix, MD, PhD, and L. Gerard Toussaint III, MD  
Funding: Texas Brain and Spine Institute Seed Grant  
Direct Costs: $15,000  
Award Dates: March 2011-March 2012.  
Role: Co-PI

Glioblastoma Invasion, Motility, Stem Cells, and Vascular Biology  
Principal Investigator: L. Gerard Toussaint III, MD  
Funding: St. Joseph Foundation, Bryan, Texas  
Direct Costs: $350,000  
Award Dates: June 2008 – Present, renewable  
Role: PI

Convection-Enhanced Delivery of Novel Therapeutics in Glioma Models  
Principal Investigators: L. Gerard Toussaint III, MD, Joon H. Uhm, M.D.  
Funding: American Brain Tumor Association Program Project Grant  
Direct Costs: $50,000  
Role: Co-Principal Investigator

Pyk2 as a target for therapeutics in GBM.  
Principal Investigators: Joseph C. Loftus, Ph.D., Joon H. Uhm, M.D., Christopher A. Lipinski, M.D.  
Funding: Mayo Clinic NCI (National Cancer Institute) SPORE (Special Projects of Research Excellence) P50, Project #3 CA108961  
Role: Co-investigator  
Direct Costs: $2,000,000 total budget  

Differential regional expression of markers of glioblastoma invasion and motility–tumor core versus invasive periphery  
Principal Investigators: C. David James, Ph.D. and Joon H. Uhm, M.D.  
Funding: Sontag Foundation, Mayo Clinic Clinician Investigator Training Program, NIH LRP, NIH Neuro-Oncology Training Grant NRSA 5 T32 NS007494-02  
Direct Costs: $500,000  
Award Dates: 2003-2006  
Role: Research Fellow

Aneurysmal Subarachnoid Hemorrhage, the Mayo Clinic Experience: modern outcome data, risks, and treatment alternatives.  
Funding: No external funding  
Direct Costs: $0  
Award Dates: 2000-2003  

Principle Investigators: Andrienne D. Cox, Ph.D. and Channing J. Der, Ph.D.  
Funding: UNC Distinguished Medical Scholars Program  
Direct Costs: $0, Tuition and Expenses Stipend award
Extracellular Matrix Changes in Failed Osteoarticular Allografts
Principle Investigator: Gayle E. Lester, Ph.D. and Gary D. Bos, M.D.
Funding: The Holderness Medical Research Fellowship and the NIH Short-Term Research Award
Direct Costs: $5000
Award Dates: 1996-1998

Identification of Novel Oncogenes in Human Brain Tumor Samples
Principle Investigator: Joseph R. Moskal, Ph.D.
Funding: The Falk Foundation and the Chicago Institute of Neurosurgery and Neuroresearch
Direct Costs: unknown
Award Dates: 1996
Role: Research Assistant, Summer 1996

Alterations in Rabbit Hippocampal Protein Expression Following Trace Conditioning Exercises
Principle Investigator: Joseph R. Moskal, Ph.D. and John F. Disterhof, Ph.D.
Funding: John Motley Morehead Scholarship and the Chicago Institute of Neurosurgery and Neuroresearch
Direct Costs: Expense stipend for summer research
Award Dates: summer 1993
Role: Research Assistant, undergraduate intern 1993.
BIOGRAPHICAL SKETCH

NAME: Jyotsna Vaid

POSITION TITLE: Professor of Psychology and Director of Organizational Development, Research and Equity, Office for Diversity, Texas A&M University

EDUCATION/TRAINING

<table>
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<th>DEGREE</th>
<th>Completion</th>
<th>FIELD OF STUDY</th>
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<tr>
<td>Vassar College</td>
<td>B.A.</td>
<td>1976</td>
<td>Biopsychology</td>
</tr>
<tr>
<td>McGill University</td>
<td>M.A.</td>
<td>1978</td>
<td>Experimental Psychology</td>
</tr>
<tr>
<td>McGill University</td>
<td>Ph.D.</td>
<td>1982</td>
<td>Experimental Psychology</td>
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</table>

A. Personal Statement

My research lies at the interface of neuropsychology, bilingualism, and cognition. I have published extensively in these areas. Over 90% of my graduate students have secured postdoctoral and/or tenure track faculty positions at research universities in the U.S. (University of Texas-Austin, University of Wisconsin) and abroad (Taiwan, Turkey, India).

B. Positions and Honors

Current Academic Positions

2015-present Director of Organizational Development, Research, and Equity, Office for Diversity
2001-present Professor of Cognition and Cognitive Neuroscience, Texas A&M University
2013-present Convenor, Diversity Science Cluster, Psychology Department, Texas A&M
2008-present Affiliated Faculty, Women’s and Gender Studies Degree Program, Texas A&M (Acting Director, 2010-11)

Editorial Experience

Editor in Chief
2009-present Writing Systems Research

Associate Editor
2017-present Journal of Cultural Cognitive Science

Editorial Board
2016-present Frontiers in Psychology (Cognition)
2014-present Journal of Neurolinguistics
2000-present Laterality: Asymmetries of Body, Brain and Cognition
Book Series Editorial Board
Psychology of Women Book Series, American Psychological Association (2014-present)
Language Play and Creativity Book Series, Mouton de Gruyter (2015-present)

Honors and Recognitions
• Elected Fellow, American Association for the Advancement of Science, Section: Psychology (2016-)
• Elected Fellow, American Psychological Association, Division 3 (Experimental), 2016-
• Elected Fellow, Association for Psychological Science, 2002-
• Elected Fellow, Society for Psychonomic Science, 2012-
• Women in Cognitive Science Mentorship Award, 2016
• Fulbright Scholar, 1985
• University Honors Teacher/Scholar Award, Texas A&M University, 2000, 2012
• Faculty Diversity Award for Outstanding Achievement, Office of Executive Vice President and Provost, Texas A&M University, 2003
• Certificate for Outstanding Service to International Studies Degree Program, Texas A&M, 2007
• Undergraduate Research Fellows Faculty Mentor Award, Texas A&M University, 2002
• Faculty Fellow, Mexican American and U.S. Latino Research Center, TAMU, 2005-2007

Selected Honors of Graduate Students Mentored
• Diversity Fellowship, Office of Graduate and Professional Studies, Texas A&M University: Karina Febre, 2017-2021; Belem Lopez, 2010-2014
• Star-Cog Graduate Research Award, Dept. of Psychology, TAMU: Omar Garcia, 2017
• American Psychological Association/Psi Chi Edwin B. Newman Graduate Research Award for Outstanding Paper: Sumeyra Tosun, 2014
• American Psychological Association Dissertation Research Award: Hsin Chin Chen, 2005; Chaitra Rao, 2008; Kayoung Kim, 2014
• Association of Former Students Distinguished Achievement Award for Excellence in Teaching, Texas A&M: Kayoung Kim, 2015
• Association of Former Students Distinguished Achievement Award for Excellence in Doctoral Research, Texas A&M: Hsin Chin Chen, 2007
• Phil Gramm Graduate Student Award: Kayoung Kim, 2007
• Murray and Celeste Fasken Distinguished Teaching Award: Kayoung Kim, 2015
• American Psychological Foundation/COGDOP Graduate Research Fellowship: Hsin Chin Chen, 2004
• Psi Chi Graduate Research Award: Sumeyra Tosun, 2011-2012

Selected Honors of Undergraduate Students Mentored
• Best Undergraduate Honors Fellows Thesis, Texas A&M: Rebecca Rhodes, 2010, Manisha Parekh, 1999
• Nicole Baxter Memorial Award for Outstanding Psychology Undergraduate, Texas A&M: Rebecca Rhodes, 2010
• Texas A&M Academic Excellence Award, Rebecca Rhodes, 2010
• Best Undergraduate Poster in Psychology, Student Research Week: Rebecca Rhodes, 2010; Estefania Lezama, 2014
• Melbern G. Glasscock Center First Prize in Humanities for Undergraduate Research, Student Research Week, Texas A&M University: Estefania Lezama, 2014
• Texas A&M Student Research Week, Rebecca Rhodes, First Prize in Psychology Research, 2010
Contributions to Science and Representative Publications

1. A primary focus of my research and on which I have published extensively concerns the factors that affect cognitive and neurocognitive processing of language by speakers of two or more languages.


2. Another line of research has examined the consequences of biomechanical and cultural influences on spatial biases in cognition, as studied in representational drawing or facial affect judgments.


3. A third area of research involves reconceptualizing the study of bilingualism. I argue that research on bilingualism needs to start with the premise that the canonical language user is multilingual. This raises new questions and reconfigures existing ones. In particular, it focuses attention on individual differences among bilingual language users.


Academic Program Review, May 2017


C. Research Support

Ongoing

2016-2017 College of Liberal Arts Salary Savings Research Grant ($11,576)
2016-2017 College of Liberal Arts International Travel Grant ($1500)
2015-2017 Professional Development Support, Office of the Vice President and Associate Provost for Diversity, Texas A&M University ($10,000)

Completed


D. Contributions to the Profession of Psychology, Teaching, and Public Understanding of Psychology

New Courses Introduced to the Curriculum:

Psychology of Language; Language and Gender; Language and Gender Across Cultures; Designing and Interpreting Research on Gender; Bilingual Minds: Gender and Race in Psychological Inquiry

Publication that Addresses the Current Status of the Psychological Profession in terms of its Gender

2016 An examination of women’s professional visibility in cognitive psychology (Vaid, J. & Geraci, L.). Feminism and Psychology.
BOOK


GUEST EDITOR FOR SPECIAL ISSUE OF JOURNAL


Refereed Journal Articles (student co-authors are indicated with an asterisk)


**Book Chapters**


BIOGRAPHICAL SKETCH

Provide the following information for the Senior/key personnel and other significant contributors in the order listed on Form Page 2. Follow this format for each person. DO NOT EXCEED FOUR PAGES.

NAME
Alice R.A. Villalobos

POSITION TITLE
Adjunct Assistant Professor

Department of Veterinary Integrative Biosciences
College of Veterinary Medicine & Biomedical Sciences
Texas A&M University

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

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

<table>
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<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE</th>
<th>MM/YY</th>
<th>FIELD OF STUDY</th>
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<td>Loyola Marymount University, Los Angeles, CA</td>
<td>B.S.</td>
<td>05/1986</td>
<td>Biology</td>
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<tr>
<td>The University of Arizona, Tucson, AZ</td>
<td>Ph.D.</td>
<td>12/1993</td>
<td>Comparative Physiology</td>
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<td>NIH-National Institute of Environmental Health Sciences, Research Triangle Park, NC</td>
<td>Post-Doctoral</td>
<td>09/1997</td>
<td>Transport of Xenobiotics</td>
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<tr>
<td>University of Connecticut - Storrs, CT</td>
<td>Post-Doctoral</td>
<td>07/2000</td>
<td>Transport of Nutrients and Xenobiotics</td>
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</table>

A. Personal Statement

My research addresses two fundamental questions, How are nutrients, xenobiotics, drugs and metabolites transported into and out of the brain? and How does transport adapt to cellular stress induced by exposure to pathophysiological and physicochemical stressors? I study how the choroid plexus transports bioactive molecules into and out of the brain at the interface of the blood and cerebrospinal fluid (CSF), i.e., the blood-CSF barrier. Choroid plexus is often called 'the kidney of the brain', because its cellular morphology and physiology are similar to those of renal proximal tubule. As the epithelium responds to physicochemical stressors originating in peripheral and central milieus, solute transport into or out of CSF may be impaired or stimulated, thereby altering nutrient, drug, or metabolite levels in the brain. Using a primary culture system for rat choroid plexus and isolated choroid plexus of spiny dogfish shark (S. acanthias) as experimental models, we are characterizing modulation of solute transport in the context of cellular stress responses to hyperthermia, cadmium exposure and zinc deficiency. We elucidate modulation of transport in the context of the cellular stress response, characterizing cytoprotective mechanisms, and regulation by select signaling pathways and stress genes. We have characterized modulation of transport of choline (precursor to the neurotransmitter acetylcholine) as the choroid plexus adapts to cadmium-induced oxidative stress and how zinc protects against oxidative stress and modulation of transport. Although choroid plexus normally accumulates zinc, mechanisms of zinc transport remain unclear. We have constructed a working model for zinc transport and are elucidating regulation by regulated in zinc deficiency vs. supplementation. In the shark model, we have characterized organic anion transport across the intact blood-CSF barrier in response to hyoperthermia and cytoprotection by Hsp70.

B. Positions and Honors

Positions

1987-1993 Graduate Teaching Assistant, Department of Physiology, College of Medicine
      The University of Arizona

1993-1996 Intramural Research Training Fellow, Laboratory of Cellular and Molecular Pharmacology
      Comparative Membrane Pharmacology Section
      NIH-National Institute of Environmental Health Sciences

1996-2000 Postdoctoral Trainee and NIH-NINDS Individual Trainee Fellow
      Department of Physiology & Neurobiology
      University of Connecticut - Storrs, CT
2000-2006 Assistant Professor, Department of Environmental Medicine
University of Rochester School of Medicine and Dentistry

2007-2014 Assistant Professor, Department of Nutrition & Food Science
Texas A&M University

2007 - 2014 Intercollegiate Faculty of Nutrition

2007 - 2014 Intercollegiate Faculty of Food Science

2007 - present Intercollegiate Faculty of Toxicology

2010 - present Adjunct Faculty, Department of Veterinary Integrative Biosciences

2011 - present Texas A&M Institute for Neuroscience

Professional Memberships
1992 - present American Physiological Society
2002 - present Society of Toxicology
2007 - present Lone Star Chapter of Society of Toxicology
2012 - present Society for Free Radical Biology and Medicine
2014 - present Human Anatomy & Physiology Society

C. Peer-reviewed Publications.


D. Research Support

**Completed Research Support**

**Texas AgriLife Research - USDA-CRIS**
Project #H-9232
Title: *Physiology and Stress Biology of Nutrient Transport in Choroid Plexus*
Role: Principal Investigator

- Texas AgriLife Research - USDA-CRIS
- Project #H-9232
- Title: Physiology and Stress Biology of Nutrient Transport in Choroid Plexus
- Role: Principal Investigator

**National Science Foundation**

**Biological Sciences-Division of Integrative Organismal Systems (IOS)**
Research Grant #IOS-1052654
Title: *Role of Zinc in Stress Regulation of Organic Solute Transport in Choroid Plexus*
Role: Principal Investigator

- National Science Foundation
- Biological Sciences-Division of Integrative Organismal Systems (IOS)
- Research Grant #IOS-1052654
- Title: Role of Zinc in Stress Regulation of Organic Solute Transport in Choroid Plexus
- Role: Principal Investigator

**Office of the Vice President of Research, Texas A&M University**
Program to Enhance Creative and Scholarly Activities (PESCA)
Title: *Effects of Nutrient Status on Zinc Transport in Choroid Plexus*
Role: Principal Investigator

- Office of the Vice President of Research, Texas A&M University
- Program to Enhance Creative and Scholarly Activities (PESCA)
- Title: Effects of Nutrient Status on Zinc Transport in Choroid Plexus
- Role: Principal Investigator

**National Science Foundation**
Division of Integrative Biology & Neuroscience (IBN)
Grant #IBN-9808616
Title: *Characterization of Choline Transport by Choroid Plexus*
Role: Principal Investigator

- National Science Foundation
- Division of Integrative Biology & Neuroscience (IBN)
- Grant #IBN-9808616
- Title: Characterization of Choline Transport by Choroid Plexus
- Role: Principal Investigator

**NIH-National Institute of Neurological Disorders and Stroke**
Award #R01-NS39452
Title: *Stress Modulation of Choline Transport by Choroid Plexus*
Role: Principal Investigator

- NIH-National Institute of Neurological Disorders and Stroke
- Award #R01-NS39452
- Title: Stress Modulation of Choline Transport by Choroid Plexus
- Role: Principal Investigator

**NIH-National Institute of Environmental Health Sciences**
Award #R01-ES10439
Title: *Modulation of Choroid Plexus Choline Transport by Metals*
Role: Principal Investigator

- NIH-National Institute of Environmental Health Sciences
- Award #R01-ES10439
- Title: Modulation of Choroid Plexus Choline Transport by Metals
- Role: Principal Investigator
NAME: Wang, Jun

eRA COMMONS USER NAME (credential, e.g., agency login): jwang188

POSITION TITLE: Assistant Professor of Neuroscience

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.)

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<th>DEGREE (if applicable)</th>
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<th>FIELD OF STUDY</th>
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<tr>
<td>Tongji Medical University, China</td>
<td>M.D.</td>
<td>06/1993</td>
<td>Clinical Medicine</td>
</tr>
<tr>
<td>Tongji Medical University, China</td>
<td>M.NS.</td>
<td>06/1996</td>
<td>Neurobiology</td>
</tr>
<tr>
<td>Shanghai Brain Research Institute, Chinese Academy of Science, China</td>
<td>Ph.D.</td>
<td>08/1999</td>
<td>Neurobiology</td>
</tr>
<tr>
<td>University of California, Berkeley with Dr. Robert S. Zucker</td>
<td>Postdoctoral</td>
<td>8/2003</td>
<td>Neurobiology</td>
</tr>
<tr>
<td>The J. David Gladstone Institutes with Drs. Lennart Mucke and Steven M. Finkbeiner</td>
<td>Postdoctoral</td>
<td>9/2004</td>
<td>Neurobiology</td>
</tr>
</tbody>
</table>

A. Personal Statement

My research training concentrated on using patch-clamp electrophysiology, neuropharmacology, and neuropsychiatric disease models to understand the synaptic and neural circuit basis of behaviors. I trained with Dr. Robert S. Zucker at University of California, Berkeley. As an electrophysiologist, I use a combination of brain slices, whole-cell recording, and mouse transgenics to investigate synaptic plasticity, including long-term potentiation (LTP), in normal and disease states. Since 2004, I joined Dr. Dorit Ron’s laboratory at the Gallo Research Center and have been focusing on alcohol’s actions in the regulation of glutamatergic plasticity. I published several high-quality papers showing that alcohol causes a long-term facilitation of NMDA receptor (NMDAR)-mediated neurotransmission, which facilitates induction of LTP of AMPA receptor (AMPAR)-mediated response in the dorsal striatum (J Neurosci 2007, 2010a, 2012). In 2012, I obtained an R01 grant from the NIH/NIAAA to study mechanisms of alcohol-mediated glutamatergic alterations. In 2013, I began my own laboratory with the goal of understanding the synaptic and circuit mechanisms of alcohol addiction. Now my laboratory has established the capacity for combined electrophysiology, optogenetics, chemogenetics (also called DREADDs), and behavioral analysis. These tools allow us to study alcohol-mediated, circuit-specific synaptic plasticity in the striatum and connected brain areas, and to assess the circuit contribution to alcohol consumption.

In the proposal, we will assess how stroke alters alcohol consumption and the underlying inflammatory and circuit mechanisms. We will also use pharmacological and optogenetic approaches to reduce post-stroke alcohol intake. I have worked for 9 years at the Gallo Research Center where I gained extensive experience in training and measuring voluntary alcohol intake in rodents using the intermittent-access 2-bottle-choice drinking procedure and the operant alcohol self-administration procedure (J Neurosci 2007, 2010a, 2012, 2015). My patch-clamp recording in particular striatal and midbrain slices were trained in Dr. David Lovinger’s laboratory at the NIAAA and in Howard Fields laboratory at the Gallo Center, respectively. My initial optogenetic research benefits from communications with Dr. Antonello Bonci’s laboratory at the Gallo Center. These trainings allow me to measure dopaminergic firing in midbrain slices and glutamatergic transmission/plasticity in striatal slices (Wang et al, J Neurosci, 2007, 2010a, 2010b, 2012; Barak, Wang et al, Addict Biol 2014), as well as to conduct optogenetic studies in normal and alcohol-drinking animals. These experiences place me a unique position to study circuit mechanisms of alcohol addiction involving in both the striatum and midbrain. With regards to the stroke part, Dr. Farida Sohrabji in the same building from the same department is a well-established senior investigator in the stroke field, and her group will conduct stroke surgery and inflammation-related experiments.
This special environment places our two laboratories a perfect position to study post-stroke alcohol abuse. The collaborations between Dr. Sohrabji's and my laboratories have generated a large amount of exciting preliminary data firmly showing that stroke increases alcohol-seeking and relapse in rats (Huang et al.). Furthermore, I have completed an ABMRF foundation grant and an NIH pilot-project grant. Importantly, I currently have an R01 grant, which has led to 1 publication (J Neurosci 2015), 1 article in press (Cheng et al., Biol psychiatry), 1 submitted manuscript (Ma et al. 1), and 4 manuscripts in written (Ma et al. 2, Hellard et al., Huang et al., Wei et al.). With these experience, I feel confident to direct the whole project together with Dr. Sohrabji.


B. Positions and Honors

Positions and Employment

2004-2007  Assistant Research Scientist, Ernest Gallo Clinic and Research Center
2007-2010  Associate Research Scientist, Ernest Gallo Clinic and Research Center
2010-2011  Senior Research Scientist, Ernest Gallo Clinic and Research Center
2011-2013  Staff Research Investigator, Ernest Gallo Clinic and Research Center
2011-2013  Adjunct Assistant Professor, Department of Neurology, University of California, San Francisco
2013-       Assistant Professor, Department of Neuroscience and Experimental Therapeutics, College of Medicine, Texas A&M University Health Science Center

Other Experience and Professional Memberships

2001-       Membership, Society for Neuroscience
2002-2006  Membership, Biophysics Society
2005-2006,  Membership, Research Society on Alcoholism
2009-

Honors

1997       First Award of Science and Technology Achievement, Department of Health, Hubei, China
1998       Di-Ao Award, Chinese Academy of Sciences, China

C. Contribution to Science

1. Alcohol facilitation of NMDAR function. Alcohol has long been known to inhibit NMDAR activity, which suppresses long-term potentiation (LTP) and memory formation. In addition, alcohol addiction is considered enhanced learning and memory which is presumably associated with increased NMDAR activity. How alcohol chronically modulates NMDAR activity is poorly understood. I found that, 1) while NMDAR activity is inhibited in the presence of alcohol (J Neurosci 2007, 2010a, 2012 and 2013; Channels 2011), the activity is persistently facilitated after alcohol is washed out in striatal slices (J Neurosci 2007) or metabolized in vivo (J Neurosci 2010a), 2) this long-term facilitation is mediated by GluN2B-, but not GluN2A-contining (J Neurosci 2007, 2010a; Channels 2011) NMDARs, and requires activation of the Src family kinase Fyn and protein tyrosine phosphatase α (PTPα) (J Neurosci 2007, 2013), and 3) this facilitation is predominantly in the dorsomedial
striatum. Importantly, we also found that hat inhibition of GluN2B-NMDARs, Fyn kinase, or PTPα in the dorsal striatum attenuates alcohol intake (J Neurosci 2007, 2010b, 2013). The research clearly demonstrates two opposite regulatory roles of alcohol on NMDARs: direct inhibition and withdrawn facilitation. Importantly, the research identified potential therapeutic targets, e.g., GluN2B-NMDAR, Fyn kinase, and PTPα, for treating alcohol addiction. I designed and conducted all the electrophysiology recordings of these studies.


2. **Alcohol-evoked AMPAR- and GABAR-mediated functional plasticity and structural changes in a circuit-specific manner.** While alcohol directly targets the NMDAR channel, the channel does not primarily mediate fast synaptic transmission like AMPARs and GABAA receptors (GABA_RS). How does alcohol alter AMPAR and GABA_R activity leading to addictive behaviors? My research reveals that 1) alcohol facilitation of NMDAR activity enhances LTP of AMPAR-mediated transmission in the striatum (J Neurosci 2012), 2) Excessive alcohol intake increases AMPAR activity and the number of mushroom spines selectively in dopamine D1 receptor-expressing striatal neurons (J Neurosci 2015), and 3) excessive alcohol intake also increases GABAergic activity in D2 receptor-expressing neurons (Cheng et al.). Importantly, our chemogenetic and optogenetic studies reveal that D1-neuron inhibition or D2-neuron excitation reduces alcohol intake in mice (Cheng et al) and alcohol seeking and relapse in rats (Hellard et al.). In addition to these cell type-specific alterations, excessive alcohol intake also differentially changes corticostriatal and amygdalostral afferents into the dorsal striatum (Ma et al.). Lastly, using triple transgenic mice, we found different expression patterns of D1 and D2 receptors in the cortex and unique connections of these cortical cells with striatal D1- and D2-neurons (Wei et al.) These studies elucidate the synaptic and circuit mechanisms of alcohol addiction: alcohol upregulates D1-neuron activity and suppresses D2-neuron function together driving excessive alcohol consumption. We also provide therapeutics strategies to reduce alcohol intake by reversal these alterations. I designed and conducted all the electrophysiology recordings and structural studies published before 2015, and directed the studies listed below.


d. Wei XY, Cheng YF, Huang CY, Wang XH, and Wang J*. Transgenic mice for accessing dopamine D1 or D2 receptor-expressing neurons in the central nervous system. Manuscript in written.

3. **Alcohol and GDNF regulation of midbrain dopaminergic activity.** Both dopamine and GDNF are critical for drug and alcohol addiction, but how alcohol and GDNF regulate dopaminergic activity is unclear. My
studies indicate that 1) GDNF acutely potentiates excitatory synaptic transmission in dopamine neurons via enhancing voltage-gated calcium channel activity (Neurosignals 2003), 2) Striatum-produced endogenous GDNF is retrogradely transported to the midbrain to regulate the excitability of dopamine neurons that project back to the striatum and to the prefrontal cortex (J Neurosci 2010a), and 3) Chronic alcohol intake reduces firing activity of midbrain dopamine neurons (Addict Biol 2014). These studies reveal that in addition to the glutamatergic system, the dopaminergic system is also regulated by alcohol intake. I conducted all electrophysiology recordings, stereotaxic infusion, and immunohistochemical staining in these studies.


4. **Glutamatergic and GABAergic synaptic plasticity in the hippocampus and dorsal striatum.** My early work examined both glutamatergic and GABAergic plasticity in the hippocampus and their regulation by postsynaptic calcium. I found that elevation of postsynaptic calcium by flash photolysis induces a short-term depression of presynaptic GABA release onto CA1 neurons (J Physiol 2001) and causes mossy-fiber LTP of glutamatergic transmission in CA3 neurons (J Neurophysiol 2004). In addition, I found in a mouse model of Alzheimer’s disease that overexpression of human amyloid protein (hAPP) impairs short-term and long-term synaptic plasticity, i.e., LTP, in the dentate gyrus. I conducted all the electrophysiology and imaging studies. Recently, using dual-channel optogenetics, we found that LTP can be reliably induced in the dorsal striatum by postsynaptic depolarization that LTP induction promotes, LTD induction suppresses, persistently operant self-administration of alcohol (Ma et al.). These findings provide a therapeutics for long-lasting relief of alcohol seeking and relapse. I directed this research.


Complete List of Published Work in MyBibliography:

D. Research Support

Ongoing Research Support

R01 AA021505, NIH/NIAAA Wang (PI) 08/01/2012-07/30/2017

Ethanol and glutamatergic transmission in the dorsal striatum

This project aims to determine whether excessive ethanol intake alters glutamatergic transmission in the dorsal striatum in an afferent input-specific (corticostriatal vs amygdalostriatal) and cell type-specific (dopamine D1 vs D2 receptor-expressing medium spiny neurons, D1R- vs D2R-MSNs) manner.

Role: PI
R01 AA024659, NIH/NIAAA        Miranda (PI)        03/10/2016-02/28/2021
Prenatal microRNA Neuro-therapeutics for fetal alcohol exposure
This project aims to determine how fetal alcohol exposure alters the development of different neuronal population in the prefrontal cortex.
Role: Co-Investigator

**Complete Research Support**

Source: Texas A&M University (Intramural)        Farida Sohrabji (PI)        08/01/2014-07/31/2015
Developmental and Risk Factors for Neuro-Aging and Disease
This project aims to determine whether fetal alcohol exposure and excessive alcohol consumption leads to increased damage following stroke and increased seizure susceptibility.
Role: Co-Investigator

John P. McGovern Award, Texas Research Society on Alcoholism        08/01/2014-07/31/2015
Pharmacogenetic manipulation of dopamine D1 receptor-expressing medium spiny neurons in the dorsomedial striatum alters alcohol consumption
This project aims to determine whether pharmacogenetic manipulation of D1R-MSNs in the dorsomedial striatum alters voluntary alcohol intake in mice.
Role: Mentor. This was awarded to my Ph.D. student, Yifeng Cheng.

P50 (Pilot project) AA017072, NIH/NIAAA        Wang (PI)        05/01/2012-04/30/2013
Ethanol consumption and long-term potentiation (LTP) in the dorsal striatum
This project aimed to use dual-channel optogenetics (Channelrhopsin-2 and C1V1) determining whether excessive ethanol consumption facilitates the induction of striatal AMPAR-LTP that is induced by pairing of corticostriatal glutamatergic and nigrostriatal dopaminergic stimulation.
Role: PI

ABMRF/The Foundation for Alcohol Research        Wang (PI)        07/01/2010-06/30/2012
Ethanol-mediated facilitation of dorostraial NMDAR activity and alcohol drinking behavior
The goal of this project was to use D1R- and D2R-eGFP mice to examine the ethanol-mediated cell type-specific alteration of glutamatergic receptors in the dorsal striatum.
Role: PI
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: Wellman, Paul

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

POSITION TITLE: Professor of Psychology

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.)

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<td>California State University Bakersfield</td>
<td>BS</td>
<td>8/31/1975</td>
<td>Psychology</td>
</tr>
<tr>
<td>Iowa State University</td>
<td>Ph.D.</td>
<td>9/1/1980</td>
<td>Psychology</td>
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A. Personal Statement

The goal of the current project is to investigate the impact of social housing conditions on morphine self-administration in rats. I have a broad background and thirty-five years experience in psychopharmacology with specific expertise in cocaine, amphetamine and nicotine behavioral effects and drug-induced sensitization. In my career, I have published more than 143 original articles and research reviews. Additionally, I have developed testing protocols for rodents with regard to eating and locomotion and have published on the neurochemistry (microdialysis) of cocaine, amphetamine and ephedrine. Of direct relevance to this application are my five papers related to iv self-administration of morphine, cocaine, and phenylpropanolamine in the rat. Specifically, we reported that cocaine self-administration (SA) alters impulsive choice in rats (Mendez et al., 2010), that morphine SA may undermine recovery from spinal cord damage (Woller et al., 2012), and that maintenance on a high-fat diet impairs the reinforcing value of cocaine. I have a demonstrated record of successful and productive research projects and research expertise that will provide key support for the present project.


B. Positions and Honors

Positions and Employment:
1980-1986 Asst. Professor of Psychology, Texas A&M University
1986-1991 Assoc. Professor of Psychology, Texas A&M University
1992-present Professor of Psychology, Texas A&M University
2014-present Associate Dean for IT and Facilities, College of Liberal Arts

Other Experience and Professional Memberships:
1982-present Society for Neuroscience
1993-present Society for the Study of Ingestive Behavior
2000-present British Association for Psychopharmacology
2010 CEBRA review panel, NIDA
2011-present Program project review 2011/10 ZRG1 IFCN-H
2012-2013 Special Emphasis Review Panel (NCATS: Therapeutic uses for Existing Molecules)
2013 ZDA1 SXC-E (Cutting Edge Basic Research Review Panel)

Honors:
1986 Distinguished Teaching Award, TAMU Former Students Association
1995 Recipient of Provost’s Achievement Award for fostering diversity in faculty and graduate students
1998 Supervisor of Lance R. McMahon: Outstanding Doctoral Research Award, Texas A&M University

C. Contributions to Science

1. My early training as a neuroscientist was in the study of CNS systems that govern food intake. That work included the ventromedial hypothalamus as well as the dorsolateral tegmentum. After completing the PhD degree, I shifted my focus toward the study of adrenergic drugs that suppress appetite (e.g. amphetamine, ephedrine, and phenylpropanolamine). That work demonstrated that these drugs act to suppress appetite via activation of alpha-1 adrenergic receptors within the PVN. Another key feature of that work was that this mechanism was consistent with the manner (vasoconstriction) by which these drugs promoted hemorrhagic stroke.


2. In a series of collaborations with Dr. Jack Nation, I worked to characterize the motivational deficits associated with heavy metal exposure in rats. These studies employed conditioned taste aversion, CPP, iv drug self-administration, and microdialysis of accumbens dopamine levels.


3. Role of ghrelin in drug abuse: My background in the role of neuropeptides and transmitters in the regulation of eating behavior led me to consider the possible role of the orexigenic peptide ghrelin in the facilitation of drug abuse. My early work showed that systemic injection of ghrelin facilitated cocaine hyperlocomotion and CPP while my most recent studies have shown that antagonism of ghrelin receptors diminishes cocaine as well as nicotine reinforcement.


Complete List of Publications in MyBibliography:


D. Research Support

1R21DA017230-01A2 Wellman PJ (PI) 4/01/05-5/1/08 (Completed)
NIDA “Psychostimulants and Alpha1-Adrenergic Receptors”

This grant examined the role of alpha1 receptor subtypes (1A and 1B) in the hypophagic and locomotor stimulating actions of cocaine and amphetamine in the rat.

2R01DA013188-04A2 Wellman, P.J. (PI) 8/1/2007-7/31/2012 (Completed)
NIDA “Heavy Metal and Drug Self-Administration: Mechanisms”

This grant examined the neurochemistry of the impact of perinatal lead exposure on cocaine self-administration.
BIOGRAPHICAL SKETCH
Provide the following information for the key personnel and other significant contributors in the order listed on Form Page 2. Follow this format for each person. DO NOT EXCEED FOUR PAGES.

NAME: Gregg B. Wells, MD, PhD

POSITION: Associate Professor

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

EDUCATION/TRAINING: (Begin with baccalaureate or other initial professional education, such as nursing, and include postdoctoral training.)

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<th>DEGREE (if applicable)</th>
<th>YEAR(S)</th>
<th>FIELD OF STUDY</th>
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<tbody>
<tr>
<td>Northwestern University, Evanston, IL</td>
<td>B.A.</td>
<td>1977-1981</td>
<td>Chemistry</td>
</tr>
<tr>
<td>University of Chicago, Chicago, IL</td>
<td>M.D., Ph.D.</td>
<td>1981-1989</td>
<td>Biophysics &amp; Medicine</td>
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</table>

A. Personal Statement
My laboratory studies at several levels how protein structure contributes to normal neurophysiology and leads to neurological diseases. The unifying theme of our research projects is the structure and function of ion channels. Our research at the level of atomic structure focuses on the Cys-loop superfamily of neurotransmitter-gated ion channels and especially the family of nicotinic acetylcholine receptors in both eukaryotes and prokaryotes. Our work with cyclic nucleotide-gated channels explains how their electrophysiological function reflects both their biophysical properties and cellular context. Our study of the function of cochlear hair cells extends our ion channel research to the levels of cellular and systems neurophysiology. Explaining neurological diseases in terms of protein structure links our neuroscience research with neuropathology, my medical specialty.

B. Positions and Honors

Graduate and Post-doctoral Research Experience

Positions and Employment
1989-1991 - Residency in Anatomic Pathology, Hospital of the University of Pennsylvania
1991-1996 - Fellowship in Neuropathology, Hospital of the University of Pennsylvania
1996-1999 - Instructor in Neuropathology, Division of Neuropathology, Department of Pathology and Laboratory Medicine, University of Pennsylvania School of Medicine
1999-2005 - Assistant Professor, Department of Pathology and Laboratory Medicine, College of Medicine, Texas A&M University System Health Science Center
2006-2008 - Assistant Professor, Department of Molecular and Cellular Medicine, College of Medicine, Texas A&M University System Health Science Center
2006-2008 - Joint Assistant Professor, Department of Veterinary Pathobiology, College of Veterinary Medicine & Biomedical Sciences, Texas A&M University
2008–2010 - Associate Professor, Department of Molecular and Cellular Medicine, College of Medicine, Texas A&M University System Health Science Center
2008–2010 - Joint Associate Professor, Department of Neuroscience and Experimental Therapeutics, College of Medicine, Texas A&M University System Health Science Center
2008–2010 - Joint Associate Professor, Department of Veterinary Pathobiology, College of Veterinary Medicine & Biomedical Sciences, Texas A&M University

Professional Certifications
1994: Diplomate in Anatomic Pathology of the American Board of Pathology

Texas A&M Institute for Neuroscience
1995: Special Certification in Neuropathology by the American Board of Pathology

Honors and Awards
1981 Phi Beta Kappa, Northwestern University
1987 Harold E. Lamport Award (best poster by a graduate student), Annual Meeting, Biophysical Society
1988 Bernard Smaller Prize in Magnetic Resonance, University of Chicago
1988 Marc Perry Galler Prize for most distinguished dissertation during the academic year in the Division of Biological Sciences, University of Chicago
1989 Sigma Xi, scientific honor society

C. Selected peer-reviewed publications


Person AM and Wells GB (2011), "Characterizing low affinity epibatidine binding to α4β2 nicotinic acetylcholine receptors with ligand depletion and nonspecific binding. BMC Biophysics 4:19.


C. Current and Completed Research Support

Completed Research Support

2 R01 DC003896 (PI: Anthony Ricci, Stanford University) 1/1/2008–12/31/2013
NIH/National Institute on Deafness and Other Communication Disorders
Title: Calcium Regulation of Mechanotransduction
The objective of this project is to answer questions how the mechanoelectric transducer (MET) channel, MET current, and calcium ions contribution to the function of cochlear hair cells.
Role: Co-investigator

1 R01 GM088670 (PI: René Anand, Ohio State University) 8/10/2009–7/31/2013
NIH/National Institute of General Medical Sciences; Exceptional, Unconventional Research Enabling Knowledge Acceleration (EUREKA) award
Title of Project: Fish Electric Organ as a Factory for Membrane Proteins
The goal of this project is to determine whether the electrocyte from fish electric organ, a cell type which produces large amounts of endogenous membrane proteins, is a good candidate as the foundation for a novel heterologous protein expression system producing large amounts of correctly folded human membrane proteins.
Role: Co-investigator
**NAME:** C. Jane Welsh

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

**POSITION TITLE:** Professor and Associate Department Head and Assistant Dean for Graduate Studies

**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.)*

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<tr>
<td>University of London, U.K.</td>
<td>B.Sc.</td>
<td>06/1976</td>
<td>Microbiology</td>
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<tr>
<td>King’s College Hospital, U.K.</td>
<td>Postdoc</td>
<td>1979-1981</td>
<td>Autoimmune liver</td>
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<td>Dept. of Pathology, Cambridge, U.K.</td>
<td>Postdoc</td>
<td>1982-1985</td>
<td>Rheumatoid arthritis</td>
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<td>Dept. of Pathology, Cambridge, U.K.</td>
<td>Postdoc</td>
<td>1985-1989</td>
<td>Multiple sclerosis</td>
</tr>
</tbody>
</table>

**A. Personal Statement**

My training as a microbiologist-immunologist and later training in neuroimmunology has provided a strong scientific background for tackling multi-disciplinary research projects such as the current proposal. The long-term goal of my research is to understand the pathogenesis of viral infections on the development of neurological and autoimmune conditions. To this end, I have been intensively investigating the Theiler’s virus-induced demyelination (TVID) model of multiple sclerosis (MS) since 1985. We have studied the immune response to Theiler’s virus (TMEV); the blood-brain barrier; role of stress in TVID; mechanisms of therapeutic actions of estrogens. I have served as PI and Co-PI on NIH funded grants to investigate the role of stress in the development of TVID which has provided experience in multi PI collaborations. Recently, I have taken on a number of leadership positions in the university: chairing the Texas Institute for Neuroscience (TAMIN) (http://tamin.tamu.edu); associate department head in Veterinary Integrative Biosciences and assistant dean for graduate studies in the College of Veterinary Medicine.

**B. Positions and Honors**

**Positions and Employment**

- 1988-1989 Special Supervisor in Pathology, Newnham College, Cambridge University
- 1989-present Visiting Assistant Professor (1989-1991), Assistant Professor (1991-2000); Associate Professor (2000-2006), Professor (2006-present) Dept. of Veterinary Integrative Biosciences and Dept. of Veterinary Pathobiology, College of Veterinary Medicine and Biomedical Sciences, Texas A&M University
- 1991-present Member of the Faculty of Neuroscience and Graduate Faculty, Texas A&M University
- 1998-present Member of the Genetics Faculty, Biotechnology Faculty, Texas A&M University
- 2002-present Departmental Graduate Advisor
- 2006-present Associate Department Head, Dept. Veterinary Integrative Biosciences
- 2007-present Joint appointments in the Dept. Neuroscience and Experimental Therapeutics, College of Medicine, Texas A&M Health Science Center and Dept. Psychology, Texas A&M University
- 2011-present Chair of the Texas A&M Institute for Neuroscience
- 2011-present Assistant Dean for Graduate Studies, College of Veterinary Medicine

Texas A&M Institute for Neuroscience
Other Experience and Professional Memberships

**External Reviewer**

- Alzheimer’s Association Grant Reviewer
- Biotechnology and Biological Sciences Research Council, UK
- NIH Brain Disorders and Clinical Neuroscience Special Emphasis Panel (ZRG1-NMB)
- NSF Fellowship Review Panel, NMSS Pilot Grant Reviewer
- 2006 NIH Brain Disorders and Clinical Neuroscience Special Emphasis Panel
- 2007, 2008, 2009 American Heart Association Grant Review Panel
- 2009 NIH Clinical Neuroimmunology and Brain Tumor Grant Review Panel
- 2010 NSF Grant Reviewer
- 2011 NIH P50 Reviewer
- 2013 Fast Forward MS Grant reviewer
- 2013 Italian Multiple Sclerosis Society
- 2015 P01 NINDS


**Awards**

- 2010 Texas A&M University’s Women’s Progress Award for faculty
- 2011 Texas A&M University’s Women’s Faculty Outstanding Mentoring Award
- 2012 Texas A&M University’s Association of Former Students Distinguished Achievement Award for Graduate Mentoring

**C. Contribution to Science**

1. My scientific career has been devoted to the study of autoimmunity from my Ph.D. work on (a) ankylosing spondylitis and uveitis and cross reactivity with enterobacteriaceae; (b) postdoctoral work with Dr. I. MacFarlane developing a monoclonal antibody to a target a liver autoantigen; (c) with Professor R.R.A. Coombs on characterizing the pathogenesis of rheumatoid arthritis in a naturalistic model; (d) with Professor Tony Nash on characterizing the immune response to Theiler’s virus and it’s role in demyelination.


2. Since coming to Texas A&M University in 1989, my research has been focused on understanding the pathogenesis of Theiler’s virus infection as a model of multiple sclerosis and neurological disorders (encephalitis, epilepsy); the role of the blood-brain barrier in TVID; the impact of different stressors on the development of demyelination, (in collaboration with Dr. Mary Meagher). More recently we have collaborated with Drs. Li and Steelman on Galectin 9 in EAE; with Dr. Brinkmeyer on the effect of environmental toxins on the development of virus induced demyelination; and with Dr. Levine we are investigating the role of the immune system in naturally occurring spinal cord injury in dogs.


Complete List of Published Work in MyBibliography:
http://www.ncbi.nlm.nih.gov/sites/myncbi/1jg1eRhaWeQY/bibliography/45906157/public/?sort=date&direction=ascending

D. Research Support

Ongoing Research Support

Effect of estrogen on the neuropathogenesis of Theiler’s virus infection
2013-17 Programmatic Development Award from Texas A&M Health Science Center
Goals: The goal of this project is to test estrogens in the treatment of virus-induced MS
Role: PI

Stat3 in myeloid cells: a regulator of autoimmune demyelination
National Multiple Sclerosis Society 04/01/2016- 3/31/19
PI: Jianrong Li
Role: Co-investigator

Membership of Training grant
Comparative Biomedical Research Training for Veterinarians NIH-NRSA Institutional Research Training Grant NIH (2 T32 OD011083-06), Institutional Training Grant for Comparative Biomedical Research Training for Veterinarians. 7/18/2015-3/31/20
PI: Dr. Ann Kier
Role: Training faculty

Proposal Submissions – pending (2016)
None

Completed Research
NIH/NINDS R01 NS060822 Meagher (PI) 12/01/2007-1/30/14
(includes two year no-cost extension)
Impact of stress-induced cytokines on an animal model of MS
Goals: The goal of this project is to identify the role of cytokines in mediating the adverse effects of social stress on Theiler’s virus infection.
Role: Co-PI

Texas A&M Institute for Neuroscience
2014-15 Peptide therapies for neurological diseases
VG Scientific
Goals: The goal of this project is to test novel peptides in the treatment of virus-induced epilepsy and MS
Role: PI

2014 TAMU One Health Initiative - Chronic Diseases and Conditions – PI: Tom Welsh, Optimizing One’s Health: Genetic and Environmental Regulation of Metabolic Health
Role: Co-investigator

2014 TAMU One Health Initiative Accessible & Affordable Health Care – PI: Arum Han, Electrical Engineering
Development of Next Generation Biologics through Microphysiological Systems
Role: Co-investigator

Comparative Biomedical Research Training for Veterinarians NIH-NRSA
Institutional Research Training Grant T32 Role Mentor 07/01/2011 – 2015
PI: Dr. Ann Kier

Ileal bacterial community as a target for multiple sclerosis treatment
Role: Co-PI
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:** Teresa Wilcox

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

**POSITION TITLE:** 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.*)

<table>
<thead>
<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
<th>Completion Date MM/YYYY</th>
<th>FIELD OF STUDY</th>
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<tr>
<td>Bethel University, St. Paul, MN</td>
<td>B.A.</td>
<td>1983</td>
<td>Psychology &amp; Education</td>
</tr>
<tr>
<td>University of California, Davis</td>
<td>M.S.</td>
<td>1988</td>
<td>Child Development</td>
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<tr>
<td>University of Arizona, Tucson</td>
<td>Ph.D.</td>
<td>1993</td>
<td>Psychology</td>
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<tr>
<td>University of Illinois, Champaign</td>
<td></td>
<td></td>
<td>Postdoctoral Fellow</td>
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</table>

**A. PERSONAL STATEMENT:**

I have the expertise, leadership, training and motivation to successfully carry out the proposed research project. I have extensive experience using eye-tracking, fNIRS, and manual behavior assessments to study the acquisition of object knowledge in infancy. This work has had a significant impact on the way we conceptualize the origins and development of object processing capacities during the first year. The motivation for current approach is to identify cognitive processes and cortical networks that support and facilitate infants' emerging capacity to individuate objects. As PI on several university-, NSF-, and NIH-funded grants, I laid the groundwork for the proposed research by developing behavioral measures to assess object individuation in the infant, identifying select experiences that can alter infants' capacity to individuate-by-feature, and fNIRS protocols that can be successfully used to assess functional organization of the infant brain. My ongoing collaboration with David Boas, an expert in the field of optical imaging, has uniquely positioned me to successfully integrate fNIRS into a multi-method approach to investigate knowledge acquisition in the infant. Our most current findings have led us to identify, for the first time, a network of structures in ventral and dorsal cortical areas that mediate object processing in the infant and how these structures change, functionally, during the first year. This work puts us at the forefront of the field of developmental cognitive neuroscience and makes us uniquely qualified to conduct the proposed research. My current application builds logically on my prior work.

B. POSITIONS AND HONORS:

Positions:

1993-1995  Postdoctoral Research Associate (Mentor: Renée Baillargeon)
Department of Psychology, University of Illinois
1995-2000  Assistant Professor
Department of Psychology, University of Texas at Arlington
2000-2004  Assistant Professor
Department of Psychology, Texas A&M University
2004-2011  Associate Professor
Department of Psychology, Texas A&M University
2011-2015  Professor
Department of Psychology, Texas A&M University
2015-current  Professor, Department of Psychology
Research Fellow, Division of Research
Texas A&M University

Other Experience and Professional Memberships:

Member: NSF Grant Panel 2008-2012
Associate Editor: Infant and Child Development (2015-current).
Editorial Board: Infancy (2005-2013), Frontiers in Developmental Psychology (Review Editor, current)
Organized: 2010 and 2013 fNIRS Workshop at Texas A&M University to educate faculty and students in diverse disciplines about fNIRS, to bring together potential users, and to facilitate collaborations among interested parties.

Honors:

APS Fellow

C. CONTRIBUTIONS TO SCIENCE:

1. My early publications focused on a long-standing debate in the developmental sciences: can infants represent objects, prior to language acquisition, as distinct individuals with unique characteristics, other than just “object”. My early work demonstrated, in contrast to conceptual models at the time, that infants can individuate objects on the basis of featural information and, to some extent, include that information into their object representations. However, there is a developmental hierarchy in the type of information to which infants attend, favoring form over surface property. These publications document the main findings, which significantly altered the way developmental scientists view the nature of object individuation and representation in infancy. I served as the primary, or senior, investigator in all of these studies.

2. Subsequent publications focused on the developmental hierarchy identified in my earlier experiments. More specifically, we investigated why infants are more sensitive than form than surface features and how infants go about learning that surface features are a reliable source of information about object identity. These publications document a number of experiences that lead infants to attend to surface features at a younger age than they typically do so. The outcome of these studies have been instrumental in revealing mechanisms that support knowledge acquisition in infants and the conditions under which the mechanisms operate. I served as the primary, or senior, investigator on all of these studies.

3. Much of my current work has focused on identifying the neural basis of infants’ emerging capacity to individuate objects as observed in our behavior work. Using fNIRS, we have begun to identify functional maturation of the cortical systems that support object individuation and representation in infants, and how this may (or may not) differ from the mature brain. These findings, which are document in the following publications, are the first to identify a network of structures in ventral and dorsal cortical areas that mediate infants' processing of physical, non-social objects, and how these pathways change during the first year.

3. Much of my current work has focused on identifying the neural basis of infants’ emerging capacity to individuate objects as observed in our behavior work. Using fNIRS, we have begun to identify functional maturation of the cortical systems that support object individuation and representation in infants, and how this may (or may not) differ from the mature brain. These findings, which are document in the following publications, are the first to identify a network of structures in ventral and dorsal cortical areas that mediate infants' processing of physical, non-social objects, and how these pathways change during the first year.


Ongoing Projects

NIH R01-HD468471 Wilcox (PI) 08/01/09-07/31/16 (NCE)

**Optical Imaging in Infants**

The objective of this application is to identify the functional development of the neural pathways that support infants' emerging capacity to use form (e.g., shape) and surface (e.g., color) features to track the identity of objects through space and time. The central hypothesis is that infants’ emerging capacity to individuate-by-feature will be associated with unique, well-defined patterns of neural activation in mid to higher level object processing areas. In addition, infants' use of perceptual cues, such as coherent motion, to extract object shape, will be associated with unique, well-defined patterns of neural activation in lower level object processing areas.
NAME: Ursula H. Winzer-Serhan  
eRA COMMONS USER NAME (credential, e.g., agency login): WINZERS  
POSITION TITLE: Associate 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.)

<table>
<thead>
<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
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<th>FIELD OF STUDY</th>
</tr>
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<tr>
<td>University of Florida, Gainesville, USA</td>
<td>M.S.</td>
<td>5/1986</td>
<td>Plant Physiology</td>
</tr>
<tr>
<td>University of Bremen, Germany</td>
<td>M.S.</td>
<td>10/1986</td>
<td>Biology</td>
</tr>
<tr>
<td>University of Bremen, Germany</td>
<td>Ph.D.</td>
<td>12/1999</td>
<td>Cell biology</td>
</tr>
<tr>
<td>University of California, Irvine</td>
<td>Post Doc</td>
<td>1993-97</td>
<td>Neuropharmacology</td>
</tr>
<tr>
<td>Virginia Commonwealth University</td>
<td>Post Doc</td>
<td>1997-2000</td>
<td>Neuropharmacology</td>
</tr>
</tbody>
</table>

A. Personal Statement

My long-term goal is to understand environmental factors that result in abnormal brain development, and contribute to the increase in the number of people suffering from neurological disease. For a number of years, I have been interested in the role of nicotine and how it alters brain development. Intrigued by the transient upregulation of nicotinic receptors during early postnatal development, I developed a postnatal exposure model to study the effects of nicotine during a developmental period that corresponds to the third human trimester. This time period is often ignored by others because of the challenges neonatal drug treatment represents to researchers. However, our rat neonatal model for chronic developmental nicotine exposure (DNE) revealed both short-term and long-term effects. Two long-term effects of neonatal DNE stand out: a) an increase in anxiety-like behavior, and b) an increase in excitatory neuronal transmission in the hippocampus. These results have been verified in other models but it is not clear how nicotine causes these changes. Alterations in excitatory transmission are particularly troublesome because they can cause an imbalance in the excitatory to inhibitory ratio which has been implicated as an underlying cause for neurological disorders. Furthermore, hyperexcitability of the hippocampus will have profound consequences for axonal projection target areas, which may explain the broad range of abnormal behaviors reported in children after maternal smoking. Thus, our work has shown that nicotine is an environmental factor that alters brain development. This finding has major biomedical implications with regards to smoking, e-cigarettes and nicotine-replacement therapy in pregnant women.


B. Positions and Honors

Positions and Employment
10/85-12/89 Research assistant, Gesellschaft für Biotechnologische Forschung (GBF), Germany
06/93-07/97 Postdoctoral researcher, UC Irvine, Dept of Pharmacology.
01/01-05/01 Senior Scientist, Ambion, Inc. Austin, TX.
06/01-12/05 Assistant Professor, Dept. Med. Pharm. & Tox., Texas A&M Uni. System, HSC.
01/06-08/07 Assistant Professor, Dept. Neurosci. & Experimental Therap., Texas A&M Uni. Sys., HSC.
Since 09/07 Associate Professor, Dept. Neurosci. & Experimental Therap., Texas A&M Uni. Sys., HSC.

Honors
Predoctoral Fulbright scholarship, 1984 to 1986

Other Experience and Professional Memberships
1994 Society for Neuroscience, since 1994, and
2001 Texas A&M Chapter for Neuroscience.
2001 Member of Texas A&M University Faculty of Neuroscience.
2001 Member of the Graduate Faculty at Texas A&M University, and the Health Science Center.
2012 Tobacco-Related Disease Research Program (TRDRP), University of California.
2015 TRDRP Chair of Study section Nicotine Dependence.
2009-10 Member of NIDA special emphasis panel, ZDA1 JXR-D05.
2009 Reviewing editor for Frontiers in Neuroscience, section Neuroanatomy.
2014 Editorial Board Member of Austin Journal of Neurological Disorders and Epilepsy.

Complete List of Published Work in PubMed:
http://www-ncbi-nlm-nih-gov.ezproxy.library.tamu.edu/pubmed/?term=winzer-serhan

D. Research Support

Ongoing Research Support
Optogenetic approaches to study complex neuronal circuits during cognitive aging,
Role: collaborator: 10% effort.

TAMU/HSC Research enhancement Grant:
Nicotinic modulation of Hippocampal development”.
Role: Principal investigator:

Completed Research Support
R01DA016487 PI: Winzer-Serhan 07/15/2004 to 05/31/2010
Nicotinic modulation of Hippocampal development
This grant evaluated the effects of chronic nicotine treatment during the brain growth spurt period equivalent to the third human trimester, on the expression of nAChRs and subunits, growth factors, and the effects on hippocampal GABAergic neurons.
Role: PI
STEVEN WOLTERING
Assistant Professor, Director of Neurobiological lab for Learning and Development
Department of Educational Psychology, Texas A&M University

Office: 718B Harrington; College Station, Texas, 77843; Mail Stop 4225.
Email: swolte@tamu.edu
Lab website: nld.tamu.edu

Keywords

- Neuroscience
- Self-regulation
- Intervention & training
- Emotion & motivation
- Development
- Psychopathology
- Learning
- Psychophysiology

Relevant Positions

2015 - Present  Director, Texas A&M University, USA
Neurobiological lab for Learning and Development (NLD).
Department of Educational Psychology.

2014 – Present.  Assistant professor, Texas A&M University, USA
Texas A&M University, USA
Department of Educational Psychology.

Behavioural & neuroscience lab for the study of executive function in ADHD
Supervisor: Dr. Rosemary Tannock.

Brain and Behaviour emotion regulation lab
Supervisor: Dr. Marc D Lewis.

2005 – 2007.  Research Coordinator. Sickkids Hospital, Canada
Community Health Systems Resource Group
Supervisors: Dr. Isabela Granic & Dr. Marc D Lewis.

2002 – 2005.  Faculty Research Associate. Vrije Universiteit Amsterdam, Netherlands
Department of Psychology and Education
Supervisors: Dr. Dorret Boomsma, Dr. Eco JC de Geus, Dr. Gonneke Willemsen

Degrees

Ph.D. University of Toronto, Canada. August, 2012.
Department: Human Development and Applied Psychology
Collaborative Programs: Neuroscience; Developmental Science
Supervisor: Dr. Marc D. Lewis

M.A. University of Toronto, Canada. 2007.
Department: Human Development and Applied Psychology
Supervisor: Dr. Marc D. Lewis

Department: Psychology and Education
Supervisors: Drs. Gonneke Willemsen & Eco de Geus

Teacher Education Degree
Supervisor: Drs. Chris Stronks

Publications

(Italicized names are coauthors who were graduate or undergraduate students I had supervised at that time)

Submitted


Articles in refereed journals:

Liu, Z., Tannock, R., & Woltering, S. (In press). Effects of Working Memory Training on Neural Correlates of Go/Nogo Response Control in Adults with ADHD: A Randomized Controlled Trial. *Neuropsychologia.*


**Book Chapters:**


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**Grant Writing Activities**

**Funded**

Total funded to date: $1,691,934.00

Advancing Literacy in Texas through Biometrics.

**PIs:** S. Woltering; B. Taylor; J. Liew; E. Cantrell; J. Thompson; M. Joshi.

**Source:** CEHD Catapult grant.

**Amount:** $30,000.00

**Status:** Funded, 2017 (Submitted October, 2016)

Integrating Biometric Responses to Human Behavior.
PIs: [S. Woltering & M. Palma]; L. Ribera; Y. Zhang; D. Shaw; C. Hall; N. Clemens; J. Liew; C. Eckel; H. Lench; S. Fields; A. Talebpour; J. Lahey; H. Cheng; Y. A. Hong; M. Benden.
Role: Dual PI, Co-initiator.
Source: TAMU Research Development Fund.
Amount: $1,200,000.00
Status: Funded, 2016 (Submitted December, 2015)

PIs: S. Woltering; N. Deutz.
Source: CEHD Catapult grant.
Amount: $40,000.00
Status: Funded, 2016 (Submitted October, 2015)

Neural Markers of Adolescent Obesity.
PIs: S. Woltering; S. Fields; J. Liew.
Source: CEHD Transforming Lives Grant.
Amount: $30,000.00
Status: Funded, 2015-2016 (Submitted November 2014)

Working Towards a Bio-Behavioral Center: Cyberlearning and Research Infrastructure Support.
PIs: S. Woltering; R. Gutierrez-Osuna; S. Pedersen.
Source: TAMU Program to Enhance Scholarly and Creative Activities (PESCA) Grant Program.
Amount: $25,000.00
Status: Funded, 2015-2016 (Submitted November 2014)

Working memory training in ADHD: Neural mechanisms of change.
PIs: R. Tannock; M. Lewis
Role: Graduate Student (Conceptualized grant and wrote major sections).
Source: Canadian Institutes of Health Research (CIHR).
Amount: $366,934.00
Status: Funded, 2011-2014 (Submitted September 2010)

Submitted & under review
EEG and fMRI studies with English speaking children in the US and Spanish speaking children in Mexico.
PIs: S. Woltering; A. A. González Garrido
Source: Consejo Nacional de Ciencia y Tecnología (CONACYT) Research Grant Program.
Amount: $24,000.00
Status: Submitted March, 2017

Investing in the Future of the Bio-Behavioral Lab in the College of Education.
PIs: N. Clemens; S. Woltering; J. Liew.
Role: Co-PI
Source: TAMU Lead By Example Campaign: College Funding Priorities.
Amount: $650,000.00
Status: Submitted May, 2015
Not funded

Enhancing Therapeutic Transfer of Cognitive Control in Students with Anxiety Using Wearable Biofeedback Devices.
Pls: S. Woltering
Source: David Wechsler Early Career Grant for Innovative Work in Cognition.
Amount: $25,000.00
Status: Submitted June, 2016

Neural indices of sleep quality in patients with anxiety disorder.
Pls: S. Woltering
Source: Brain Research Foundation (Fay/Frank Seed Grant).
Amount: $80,000.00
Status: Submitted, 2016

Pls: S. Chu; L. Geraci; S. Woltering
Role: Co-PI (wrote minor sections, contributed conceptually)
Source: National Science Foundation (NSF): Information and Intelligent Systems.
Amount: $500,000.00
Status: Submitted, 2015

A Connectomics Approach to Identifying Common and Separate Biomarkers of Childhood and Adult ADHD.
Pls: J. Orr; S. Woltering; J. Ji.
Role: Co-PI (wrote sections, contributed conceptually)
Source: TAMU, Seed Grant for Interdisciplinary Research in Big Data.
Amount: $50,000.00
Status: Submitted, 2015

Neural Indices of Sleep Quality on Academic and Socioemotional Functioning.
Pls: S. Woltering
Source: Brain Research Foundation (Fay/Frank Seed Grant).
Amount: $80,000.00
Status: Submitted, 2015

Reducing Stress in College Students using Biofeedback Technology.
Pls: S. Woltering
Source: Oak Ridge Associated Universities (ORAU): Ralph E. Powe Junior Faculty Enhancement Awards.
Amount: $10,000.00
Status: Submitted, 2015

Integrating Philosophy, Psychology, and Neuroscience to the Study of the Moral Self in Latino/a Young Adults and their Responses to Acts of Kindness and Injustice.
Pls: J. Liew; S. Woltering; D. Raymond.
Role: Co-PI (wrote sections, contributed conceptually)
Source: John Templeton Foundation: the self, motivation, & virtue project.
Presentations, talks, and posters

Refereed presentations


*Winner of Best paper award.


In-service presentations


Woltering, S. (2015, October). *Do Brain Training Programs Work for Students with ADHD?* Seminar talk presented for the Cognoscenti group at the Department of Psychology. Texas A&M University, USA.


---

**Teaching, mentorship, and other supervision**

*Course instruction*

EPSY634 – Educational Neuroscience. FTF. TAMU  
Semesters taught: 2016 (F)

EPSY673 – Learning Theories: Traditional and contemporary Perspectives (FTF). TAMU.  
Semesters taught: 2015 (S + F); 2016 (S)

EPSY673 – Learning Theories: Traditional and contemporary Perspectives (Online). TAMU.  
Semesters taught: 2015 (S + F); 2016 (S + F)

EPSY685 – Special Topics: Research Practicum (FTF). TAMU  
Semesters taught: 2014 (F); 2015 (S + F); 2016 (S + F)

EPSY485 – Special Topics: Research Practicum (FTF). TAMU  
Semesters taught: 2015 (S + F); 2016 (S + F)

Semesters taught: 2008 (F)

*Course development*

EPSY634 – Educational Neuroscience  
2016
Developed 100% of conceptualization and material
EPSY673 – Learning Theories 2015

Developed 70% of conceptualization and material
EDU5537 – The adolescent brain: Implications for instruction. 2008

Developed 90% of conceptualization and material

**Dissertation Committee Membership (*/ Chair/Co-chair)**

<table>
<thead>
<tr>
<th>Name</th>
<th>Degree</th>
<th>Program</th>
<th>Year Proposed</th>
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<tbody>
<tr>
<td>Zhengzheng Zhao, MS, EPSY</td>
<td></td>
<td></td>
<td>2016</td>
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<tr>
<td>*Sweta Parameshwaran, MED, EPSY</td>
<td></td>
<td></td>
<td>2016</td>
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<tr>
<td>Wen-Chieh Lee, MED, EDTC</td>
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<td>Zhiqing Zhou, MED, EPSY</td>
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<tr>
<td>Jane Carter, PHD, CLPY</td>
<td></td>
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<tr>
<td>Yee Lok Dorothy, MED, EPSY</td>
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<tr>
<td>*Mahati Kopparla, PHD, EDCI</td>
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<td>Armanto Sutedjo, PHD, ESPY</td>
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<tr>
<td>Ryan Hinojosa, PHD, SPSY</td>
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<tr>
<td>Kailiegh Byrne, PHD, PSYCH</td>
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<tr>
<td>Ross DeForrest, PHD, PSYCH</td>
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<tr>
<td>Stephanie Vidrine, PHD, SPSY</td>
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<tr>
<td>Amanda Lomax, PHD, SPSY</td>
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<tr>
<td>Justin K Meyer, PHD, CLPY</td>
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<tr>
<td>Brittany Penson, MS, PSYCH (Graduated, November 2016)</td>
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<tr>
<td>Yaoping Peng MS, EPSY (Graduated, April 2015)</td>
<td></td>
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<td>Luchen Jiang, MED, EDCI (Graduated, April 2016)</td>
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<tr>
<td>Siqi Chen, MS, EPSY (Graduated, July 2016)</td>
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<tr>
<td>*Yajun Jia, MS, EPSY (Graduated, July 2016)</td>
<td></td>
<td>2015</td>
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**Neurobiological Lab-related supervision & projects**

**CTRAL Sleep PSG training, TAMU (Weekly)**

*Lead intense training of interdisciplinary team of 5+ people on sleep equipment.*

**Lab meetings, TAMU (Bimonthly)**

*Lead discussion on organization or content, organize or give presentations, distribute readings, guest speakers.*

**Lab workshops, Tamu (Seasonally)**

*Provide intense training sessions for students to run complex EEG experiments or analysis.*
Lead monthly lab group seminars, UoT 2009 – 2014

Provide specialized training to research projects students, UoT 2009 – 2013

Non-academic teaching experience

Private Tutor (part-time) 2005 - 2007

School Teacher (part-time) 1998 - 2005

Academic Service

Committee membership

A1 Advisory Committee 2015-Present
Search Committee member (Learning Sciences Program) 2016-Present
Assistant professor representative 2015-2016
Search Committee member Assistant prof (School Psychology) 2015-2016
Search Committee member Associate prof (School Psychology) 2015-2016

Grant reviewer.
2017 - National Science Centre, Poland
2014 - The Louisiana Board of Regents Research Competitiveness.

Ad hoc journal reviewer (* = Co-reviewed with student).
2015 Biological Psychiatry; Biological Psychology; Child and Adolescent Mental Health; Psychiatry Research: Neuroimaging; ADHD*; Archives of Clinical Neuropsychology*; Journal of Abnormal Child Psychology*.
2014 Journal of Psychiatric Research; Developmental Science; Child Development
2013 Frontiers in Human Neuroscience; Journal of Neuropsychiatric Disease and Treatment; Biological Psychology; Clinical Neuropsychology; Research in Developmental Disabilities; Clinical Neurophysiology; Behavioral and Brain Functions; Biological Psychiatry; Journal of Attention Disorders; Journal of Pharmacology and Pharmacotherapeutics; NeuroImage.
Community outreach.

Professional consultancy.

Workgroup "From Science to Practice and Back: Mechanisms of Change in Developmental Psychopathology". Sickkids Hospital - Community Health Systems Resource Group. October, 2008


---

Awards & honors

National:

Awarded Elsevier Outstanding Reviewer Status. 2015

Social Sciences and Humanities Research Council Scholarship 2010

Mary Gertrude l’Anson Scholarship 2008

Provincial/State:

Ontario Graduate Scholarship 2011

Ontario Graduate Scholarship (Declined) 2010

Institutional:

Graduate Assistantship - Research Award 2016
CEHD Strategic Research Award. $34,000.00
1 student funded, 2016-2017 (Submitted March, 2016)

2016 USRI Award for undergraduate work-study student, TAMU 2015

Departmental Climate Award (awarded to staff and faculty for exceptional contributions to a positive climate). TAMU 2015

Academic Excellence Award, University of Toronto 2011
SGS Travel Award, University of Toronto \hspace{2cm} 2011

Doctoral Completion Award, University of Toronto \hspace{2cm} 2011

Graduate Student Award, University of Toronto \hspace{2cm} 2006 - 2010
Darrell A. Worthy, Ph.D.

POSITION TITLE
Associate Professor

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

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

<table>
<thead>
<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
<th>MM/YY</th>
<th>FIELD OF STUDY</th>
</tr>
</thead>
<tbody>
<tr>
<td>University of North Texas</td>
<td>B.S.</td>
<td>05/2005</td>
<td>Psychology</td>
</tr>
<tr>
<td>University of Texas at Austin</td>
<td>M.A.</td>
<td>12/2007</td>
<td>Psychology</td>
</tr>
<tr>
<td>University of Texas at Austin</td>
<td>Ph.D.</td>
<td>08/2010</td>
<td>Psychology</td>
</tr>
</tbody>
</table>

NOTE: The Biographical Sketch may not exceed four pages. Follow the formats and instructions below.

A. Personal Statement

My research program aims to develop a full understanding of human learning and decision-making using a computational cognitive neuroscience approach. Decision-making is a pervasive task that people must engage in on a daily basis, and many decisions have serious and long-term consequences. My goal is to examine the behavioral, computational, and neural mechanisms by which different types of decisions are made, and to also examine how a variety of different situational and dispositional factors affect learning and decision-making processes. Some of the central questions I examine are: What affects people’s ability to focus on both immediate and delayed outcomes of their decisions? How do people respond to gains and losses and to improvements or declines in the rewards they receive for their actions? What affects people’s preferences for novel choices when they are faced with a decision, and how do preferences become entrenched? What motivational, emotional and individual difference factors affect learning and decision-making? And, what types of neural systems mediate different forms of learning and to what degree is knowledge available for explicit, verbalizable representation? In empirically examining these issues I attempt to focus on addressing the theoretical issues that are relevant to Cognitive Psychology and to the field of Psychological Science as a whole, and to also be mindful of the applied relevance and implications of my research.

I use a combination of behavioral, computational, and neuroscientific approaches in my research. Behaviorally, I utilize a broad range of experimental tasks that are designed to pinpoint how different situational and dispositional factors influence aspects of decision-making like preferences for immediate versus delayed rewards, responses to gains versus losses, preferences for novel options, and sensitivity to misleading information. The tasks I use are all amenable to computational modeling and most can be modified to run using fMRI. The development and use of computational models to describe behavior is a key aim of my research program. I view models as falsifiable theories of cognition, and I feel that a greater deal of rigor and scientific precision can be obtained through their use. The majority of my work has utilized behavioral and computational modeling methods to understand learning and decision-making. However, my current work is also focused on incorporating neuroscientific methods to fully understand decision-making - from brain mechanisms to computational processes to behavior. I have recently begun work aimed at examining how genetic polymorphisms in genes responsible for controlling reuptake in neurotransmitters like dopamine and serotonin affect important cognitive processes. I also have ongoing work that utilizes fMRI to understand how age and performance pressure affect neural mechanisms involved in decision-making.
B. Positions and Honors

**Positions and Employment**

2005-2010  Graduate RA, Drs. W. Todd Maddox, and Arthur B. Markman, Department of Psychology, University of Texas at Austin

2010-2015  Assistant Professor, Department of Psychology, Texas A&M University

2015-      Associate Professor, Department of Psychology, Texas A&M University

**Other Experience and Professional Memberships**

2006-      Cognitive Neuroscience Society 2006-present

2007-      Society for Neuroeconomics 2007-present

2008-      Cognitive Science Society 2008-present

2011-      Society for Neuroscience 2011-present

2010-      Psychonomic Society

Ad-hoc Reviewer

**Grant Panels**

- National Science Foundation: Perception, Action, and Cognition Panel
- Swiss National Science Foundation

**Scientific Journals**

- Aging Neuropsychology, and Cognition
- Behavioral Brain Research
- Behavioral Research Methods
- Canadian Journal of Experimental Psychology
- Cognition
- Cognitive, Affective, and Behavioral Neuroscience
- Cognitive Psychology
- Cognitive Science
- Current Directions in Psychological Science
- Decision
- Educational Psychology
- European Journal of Information Systems
- Experimental Brain Research
- Frontiers in Cognitive Science (Review Editor 2016 - )
- Frontiers in Neuroscience
- Frontiers in Psychology
- Journal of Applied Psychology
- Journal of Applied Sport Psychology
- Journal of Cognitive Neuroscience
- Journal of Consumer Research
- Journal of Experimental Psychology: Applied
- Journal of Experimental Psychology: General
- Journal of Experimental Psychology: Human Perception and Performance
- Journal of Experimental Psychology: Learning, Memory, and Cognition
- Journal of Experimental Social Psychology
- Journal of Gerontology Series B: Psychological Sciences
- Journal of Sport and Exercise Psychology
- Memory & Cognition
- Neurobiology of Aging
- Personality and Social Psychology Review
- PLOS One
- Proceedings of the Cognitive Science Society
- Psychonomic Bulletin & Review
C. Selected Peer-reviewed Publications


Neuroscience, 27, 509-521. (5-Year Impact Factor: 5.705. Equal contribution from the first two authors).


*Denotes supervised graduate student contribution
**Denotes supervised undergraduate student contribution

BOOK CHAPTERS AND OTHER PUBLICATIONS


PEER-REVIEWED CONFERENCE PROCEEDINGS


D. Research Support

Principal Investigator (Co-PI: W. Todd Maddox).
NIMH R01, PAR-11-337; Total Direct Costs: $1,182,700
Impact Score: 20; Percentile: 6th
A Computational Neuroscience Approach to Frontal Compensation in Decision-Making (Funding Period – 01/01/2014 – 01/01/2019).
NAME: Young, Keith A.

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

POSITION TITLE: Pharmacologist, VA Research; Professor, Psychiatry & Behavioral Science

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.)

<table>
<thead>
<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
<th>Completion Date MM/YYYY</th>
<th>FIELD OF STUDY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baylor University, Waco, TX</td>
<td>B.S.</td>
<td>1981</td>
<td>Biology</td>
</tr>
<tr>
<td>University of Texas at Austin, Austin, TX</td>
<td>M.S.</td>
<td>1986</td>
<td>Pharmacology</td>
</tr>
<tr>
<td>University of Texas at Austin, Austin, TX</td>
<td>Ph.D.</td>
<td>1990</td>
<td>Pharmacology</td>
</tr>
<tr>
<td>Texas A&amp;M University College of Medicine, Temple, TX</td>
<td>Postdoctoral Fellowship</td>
<td>1992</td>
<td>Psychiatry</td>
</tr>
</tbody>
</table>

Personal Statement
I am a Professor of Psychiatry in the TAMHSC Department of Psychiatry and Behavioral Science and an investigator Investigator for the VISN 17 Center of Excellence for Research on Returning War Veterans. My lab has been performing research into biological factors mediating susceptibility to mental conditions at the Central Texas Veterans Health Care System and at Texas A&M College of Medicine for over 28 years. Our lab currently specializes in human blood and brain molecular, genetic and anatomical studies and in longitudinal studies related to stress and PTSD. We have recruited and phenotyped over 6000 veterans and active duty troops in Central Texas over the past 8 years and are currently performing genetic and epigenetic research on samples from these cohorts. Our currently funded research focuses on investigation of traits and genetic factors influencing blood and brain biomarkers for PTSD and other deployment-related mental conditions.

Positions and Honors

Positions and Employment
1992-2003 Assistant/Associate Professor, Psychiatry and Behavioral Science, Texas A&M Health Science Center (TAMHSC), Temple, TX
1993-Present Pharmacologist/Research Coordinator and Site Manager for Research, Central Texas Veterans Health Care System (CTVHCS), Temple, TX
1996-Present Director, Department of Psychiatry Neuropsychiatry Research Program, Temple, TX
2004-2005 Associate Chief of Staff for Research, CTVHCS (Acting), Temple, TX
2012-Present Professor, Psychiatry and Behavioral Science, TAMHSC, Temple, TX
2013-2014 Associate Chief of Staff for Research, CTVHCS (Acting), Temple, TX
2014-Present Investigator, Biomarker and Genetics Core, VISN 17 Center of Excellence for Research on Returning Veterans (CoE), CTVHCS, Waco, TX

Other Experience and Professional Memberships
1996-2008 Executive Director, TEMPVA Research Group, Inc. (VA non-profit Foundation)
1998-2014 Member (2002-2003 Chair), VISN 17 Research Committee, CTVHCS
2004-Present Member (2012-2013 Chair), Research Advisory Council, TAMHSC
2005-Present Vice Chair for Research, Department of Psychiatry and Behavioral Science, TAMHSC
2005-2014 Member (2009-2013 Chair), Tissue Advisory Board, Autism Speaks Autism Tissue Program
2011-Present Member, Education Committee, VISN 17 CoE
2011-Present Member, STRONGSTAR Research Consortium PI Council
2013-Present Member, UK Brain Bank for Autism & Related Developmental Research Advisory Group
2014-Present Member, Steering Committee, VA National PTSD Brain Bank
2015-2016 Chair, VA Merit Review Panel SPL/Genomics/Genetics
2017-Present Member, Scientific Review Board, Simons Foundation Autism BrainNet

Honors
1990 NIH Travel Fellowship: 1st American Course on Stereology for the Neurosciences
1991 Young Investigator Award, International Congress on Schizophrenia Research
1997 VISN 17 New Investigator Award
2000 VISN 17 Young Researcher of the Year

Bibliography:


Young KA and Wilcox RE. *Characterization of D2 receptors and dopamine levels in the thalamus of the rat.* Life Science 48; 1845-1852 (1991).

Young KA, Zavodny R and Hicks PB. *Subchronic buspirone, mesulergine, and ICS 205-930 lack effects on D1 and D2 DA binding during chronic haloperidol treatment.* J Neural Transm 86; 223-228 (1991).

Young KA, Zavodny R and Hicks PB. *Effects of serotonergic agents on apomorphine-induced locomotor activity.* Psychopharmacology 110; 97-112 (1993).

Young KA, Hicks PB, Randall PK and Wilcox RE. *Behavioral and frontal cortical metabolic effects of apomorphine and muscimol microinjections into the mediodorsal thalamic nucleus.* J Neural Transm 98; 119-132 (1994).


Wadenberg M-L, Young KA, Richter JT and Hicks PB. *Effects of local application of 5HT into the dorsal or median raphe nuclei on haloperidol-induced catalepsy in the rat.* Neuropharmacology 38; 151-156 (1999).

Barwick VS, Young KA, Jones DH, Richter T and Hicks PB. *Subthalamic microinjections of Clozapine and other 5HT2A antagonists reverse apomorphine-induced stereotypy.* Neuroreport 11; 267-270 (2000).

Young KA, Manaye KF, Liang C-L, Hicks PB and German DC. *Reduced number of mediodorsal and anterior thalamic neurons in schizophrenics.* Biol Psychiatry 47; 944-954 (2000).

Young KA, Smith M, Rawls T, Elliot DB, Steele Russell I and Hicks PB. *N100 evoked potential latency variation and startle in schizophrenia.* Neuroreport 12; 1-7 (2001).

Wadenberg M-L, Browning JL, Young KA, Hicks PB. *Antagonism at 5-HT2A receptors potentiates the effect of haloperidol in a conditioned avoidance response task in rats.* Pharmacol Biochem Behav 68; 363-370 (2001).


Schubert MS, Young KA, Hicks PB. *Galantamine improves cognition in schizophrenics stabilized on risperidone.* Biol Psychiatry 60; 530–533 (2006).


Young KA, Holcomb LA, Yazdani U, Bonkale W, Hicks PB, German DC. *5HTTLPR polymorphism and enlargement of the pulvinar: Unlocking the backdoor to the limbic system.* Biol Psychiatry 61; 813-8 (2007).


Anderson NE, Wan L, Young KA, Stanford MS *Psychopathic Traits Predict Startle Habituation but not Modulation in an Emotional Faces Task.* Personal Indiv Diff 50; 712–716 (2011.)


BIOGRAPHICAL SKETCH

Provide the following information for the Senior/key personnel and other significant contributors in the order listed on Form Page 2. Follow this format for each person. DO NOT EXCEED FOUR PAGES.

NAME
Zoran, Mark J.

POSITION TITLE
Professor of Biology and Neuroscience
Associate Dean of Science

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

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

<table>
<thead>
<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
<th>MM/YY</th>
<th>FIELD OF STUDY</th>
</tr>
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<tbody>
<tr>
<td>Augustana College, Rock Island, IL</td>
<td>B.A.</td>
<td>1979</td>
<td>Biology</td>
</tr>
<tr>
<td>Illinois State University, Normal, IL</td>
<td>M.S.</td>
<td>1981</td>
<td>Biological Sciences</td>
</tr>
<tr>
<td>Iowa State University, Ames, IA</td>
<td>Ph.D.</td>
<td>1987</td>
<td>Zoology</td>
</tr>
<tr>
<td>Iowa State University, Ames, IA</td>
<td>Postdoc.</td>
<td>1987-91</td>
<td>Neuroscience</td>
</tr>
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A. Personal Statement

The goal of my lab’s research is to investigate the plasticity of neural signaling between cells of animal nervous systems, as it relates to neural function and behavior. Our NIH-funded research has involved multiple model systems and contexts, including neural development, synaptic plasticity and biological rhythms. One of my research projects aims to understand the cellular and molecular links between the circadian clock, astrocyte signaling, metabolism and diseases. My lab has recently demonstrated that ATP production and release from brain glial cells is under circadian control. This work has been conducted in collaboration with Dr. David Earnest of the TAMU College of Medicine. We hypothesize that rhythmic production and release of ATP by glia feeds back to regulate circadian oscillator function. My lab utilizes both in vivo and powerful in vitro model system to explore the regulation of synapses and neural cell communication. One of these contexts is involved in a NIH-funded project using Drosophila behavioral genetics, in collaboration with Dr. Vlad Panin, to determine the role of sialyltransferase genes in neural signaling. This work has demonstrated a critical role for this enzyme and glycosylation in the regulation of neural function and synaptic plasticity. Furthermore, these mutant flies have blood brain barrier (BBB) defects that likely involve glia dysfunctions, and this is a important new line of research involve my collaboration with Dr. Panin’s lab and co-mentored graduate students.

I have a broad background in electrophysiology in both vertebrate and invertebrate species as a neuroscience researcher. With over 25 years of experience as a cellular neurobiologist with focus on neural cell signaling and published over 40 peer-reviewed papers on synaptic function. I possess the requisite scientific expertise and managerial skills needed to carry out the research aims of this proposal. I have overseen the day-to-day operations of an NIH P01-supported cellular and molecular imaging core (within the Texas A&M Center for Biological Cocks Research) for over 10 years (Bell-Pedersen et al., 2005). Additionally, I am an Associate Dean in the College of Science and serve on the steering committees of three federally (NSF)-funded programs to recruit and mentor underrepresented minority graduate students through the doctorate, to provide STEM teaching professional development to minority graduate students and postdocs, and mentor junior STEM women faculty of color through the promotion and tenure process. Thus, I am well qualified to conduct the administrative duties of this project. To summarize, I have a strong record of research productivity in the fields of neurobiology and circadian biology, and the necessary scientific expertise and administrative experience to support the successful completion of the proposed studies and mentoring of personnel involved.

B. Positions and Honors

Professional Experience

<table>
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<tr>
<th>Year Range</th>
<th>Position/Role</th>
<th>Institution</th>
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<tr>
<td>1987-1991</td>
<td>Postdoctoral Fellow</td>
<td>Department of Zoology and Genetics, Iowa State University</td>
</tr>
<tr>
<td>1991-1997</td>
<td>Assistant Professor</td>
<td>Department of Biology, Texas A&amp;M University</td>
</tr>
<tr>
<td>1997-2011</td>
<td>Associate Professor</td>
<td>Department of Biology, Texas A&amp;M University</td>
</tr>
<tr>
<td>2001-2007</td>
<td>Chair, Faculty of Neuroscience</td>
<td>Texas A&amp;M University</td>
</tr>
<tr>
<td>2003-Present</td>
<td>Associate Dean for Graduate Studies</td>
<td>College of Science, Texas A&amp;M</td>
</tr>
<tr>
<td>2011-Present</td>
<td>Professor, Department of Biology and Neuroscience</td>
<td>Texas A&amp;M University</td>
</tr>
<tr>
<td>2013-Present</td>
<td>Associate Dean for Faculty Affairs and Graduate Studies, College of Science, TAMU</td>
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Other Experience and Professional Memberships

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<th>Activity</th>
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<tr>
<td>1987-Present</td>
<td>Member, Society for Neuroscience</td>
<td></td>
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<tr>
<td>1999-Present</td>
<td>Director, Cellular and Molecular Imaging Facility</td>
<td>Texas A&amp;M University</td>
</tr>
<tr>
<td>2001-Present</td>
<td>Member, Center for Biological Clocks Research</td>
<td>Texas A&amp;M University</td>
</tr>
<tr>
<td>2003-Present</td>
<td>Chair, College of Science, Graduate Instruction Committee</td>
<td></td>
</tr>
<tr>
<td>2003-Present</td>
<td>Member, TAMU Graduate Operations Committee</td>
<td></td>
</tr>
<tr>
<td>2005-Present</td>
<td>Member, Society for Research on Biological Rhythms</td>
<td></td>
</tr>
<tr>
<td>2005-Present</td>
<td>Research Associate</td>
<td>Texas Brain and Spine Institute</td>
</tr>
<tr>
<td>2006-Present</td>
<td>NSF LSAMP Bridge to the Doctorate Program</td>
<td>TAMU Advisory Group</td>
</tr>
<tr>
<td>2009-2011</td>
<td>Member, Biosafety Advisory Committee</td>
<td>Texas A&amp;M VP for Research</td>
</tr>
<tr>
<td>2011-2015</td>
<td>Chair, Texas A&amp;M University Graduate Council</td>
<td></td>
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Honors

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<tr>
<td>2008</td>
<td>Association of Former Students, Distinguished Achievement Award</td>
</tr>
<tr>
<td>2009</td>
<td>Interdisciplinary Faculty of Neuroscience Service Award</td>
</tr>
<tr>
<td>2011</td>
<td>President, TAMU Chapter, Society for Neuroscience</td>
</tr>
<tr>
<td>2011</td>
<td>President, TAMU Chapter, Sigma Xi Society</td>
</tr>
<tr>
<td>2012</td>
<td>Sigma Xi Society Meritorious Service Award</td>
</tr>
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</table>

C. Contribution to Science

M.S and Ph.D. in Biological and Zoological Sciences

As a Master’s student at Illinois State University, I studied in the lab of Jack Ward on the ethology of fishes. My research investigated the contributions of males and females in reproductive behavior, which in the Asia cichlid *Etroplus maculatus* is biparental with equal contributions of both sexes. I determined the parental investment with regard to nest (egg) fanning behavior and its physiological benefit to the embryo and cost to the adult (Zoran & Ward, 1983). At the time, these studies of biparental care in the cichlid fish were novel in demonstrating how the constraints of a reproductive system (in this case colonial breeding) shaped physiological and behavioral traits. These early research experiences in many ways impacted my future research career. These studies provided me with an appreciation for interface of physiology and behavior, which fostered my interest in a neuroscience career. It taught me the importance of experimental design and the complexities of behavioral research, which shape my research still today.


Development and Regeneration of Identified Neural Networks

As a doctoral student at Iowa State University, I studied the neural networks that mediate rapid escape behavior in annelid worms. With my advisor, Charles Drewes, I determined the neurophysiological and neuroanatomical substrates of escape circuits in diverse taxa: earthworms, mud worms and aquatic worms. It was these studies that build the foundation for the subsequent 30 years of research into the cellular and molecular mechanisms governing communication among neural cells and their recovery of communication following injury. One of the projects during my doctoral research focused on a form of nervous system regeneration, called neural morphallaxis, in the mud worm *Lumbriculus variegatus*. This species has the rare
capacity for rapid regeneration of head and tail segments following loss of body parts. During this regeneration, segments undergo a change in positional identity and subsequently the underlying nervous system in these segments change via neural morphallaxis. My lab at Texas A&M University has continued to investigate this neural regeneration model and have discovered that neural regeneration and asexual reproduction share common molecular changes (Martinez et al., 2005). Specifically, expression of a neural glycoprotein is upregulated at during morphallaxis, which is a critical underpinning of rapid neural circuit switching associated with regenerative and reproductive transition in behavior (Lybrand and Zoran, 2012).


Cellular Mechanisms of Synapse Formation and Plasticity
Over the last 25 years as a researcher at Texas A&M University, my laboratory has studies the cellular and molecular mechanisms governing communication among nerve cells and their target, both in the context of synapse development and plasticity. One project in this regard has focused on the role of electrical synapses in shaping the formation of identified neural networks. The snail, Helisoma trivolvis, has been the model system for most this work due to the ability to readily grow and manipulated specific neuronal connection in culture.


Role of Glycosylation in Nervous System Function
In collaboration with Dr. Vlad Panin, Texas A&M University, our NIH-funded projects using *Drosophila* behavioral genetics have determined the role of glycobiological processes in the regulation in neural cell signaling. This work has demonstrated a critical role for glycosylation in nervous system function and animal behavior, using genetic and electrophysiological approaches.


Circadian Regulation of Neural Signaling
Another project centers on biological clock regulation of neural cell communication in the mammalian brain. His circadian neuroscience studies investigate the role of adenosine triphosphate (ATP) signaling as an important output of the mouse biological clock of specific brain cells called astrocytes. His lab aims to determine the role of this clock-controlled signaling in normal brain function and in various neurological disorders.


Other Contributions to Science at Texas A&M University

As Associate Dean for Faculty Affairs and Graduate Studies in the College of Science at Texas A&M University, I oversee the administration of all graduate programs and faculty, including programs in Biology, Chemistry, Mathematics, Neuroscience, Physics, Astronomy and Statistics. I served as the Chair of the Texas A&M University Faculty of Neuroscience for 6 years. I am a past-President of the TAMU Chapters of the Society for Neuroscience and Society for Sigma Xi. In terms of advancement of underrepresented minorities in STEM and the Biomedical Sciences, I am on the steering committees of the TAMU NSF LSAMP-BTD, NSF AGEP and NSF ADVANCE programs. I am also the representative to the GEM Consortium for minority student placement in science. I am a Research Associate of the Texas Brain and Spine Institute (TBSI), an organization of local neurologists and neuroscientist that brings basic scientists and medical professionals together to foster medical research. I have trained 14 graduate students, 52 undergraduate students and 2 high school students, for a total of 68 trainees (including 39 women and 11 underrepresented minorities).

D. Research Support

Completed Research Support

R01 NS075534 V. Panin (PI) 8/01/2011-7/31/2016
NIH/NINDS
The Control of Neural Transmission by Glycosylation
The goal of this project is to determine the cellular and molecular role of glycosylation in nervous system function, particularly neuron excitability. Neurophysiology conducted by M.J. Zoran.
Role: Co-I

P01 NS39546 D. Bell-Pedersen (PI) 07/01/06-06/30/11
NIH/NINDS
Coordination of Circadian Physiology of Diverse Species
The goal of this project is to determine the cellular and molecular mechanisms that regulate biological clock function is a diverse array of organisms, from bacteria to mammals. Imaging core is directed by M.J. Zoran.
Role: Co-I

P01 NS39546 V. Cassone (PI) 12/01/99-6/30/06
NIH/NINDS
Coordination of Circadian Physiology of Diverse Species
The goal of this project is to determine the cellular and molecular mechanisms that regulate biological clock function is a diverse array of organisms, from bacteria to mammals. Imaging core is directed by M.J. Zoran.
Role: Co-I

IBN-9421372 M. Zoran (PI) 06/01/95-05/31/99
NSF-Developmental Neuroscience
Target-Induced Regulation of Neuronal Synapse Formation
The goal of this project was to determine, using identified neuronal cell cultures and electrophysiological approaches, the cellular and molecular determinants of target-specific neuromuscular synapse formation.
Role: PI
APPENDIX U

Faculty Publications
ABBOTT, Louise C.


Daniel E. Miller, Raj Shah, Wencong Zhang, Jaewook Yoo, Jaerock Kwon, David Mayerich, John Keyser, Louise C. Abbott, Yoonsuck Choe. Fast Submicrometer Scale Imaging of Whole Zebrafish Using the KnifeEdge

Texas A&M Institute for Neuroscience
Transmission electron microscopic evaluation of neuronal changes in methylmercury-exposed zebrafish embryos (Danio rerio)
By: Hassan, Said A.; Farouk, Sameh M.; Abbott, Louise C.
ULTRASTRUCTURAL PATHOLOGY  Volume: 40  Issue: 6  Pages: 333-341  Published: NOV-DEC 2016

Neural cell proliferation and survival in the hippocampus of adult CaV 2.1 calcium ion channel mutant mice
By: Nigussie, Fikru; Huang, Pei-San; Lukauskis, Kris; et al.
BRAIN RESEARCH  Volume: 1650  Pages: 162-171  Published: NOV 1 2016

Effects of methyl mercury exposure on pancreatic beta cell development and function
By: Schumacher, Lauren; Abbott, Louise C.
JOURNAL OF APPLIED TOXICOLOGY  Volume: 37  Issue: 1  Pages: 4-12  Published: JAN 2017
AMREIN, Hubert Otto


Ahn, J.E., Chen, Y., and Amrein, H. (submitted). Ir25a and Ir76a are necessary for fatty acid taste in Drosophila.


Book Chapters


BEHMER, Spencer T.


In Press


2015


2014


2013


2012


BOLAÑOS-GUZMAN, Carlos A.


†Graduate student.
*Undergraduate student.
BUCHANAN, John J.


CARDOSO, Rodolfo C.


CARNEY, Ginger E.

**Peer-reviewed Publications:** (*indicates undergraduate co-author; underlining indicates Carney graduate student)


Video abstract featured by Bioessays: [https://www.youtube.com/watch?v=ZXAEI_aKJS&feature=youtu.be](https://www.youtube.com/watch?v=ZXAEI_aKJS&feature=youtu.be)

*Featured by The Society for Integrative and Comparative Biology May 2014* (http://sicb.org/students/gilmanc.php).


CHEN, Wei-Jung


2016


2015

Yoonsuck Choe, Jaewook Yoo, and Qinbo Li. Tool construction and use challenge: Tooling test rebooted. In AAAI-15 Workshop on Beyond the Turing Test, 2015. 2 pages.


Qinbo Li, Jaewook Yoo, and Yoonsuck Choe. Emergence of tool use in an articulated limb controlled by evolved neural circuits. In *Proceedings of the International Joint Conference on Neural Networks*, 2015. DOI: 10.1109/IJCNN.2015.7280564.


2014


2013


2012


DEMORROW, Sharon


Inhibition of histidine decarboxylase ablates the autocrine tumorigenic effects of histamine in human cholangiocarcinoma. Francis H, DeMorrow S, Venter J, Onori P, White M, Gaudio E, Francis T, Greene JF Jr,

DINDOT, Scott V.


DuBois, Dustin W.


* Corresponding authors.


EITAN, Shoshana


Emery MA, Bates ML, Wellman PJ, Eitan S. "Hydrocodone, but neither morphine nor oxycodone, is effective in suppressing the development of burn-induced mechanical allodynia." Pain Medicine, 2017, in press

Emery MA, Bates ML, Wellman PJ, Eitan S. "Hydrocodone, but neither morphine nor oxycodone, is effective in suppressing burn-induced mechanical allodynia in the uninjured foot contralateral to the burn" Journal of Burn Care and Research, 2017, in press

Kibola, A, McClelland, S, Hlavin, J, Friedman, JA. Pilomyxoid Astrocytoma in an Adult Woman: Case Report. In Preparation


FRYE, Gerald D.


GARCIA, Luis Rene


Guo, X and García, LR. 2014. SIR-2.1 integrates metabolic homeostasis with the reproductive neuromuscular excitability in aging male C. elegans. *eLife*:3:e01730


GARCIA, Tanya P.


Geraci, Lisa


GRAU, James


Damborsky, J, **Griffith, WH**, Winzer-Serhan, UH. Chronic neonatal nicotine exposure increases excitation in the young adult rat hippocampus in a sex-dependent manner. Brain Research.1430: 8-17, 2012.[PMID: 22119395]


HAMOUDA, Ayman K.

Research Papers: (* = corresponding Author; ** = Equal Contributions)


**Book Chapters:**

Abstracts/ Poster Presentations:


Hamouda, A. K. Wang Z.J, Mohamed T.S., Alaskari A. (2016) “3-(2-chlorophenyl)-5-(5-methyl-1-(piperidin-4yl)-1H-pyrazol-4-yl)isoxazole (CMPI) is a selective positive allosteric modulator of low-sensitivity (α4)3(β2)2 nicotinic acetylcholine receptor”. *Society for Neuroscience, 667.15*

**HAN, Arum**


**HARDIN, Paul E.**


Edited Book Chapters


Journal Articles


JAFARI, Roozbeh

Jian Wu, **Roozbeh Jafari**, Seamless Vision-assisted Placement Calibration for Wearable Inertial sensors, ACM Transactions on Embedded Computing Systems, Accepted for publication.

Terrell R. Bennett, Nicholas Gans, **Roozbeh Jafari**, Data-Driven Synchronization for Internet of Things Systems, ACM Transactions on Embedded Computing Systems, Accepted for publication.


Chen Chen, **Roozbeh Jafari**, Nasser Kehtarnavaz, A Survey of Depth and Inertial Sensor Fusion for Human Action Recognition, Multimedia Tools and Applications, Accepted for publication.

Hassan Ghasemzadeh, Ramin Fallahzadeh, **Roozbeh Jafari**, A Hardware-Assisted Energy-Efficient Processing Model for Activity Recognition using Wearables, ACM Transactions on Design Automation of Electronic Systems (TODAES), Accepted for publication.


Viswam Nathan, **Roozbeh Jafari**, Design Principles and Dynamic Front End Reconfiguration for Low Noise EEG Acquisition with Finger Based Dry Electrodes, IEEE Transactions on Biomedical Circuits and Systems (T-BioCAS), Accepted for publication.

Simi Susan Thomas, Viswam Nathan, Chengzhi Zong, Karthikeyan Soundarapandian, Xiangrong Shi, **Roozbeh Jafari**, BioWatch: A Non-invasive Wrist-based Blood Pressure Monitor that Incorporates Training Techniques for Posture and Subject Variability, IEEE Journal of Biomedical and Health Informatics (J-BHI), Accepted for publication.

Yuan Zou, Viswam Nathan, **Roozbeh Jafari**, Automatic Identification of Artifact-related Independent Components for Artifact Removal in EEG Recordings, IEEE Journal of Biomedical and Health Informatics (J-BHI), Accepted for publication.


Conference and Workshop Papers


Somok Mondal, Chung-Lun Hsu, Roozbeh Jafari, Drew Hall, A Dynamically Reconfigurable ECG Analog Front-End with a 2.5× Data-Dependent Power Reduction, IEEE Custom Integrated Circuits Conference (CICC), April 30 - May 3, 2017, Austin, TX, USA.


Varun Kumar, Xiaobo Guo, Roozbeh Jafari, Siavash Pourkamali, A Tunable Digitally Operated MEMS Accelerometer, IEEE Sensors, November 1-4, 2015, Busan, South Korea.


Chen Chen, Roozbeh Jafari, Nasser Kehtarnavaz, UTD-MHAD: A Multimodal Dataset for Human Action Recognition Utilizing a Depth Camera and a Wearable Inertial Sensor, IEEE International Conference on Image Processing (ICIP), September 27-30, 2015, Quebec City, Canada.


Terrell R. Bennett, Nicholas Gans, Roozbeh Jafari, A Data-driven Synchronization Technique for Cyber-Physical Systems, 2nd International Workshop on the Swarm at the Edge of the Cloud, in conjunction with CPSWeek 2015, April 13-16, 2015, Seattle, WA, USA.

Chengzhi Zong, Somok Mondal, Drew Hall, Roozbeh Jafari, Digitally Assisted Analog Front End Power Management Strategy via Dynamic Reconfigurability for Robust Heart Rate Monitoring, 7th Workshop on Adaptive and Reconfigurable Embedded Systems, in conjunction with CPSWeek 2015, April 13-16, 2015, Seattle, WA, USA.


Viswam Nathan, Roozbeh Jafari, Reducing the Noise Level of EEG Signal Acquisition through Reconfiguration of Dry Contact Electrodes, IEEE Biomedical Circuits and Systems Conference (BioCAS), October 22-24, 2014, Lausanne, Switzerland.


Terrell R. Bennett, Claudio Savaglio, David Lu, Hunter Massey, Xianan Wang, Jian Wu, Roozbeh Jafari, MotionSynthesis Toolset (MoST): A Toolset for Human Motion Data Synthesis and Validation, MobileHealth 2014, August 11-14, 2014, Philadelphia, PA.

Terrell R. Bennett, Roozbeh Jafari, Nicholas Gans, Motion Based Acceleration Correction for Improved Sensor Orientation Estimates, 2014 International Conference on Wearable and Implantable Body Sensor Networks (BSN), June 16-19, Zurich, Switzerland.


Omid Dehzangi, Yuan Zou, Roozbeh Jafari, Simultaneous Classification of Motor Imagery and SSVEP EEG Signals, IEEE/EMBS Conference on Neural Engineering (NER), November 5-6, 2013, San Diego, CA.


Akshay Sridharan, Carl Sechen, Roozbeh Jafari, Low-Voltage Low-Overhead Asynchronous Logic, International Symposium on Low Power Electronics and Design (ISLPED), September 4 - 6, 2013, Beijing, China. (acceptance rate: 23%)

Terrell Bennett, Roozbeh Jafari, Nicholas Gans, An Extended Kalman Filter to Estimate Human Gait Parameters and Walking Distance, American Control Conference (ACC), June 17 - 19, 2013, Washington, DC.


Mohammad-Mahdi Bidmeshki, Roozbeh Jafari, Low Power Programmable Architecture for Periodic Activity Monitoring, ACM/IEEE International Conference on Cyber-Physical Systems (ICCPS), April 8-11, 2013, Philadelphia, PA. (acceptance rate: 23%)


KERWIN, Sharon C.


KHOSRAVIAN, Homa

PEER-REVIEWED JOURNAL PAPERS

Homa Khosravian, “Controlled synthesis of Rh nanoparticles on TiO2(110) surface via Rh6(CO)16 : An experimental study”, Submitted.


SELECTED CONFERENCE PROCEEDINGS


KLEMM, W. R.

Selected Publications (neuroscience)


Selected Publications (education)


Klemm, W. R. 2013. Teaching Beginning College Students with Adapted Published Research Reports. J. Effective Teaching. 13 (2), 6-20.


KO, Gladys Yi-Ping

US Patent
US Patent Title: Lv PEPTIDE, ANTI-LV ANTIBODY and METHODS THEREOF.
**Inventor:** Gladys Ko (75%); co-Inventor: Lih Kuo (25%).
The original invention disclosure filed to Texas A&M University on October 27, 2015, titled “Peptide Lv, anti-peptide Lv antibody, and their applications” (TAMUS 4411).

**Publications:**
Peer-reviewed research papers:
Calcineurin serves in the circadian output pathway to regulate the daily rhythm of L-type voltage-gated calcium channels in the retina. J. Cellular Biochemistry, 113: 911-922, 2012. **PMCID:** 3296962; **NIHMS:** 342130.

**Book Chapter**  
KORNEGAY, Joe N.


LEIOBOWITZ, Julian


LEVINÉ, Jonathan M.


Young BD, Mankin JM, Griffin JF, Fosgate GT, Fowler JL, Levine JM. Comparison of two fat-suppressed magnetic resonance imaging pulse sequences to standard T2-weighted images for brain parenchymal contrast and lesion detection in dogs with inflammatory intracranial disease. Vet Radiol Ultrasound 2015; 56: 204-211.


Bentley RT, Burcham GN, Heng HG, Levine JM, Longshore R, Carrera-Justiz S, Cameron S, Koft K, Miller MA. A comparison of clinical, magnetic resonance imaging, and pathological findings in dogs with gliomatosis cerebri, focusing on cases with minimal magnetic resonance imaging changes. Vet Comp Oncol 2014 (Epub Ahead of Print PMID: 24945683)


Stout-Steele M, Levine JM*. Intermittent pain and scratching in a Cavalier King Charles Spaniel. Clinicians Brief 2014; 66-70.


Levine JM. Vertebral column MRI. *Clinician’s Brief* 2012; 10: 65-68.
Li, Jianrong

Original Research Reports


Invited reviews and book chapters


Li, Peng


*Zhuo Feng and Peng Li, “Fast thermal analysis on GPU for 3D-ICs with integrated microchannel cooling,” IEEE Trans. on Very Large Scale Integration Systems, volume 21, issue 8, pp. 1526-1539, August 2013.


*Yongtae Kim and Peng Li, “A 0.003-mm2, 0.35-V, 82-pJ/conversion ultra-low power CMOS all digital temperature sensor for on-die thermal management,” Analog Integrated Circuits and Signal Processing, volume 75, issue 1, pp 147-156, April 2013.


**Refereed Conference Papers**

*Ang Li, Peng Li, Tingwen Huang, and Edgar Sanchez-Sinencio, “Noise-sensitive feedback loop identification in linear time-varying analog circuits,” in Proc. of Design Automation and Test In Europe Conference & Exhibition (DATE), March 2017 (accepted).


*Ya Wang, *Di Gao, Dani A. Tannir, and Peng Li, “Multi-harmonic nonlinear modeling of low-power PWM DC-DC converters operating in CCM and DCM,” in Proc. of Design Automation and Test In Europe Conference & Exhibition (DATE), pp. 409 – 414, March 2016, (long presentation, acceptance rate 24%).


Andrew Fisher, Chris Myers, and Peng Li, “Reachability analysis using extremal rates,” in Proc. of NASA Formal Methods Symposium, April 2015 (acceptance rate: 30.6%).


(IEEE/ACM William J. McCalla ICCAD Best Paper Award, one out of 338 submissions).


LOCKLESS, Steve W.


MACNAMARA, Annemarie


Book chapters


MAREN, Stephen A.


(http://jama.jamanetwork.com/article.aspx?articleid=2422534)


MATHUR, Vani Anshu Dawson


MCMAHAN, Uel J.


MEAGHER, Mary

PUBLICATION POLICY
My publication policy is designed to promote my graduate students and postdoctoral trainees (*) as well as select undergraduates (**) . I frequently assume the last author position (alternating last and second-to-last with collaborators) when publishing with students, which designates the senior investigator in the neuroscience literature.


MANUSCRIPTS IN PREPARATION


Book Chapters and Literature Reviews


**Published Abstracts**


MEHTA, Ranjana

Mehta, RK. & Rhee, J. 2 (Accepted). Age-specific neural strategies to maintain motor performance after an acute social stress bout. Experimental Brain Research. DOI: 10.1007/s00221-017-4949-9


Mehta, RK. & Cauvoto, LA. (2015). The effects of obesity, age, and relative force levels on handgrip endurance. Applied Ergonomics, 46(A), 91-95

Mehta, RK. & Agnew, MJ. (2015). Subjective evaluation of physical and mental workload interactions across different muscle groups. Journal of Occupational and Environmental Hygiene, 10(1), 62-68


Mehta, RK. & Parijat, P. (2012). Associations between psychosocial risk factors and musculoskeletal disorders: Application to the IT profession in India. Work, 41(Suppl. 1), 2438-44.

Mehta, RK. & Agnew, MJ. (2012). Effects of physical and mental demands on shoulder muscle fatigue. Work, 41(Suppl. 1), 2897-2901.
MENET, Jerome


**MERLIN, Christine**

**Peer-reviewed articles**


**Reviews, Book chapters**


**Manuscript in revision**


MORGAN, Caurnel


NAGAYA, Naomi


NGHIEM, Peter P.


ORR, Joseph M.


PANIN, Vladislav M.

In reverse chronological order, graduate and undergraduate students are underlined and dashed underlined, respectively:


Nakamura M, Pandey D, Panin VM (2012) Genetic interactions between Drosophila sialyltransferase and β1,4-N-acetylgalactosaminyltransferase-A genes indicate their involvement in the same pathway. G3: Genes Genomes Genetics, 2(6): 653-656

Published Abstracts


Castillo, P. and Pietrantonio, P. V.* 2013. Differences in sNPF receptor-expressing neurons in brains of fire ant (Solenopsis invicta Buren) worker subcastes: indicators for division of labor and nutritional status? PLoS ONE 8(12): e83966. doi:10.1371/journal.pone.0083966


Molecular regulation of water balance in ticks: tick genomics, and GPCR signaling for neuropeptides and serotonin. We characterized the first neuropeptide receptor from the Acari and the first neuropeptides by MALDI-TOF. We identified, cloned and helped annotate GPCRs for the genome publication of the tick Ix. scapularis.


PORTER, Brian F.


Hensel ME, Pasmakova M, **Porter BF**: Fatal caffeine intoxication in a dog. Brazilian Journal of Veterinary Pathology (in press)
RAMADOSS, Jayanth


Reddy, D. Samba


RICCIO, Cynthia A.

Refereed Journal articles


Books
Book Chapters


ROSENTHAL, Gil G.


Books


Articles


Abstracts


SCOTT, Erin M.


SHAPIRO, Lee A.


SHEA, Charles H.


SMITH, Laura N.


SMITH, Rachel J.


SMOTHERMAN, Michael S.


Smotherman, M., Bohn, K., Davis, K., Rogers, K. and C.P. Schwartz (2016) Daily and seasonal patterns of singing by the Mexican free-tailed bat, Tadarida brasiliensis. In Sociality in Bats, J. Ortega (ed.) Springer, Switzerland. DOI 10.1007/978-3-319-38953-0_9


SRINIVASAN, Rahul


STOICA, George


Delgado J., **Stoica G**. et al., Spontaneous multicentric soft tissue sarcoma in a captive African pygmy hedgehog. The Journal of Veterinary Medicine. Accepted for publication, 2016-10-24
SUN, Yuxiang


Featured on World Biomedical Frontiers 2015


Invited by the editor to write an editorial on the subject, which was published on Oncotarget


Special issue on “Neurobiological Perspectives on Ghrelin”
TASSINARY, Louis G.

Chapters


Books


Technical Reports


Articles


THOMPSON, Wes


TIFFANY-CASTILGLIONI, Evelyn


TOUSSAINT, Gerard


VAID, Jyotsna


BOOK

Refereed Journal Articles (student co-authors are indicated with an asterisk)


**Book Chapters**


VILLALOBOS, Alica R. A.


WANG, Jun


Wei, X. Y., Huang, C. Y., Wang, X. H., Lu, J. Y., Wang, J., Accepted. Dopamine D1 or D2 receptor-expressing neurons in the central nervous system. Addict Biol, in press.


WELSH, C. Jane


BOOK Chapters


Publications (students underlined)


Manuscripts under Review or In Preparation


WINZER-SERHAN, Ursula H.


WOLTERING, Steven

Liu, Z., Tannock, R., & Woltering, S. (In press). Effects of Working Memory Training on Neural Correlates of Go/Nogo Response Control in Adults with ADHD: A Randomized Controlled Trial. *Neuropsychologia*.


YOUNG, Keith A.


ZORAN, Mark J.

Cherrstrom CA, Richardson R, Fowler D, Autenrieth R, and Zoran MJ Creating teaching opportunities for STEM future faculty development. J STEM Teacher Education (Accepted)

Lybrand ZR, Martinez-Acosta, and Zoran MJ Coupled sensory interneurons mediate escape neural circuit processing in an aquatic annelid worm, Lumbriculus variegatus. J. Comp. Neurol. (Accepted)


APPENDIX V

Faculty Grants
<table>
<thead>
<tr>
<th>Award Title</th>
<th>Organization</th>
<th>Sponsor</th>
<th>Start Date</th>
<th>End Date</th>
<th>Principal Investigator</th>
<th>Award Amount</th>
<th>Fiscal Year</th>
</tr>
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<tbody>
<tr>
<td>Convergence of Cellular and Molecular Pathways in Appetitive Taste</td>
<td>TAMHSC-Molecular And Cellular Medicine</td>
<td>DHHS-NIH-National Institute of Child Health &amp; Human Development</td>
<td>4/1/12</td>
<td>11/30/20</td>
<td>Amrein, Hubert</td>
<td>$1,577,815.00</td>
<td>2016</td>
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<td>Molecular Basis for the Detection of Nutrients and Toxins by the Honeybee</td>
<td>TAMHSC-Molecular And Cellular Medicine</td>
<td>Newcastle University</td>
<td>2/3/15</td>
<td>2/2/18</td>
<td>Amrein, Hubert</td>
<td>$20,877.00</td>
<td>2016</td>
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<td>Gustatory Receptors sense RNA and ribonucic acid metabolites as nutrients and signaling molecules during rapid growth</td>
<td>TAMHSC-Molecular And Cellular Medicine</td>
<td>National Institutes of Health</td>
<td>4/1/16</td>
<td>3/31/17</td>
<td>Amrein, Hubert</td>
<td>$408,375.00</td>
<td>2016</td>
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<td>Nutritionally-mediated variation in Helicoverpa zea susceptibility to Bt transgenic crops</td>
<td>AL-RSRCH-Entomology</td>
<td>USDA-National Institute of Food And Agriculture</td>
<td>9/1/15</td>
<td>8/31/18</td>
<td>Behmer, Spencer</td>
<td>$249,516.50</td>
<td>2016</td>
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<td>Insect sterol requirements: a novel target for controlling insect herbivore pests</td>
<td>AL-RSRCH-Entomology</td>
<td>USDA-National Institute of Food And Agriculture</td>
<td>1/1/16</td>
<td>12/31/18</td>
<td>Behmer, Spencer</td>
<td>$227,000.00</td>
<td>2016</td>
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<td>Cerebelllo-Prefrontal Involvement in Error Processing and Rule Learning in Youth at Ultra High-Risk for Psychosis</td>
<td>TAMU-Psychology</td>
<td>Brain &amp; Behavior Research Foundation</td>
<td>1/15/15</td>
<td>1/14/17</td>
<td>Bernard, Jessica</td>
<td>$53,400.00</td>
<td>2016</td>
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<td>A High-Throughput Microfluidic in Vitro CNS Myelination Model towards Drug Screening</td>
<td>TEES-Electrical And Computer Engineering</td>
<td>National Institutes of Health</td>
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<td>6/30/17</td>
<td>Choe, Yoonsuck</td>
<td>$59,436.00</td>
<td>2016</td>
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<td>Neuroendocrine Effects of Alcohol on Puberty Award</td>
<td>Veterinary Integrative Biosciences</td>
<td>National Institutes of Health</td>
<td>9/1/15</td>
<td>5/31/20</td>
<td>Dees, William</td>
<td>$556,986.38</td>
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<td>The Epigenetics of the Aging Astrocyst: Implications for Stroke</td>
<td>TAMU-Vet-Pathobiology</td>
<td>Texas A&amp;M University Health Science Center</td>
<td>9/1/12</td>
<td>5/31/17</td>
<td>Dindot, Scott</td>
<td>$9,102.00</td>
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<td>Identification and Characterization of Novel Therapeutics for Angelman Syndrome</td>
<td>AL-RSRCH-Vet-Pathobiology</td>
<td>University of South Florida</td>
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<td>4/30/17</td>
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<td>$179,635.00</td>
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<td>Mouse Model of Dup15q Syndrome Award Number</td>
<td>AL-RSRCH-Vet-Pathobiology</td>
<td>Simons Foundation</td>
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<td>9/30/16</td>
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<td>Effect of inflammation on recovery and pain after spinal cord injury Award</td>
<td>TAMU-Psychology</td>
<td>DHHS-NIH-National Institute of Neurological Disorders and Stroke</td>
<td>2/1/16</td>
<td>1/31/18</td>
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<td>$402,205.00</td>
<td>2016</td>
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<td>Award Title</td>
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<td>End Date</td>
<td>Principal Investigator</td>
<td>Award Amount</td>
<td>Fiscal Year</td>
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<td>Optogenetic approaches to study complex neuronal circuits during cognitive aging</td>
<td>TAMHSC-Neuroscience &amp; Experimental Therapeutics</td>
<td>DHHS-NIH-National Institute On Aging</td>
<td>6/15/14</td>
<td>2/28/19</td>
<td>Griffith, William</td>
<td>$1,299,677.00</td>
<td>2016</td>
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<td>EFRI-PSBR: Microalgae Lab-ON-Chip Photobioreactor Platform for Genetic Screening and Metabolic Analysis Leading To Scalable</td>
<td>TAMU-Electrical And Computer Engineering</td>
<td>National Science Foundation</td>
<td>5/15/12</td>
<td>7/31/18</td>
<td>Han, Arum</td>
<td>$50,000.00</td>
<td>2016</td>
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<td>MRI: Development of a microfluidic flow cytometer for high-throughput non-invasive single-cell physio-chemical analysis</td>
<td>TEES-Biomedical Engineering</td>
<td>National Science Foundation</td>
<td>8/15/15</td>
<td>7/31/18</td>
<td>Han, Arum</td>
<td>$355,282.00</td>
<td>2016</td>
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<td>A High-Throughput Microfluidic In Vitro CNS Myelination Model towards Drug Screening</td>
<td>TEES-Electrical And Computer Engineering</td>
<td>National Institutes of Health</td>
<td>9/15/15</td>
<td>6/30/17</td>
<td>Han, Arum</td>
<td>$185,649.00</td>
<td>2016</td>
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<td>The TerraSwarm Research Center Award Number: M1600190</td>
<td>TEES-Biomedical Engineering</td>
<td>University of California - Berkeley</td>
<td>7/1/15</td>
<td>10/31/15</td>
<td>Jafari, Roozbeh</td>
<td>$659,000.00</td>
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<td>Ultra-Low Power Inertial MEMS for Pervasive Wearable Computing</td>
<td>TEES-Biomedical Engineering</td>
<td>National Science Foundation</td>
<td>8/9/15</td>
<td>7/31/18</td>
<td>Jafari, Roozbeh</td>
<td>$359,957.00</td>
<td>2016</td>
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<td>Using Gait and Sway Biofeedback to Reduce Falls in the Elderly</td>
<td>TEES-Biomedical Engineering</td>
<td>University of Texas-Dallas</td>
<td>10/21/15</td>
<td>8/31/16</td>
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<td>$158,451.00</td>
<td>2016</td>
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<td>Big Data Analysis of Gait Parkinson Disease</td>
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<td>University of Texas-Southwestern Medical Center</td>
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<td>8/31/16</td>
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<td>2016</td>
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<td>DP ARF Ultrasound for Monitoring Muscle Degeneration In Duchenne Muscular Dystrophy</td>
<td>AL-RSRCH-Veterinary Integrative Biosciences</td>
<td>University of North Carolina</td>
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<td>8/31/16</td>
<td>Kornegay, Joe</td>
<td>$160,383.00</td>
<td>2016</td>
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<td>Development of a Porcine Model of Duchenne Muscular Dystrophy</td>
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<td>Exemplar Genetics LLC</td>
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<td>3/31/16</td>
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<td>2016</td>
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<td>ASSESSMENT OF SAFETY/BIODISTRIBUTION/EFFICACY OF IV AAV-MICRO-DYSTROPHIN CONSTRUCT USING GRMD DOGS</td>
<td>AL-RSRCH-Veterinary Integrative Biosciences</td>
<td>University of Florida</td>
<td>9/1/15</td>
<td>8/31/16</td>
<td>Kornegay, Joe</td>
<td>$521,488.00</td>
<td>2016</td>
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<td>Image and Histopathologic Assessment of GRMD Dogs treated with AAV-microdystrophin constructs</td>
<td>AL-RSRCH-Veterinary Integrative Biosciences</td>
<td>Solid GT</td>
<td>10/1/15</td>
<td>9/30/17</td>
<td>Kornegay, Joe</td>
<td>$179,010.00</td>
<td>2016</td>
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<td>Award Title</td>
<td>Organization</td>
<td>Sponsor</td>
<td>Start Date</td>
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<td>Principal Investigator</td>
<td>Award Amount</td>
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<td>PROJECT DEVELOPMENT PLANNING Award Number: M1600559</td>
<td>AL-RSrch-Veterinary Integrative Biosciences</td>
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<td>Imaging of Whale Specimens Award Number: M1502386</td>
<td>TAMU-Texas Institute For Preclinical Studies</td>
<td>Institute for Global Health &amp; Health Policy</td>
<td>4/1/15</td>
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<td>Conserved RNA Secondary Structures in three Betacoronaviruses: MHV, BCoV, and MERS-CoV Award Number: M1601868</td>
<td>TAMHSC-Microbial Pathogenesis And Immunology</td>
<td>National Institutes of Health</td>
<td>3/15/16</td>
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<td>Leibowitz, Julian</td>
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<td>Adhesion G protein-coupled receptors in CNS development and regeneration Award Number: M1602493</td>
<td>AL-RSrch-Veterinary Integrative Biosciences</td>
<td>Boston Children’s Hospital</td>
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<td>The role of GRP56 in CNS myelination and myelin repair Award Number: M1600955</td>
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<td>Methods for Efficient Analysis of Analog ICs with Large Extracted Power Networks Award Number: M1503557</td>
<td>TEES-Electrical And Computer Engineering</td>
<td>Intel Corporation</td>
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<td>The Structural Basis for Lipid Regulation of Membrane Protein Function Award Number: M1302064</td>
<td>TAMU-Biology</td>
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<td>Lockless, Steve</td>
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<td>Neural Circuits for Reinstatement of Fear Award Number: M1601617</td>
<td>TAMU-Psychology</td>
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<td>8/1/16</td>
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<td>Maren, Stephen</td>
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<td>Effect of Alcohol Withdrawal on Pain Sensitization Award Number: M1502530</td>
<td>TAMU-Psychology</td>
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<td>Meagher, Mary</td>
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<td>Validation And Application Of A Self-Report Measure To Assess Office Ergonomic Risks Award Number: M1601242</td>
<td>TAMHSC-Environmental And Occupational Health</td>
<td>Office Ergonomics Research Committee</td>
<td>1/1/16</td>
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<td>Texas A&amp;M Health Science Center Occupational Safety &amp; Health Training Program</td>
<td>TAMHSC-Environmental And Occupational Health</td>
<td>DHHS-CDC-National Institute for Occupational Safety and Health</td>
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<td>Mehta, Ranjana</td>
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<td>Revised Force-Endurance Models for the U.S. Workforce</td>
<td>TAMHSC-Environmental And Occupational Health</td>
<td>University at Buffalo - Suny</td>
<td>9/1/15</td>
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<td>Optical Coherence Tomography to Study Effect of Poly-Drug Exposure on Fetal Brain Development</td>
<td>TAMHSC-Neuroscience &amp; Experimental Therapeutics</td>
<td>University of Buffalo - Suny</td>
<td>1/1/16</td>
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<td>Miranda, Rajesh</td>
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<td>Early Identification of Affected Children and Risk Factors for FASD in Ukraine</td>
<td>TAMHSC-Neuroscience &amp; Experimental Therapeutics</td>
<td>University of California - San Diego</td>
<td>6/1/14</td>
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<td>Prenatal microRNA neuro-therapeutics for fetal alcohol exposure</td>
<td>TAMHSC-Neuroscience &amp; Experimental Therapeutics</td>
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<td>Effect of inflammation on recovery and pain after spinal cord injury</td>
<td>TAMHSC-Neuroscience &amp; Experimental Therapeutics</td>
<td>Texas A&amp;M University</td>
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<td>Assessments of phosphatidylethanol</td>
<td>TAMHSC-Neuroscience &amp; Experimental Therapeutics</td>
<td>Department of State Health Services</td>
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<td>Development of molecular diagnostics tools for detection of Cry1Ac/Cry1Ab (Bt) resistance in bollworm (H. zea)</td>
<td>AL-RSRCH-Entomology</td>
<td>Cotton Incorporated</td>
<td>1/1/16</td>
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<td>Pietrantonio, Patricia</td>
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<td>Towards novel acaricide development against cattle fever tick: GPCR target validation and identification of chemical leads</td>
<td>AL-RSRCH-Entomology</td>
<td>USDA-National Institute of Food And Agriculture</td>
<td>2/15/16</td>
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<td>Pietrantonio, Patricia</td>
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<td>Neurosteroid Treatment for OP Intoxication</td>
<td>TAMHSC-Neuroscience &amp; Experimental Therapeutics</td>
<td>DHHS-NIH-National Institute of Neurological Disorders and Stroke</td>
<td>9/1/15</td>
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<td>Reddy, Doodipala</td>
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<td>Sprouting Capacity Upon Partial/complete Denervation in an Intermediate SMA Mouse Model</td>
<td>TAMHSC-Neuroscience &amp; Experimental Therapeutics</td>
<td>University of Southern California</td>
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<td>Psychological Wellbeing In Rodent Model of Spinal Cord Injury</td>
<td>TAMHSC-Neuroscience &amp; Experimental Therapeutics</td>
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<td>Vagus Nerve Stimulation as a Treatment Strategy for Gulf War Illness</td>
<td>TAMHSC-Surgery Temple Campus</td>
<td>DOD-Army-Medical Research and Materiel Command</td>
<td>Shapiro, Lee</td>
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<td>The Contribution of CD74 to Acquired Epilepsy</td>
<td>TAMHSC-Surgery Temple Campus</td>
<td>Citizens United for Research in Epilepsy Cure</td>
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<td>Novel Roles For NIK In High-Grade Glioma: Regulation of Mitochondrial Dynamics To Control Cell Migration and Invasion</td>
<td>TAMHSC-Molecular And Cellular Medicine</td>
<td>Cancer Prevention and Research Institute of Texas</td>
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<td>Novel roles for NIK/MAP3K14 in high-grade glioma: regulation of mitochondrial dynamics to control cell migration and invasion</td>
<td>TAMHSC-Molecular And Cellular Medicine</td>
<td>The Texas Brain and Spine Institute</td>
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<td>Opposing Roles of Distinct Output Projections From Prefrontal Cortex</td>
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<td>Smith, Rachel</td>
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<td>A biomimetic whistle for use as a bat deterrent on wind turbines</td>
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<td>University of Massachusetts - Amherst</td>
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<td>Formation and maintenance of neuromuscular synapses</td>
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<td>SJP - Toussaint: High Grade Glioma: Biology and Therapeutic Targets</td>
<td>TAMHSC-Neuroscience &amp; Experimental Therapeutics</td>
<td>St. Joseph Physician Associates</td>
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<td>Obesity, Stress, and Neuromuscular Function in the Elderly</td>
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<td>Texas A&amp;M University Health Science Center</td>
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<td>Large-Scale Reconstruction of Microvascular Networks and the Surrounding Cellular Morphology</td>
<td>AL-RSRCH-Veterinary Integrative Biosciences</td>
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<td>Large-Scale Reconstruction of Microvascular Networks and the Surrounding Cellular Morphology</td>
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<td>Regulation of Feeding Behavior by Brain-based Nutrient Sensors</td>
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<td>Collaborative Research: ARWED - Augmented Perception for Upper-Limb Rehabilitation</td>
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<td>Exoskeleton Based Stroke Rehabilitation with Augmented Reality – Cycle 7</td>
<td>TEES-Engineering Technology</td>
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<td>Actions of Manganese On Neuroendocrine Development</td>
<td>AL-RSRCH-Veterinary Integrative Biosciences</td>
<td>National Institutes of Health</td>
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<td>Dees, William</td>
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<td>The Role of Hypothalamic Neuropeptides on Biliary Function During Cholestasis</td>
<td>TAMHSC-Internal Medicine Temple Campus</td>
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<td>The Role of Hypothalamic Neuropeptides on Biliary Function During Cholestasis</td>
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<td>Identification and Characterization of Novel Therapeutics for Angelman Syndrome</td>
<td>AL-RSRCH-Vet-Pathobiology</td>
<td>University of South Florida</td>
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<td>Identification and Characterization of Novel Therapeutics for Angelman Syndrome</td>
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<td>University of South Florida</td>
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<td>Development and characterization of a pig model of Angelman syndrome</td>
<td>AL-RSRCH-Vet-Pathobiology</td>
<td>Foundation for Angelman Syndrome Foundation</td>
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<td>ASF Undergraduate Summer Research Grant for Dylan James Ritter</td>
<td>AL-RSRCH-Vet-Pathobiology</td>
<td>Autism Science Foundation</td>
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<td>The Interaction of Varenicline, Ethanol, and CNS Development</td>
<td>Neuroscience &amp; Experimental Therapeutics</td>
<td>National Institutes of Health</td>
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<td>Circadian Clocks and Neuroprotection in Response to Stroke during Reproductive Aging</td>
<td>TAMHSC-Neuroscience &amp; Experimental Therapeutics</td>
<td>American Heart - South West</td>
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<td>Graduate Student Expenses (Liu, Guo, Nunez, Chen, Banerjee, Wan, Goncalves)</td>
<td>TAMU-Biology</td>
<td>Howard Hughes Medical Institute</td>
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<td>Garcia, Luis</td>
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<td>Stress and Glucocorticoids exacerbate recovery after SCI</td>
<td>TAMU-Psychology</td>
<td>Paralyzed Veterans of America</td>
<td>8/1/14</td>
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<td>Grau, James</td>
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<td>How and when does peripheral input affect recovery after SCI</td>
<td>TAMU-Psychology</td>
<td>Craig H. Neilsen Foundation</td>
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<td>Estrogens, Ovarian Aging and Calcium Channel Modulation</td>
<td>Neuroscience &amp; Experimental Therapeutics</td>
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<td>Identification of positive allosteric modulator binding sites in a4ß2 nicotinic acetylcholine receptor</td>
<td>TAMHSC-Cop-Pharmaceutica Sciences</td>
<td>American Heart - South West</td>
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<td>Neuronal Nicotinic Acetylcholine Receptors (nAChR)s</td>
<td>TAMHSC-Cop-Pharmaceutica Sciences</td>
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<td>EFRI-PSBR: Microalgae Lab-ON-Chip Photobioreactor Platform for Genetic Screenin...</td>
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<td>Han, Arum</td>
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<td>Self-sustainable and Highly Efficient Desalination System based on Microbe-Nanostructure Hybrids</td>
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<td>Circadian clock activation and tissue specificity in Drosophila</td>
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<td>Advanced Gene Therapy for Treatment of Cardiomyopathy and Respiratory Insufficiency in Duchene Muscular Dystrophy</td>
<td>AL-RSRCH-Veterinary Integrative Biosciences</td>
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<td>Laminin-111 protein therapy for Duchenne Muscular Dystrophy</td>
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<td>Imaging of Whale Specimens</td>
<td>TAMU-Texas Institute For Preclinical Studies</td>
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<td>4/1/15</td>
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<td>Advanced Gene Therapy for Treatment of Cardiomyopathy and Respiratory Insufficiency in Duchene Muscular Dystrophy</td>
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<td>Development of a canine pelvic limb neuromusculoskeletal computer simulation gait model to characterize functional recovery following intervertebral disk herniation</td>
<td>AL-RSRCH-Vet-Pathobiology</td>
<td>University of Louisville</td>
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<td>Describing the Kinetic and Kinematic Recovery of Dachshunds with Spinal Cord Injury</td>
<td>AL-RSRCH-Vet-Pathobiology</td>
<td>American Kennel Club Canine Health Foundation</td>
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<td>Collaborative Research: The Genetic and Anatomical Determinants of Olfaction</td>
<td>AL-RSRCH-Veterinary Integrative Biosciences</td>
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<td>Role of Adhesion G protein-coupled receptors in glial cell development and</td>
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<td>Boston Children’s Hospital</td>
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<td>Role of Caspase-8 in Neuroinflammation, Demyelination and Myelination</td>
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<td>Neural Substrates of Contextual Memory In Fear Extinction Award Number:</td>
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<td>Effect of Alcohol Withdrawal on Pain Sensitization Award Number: M1502530</td>
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<td>Occupational Safety and Health Training Program Award Number: M1303223</td>
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<td>Revised Force-Endurance Models for the US Workforce Award Number: M1500485</td>
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<td>How and when does peripheral input affect recovery after SCI? Award Number: M1501832</td>
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<td>MicroRNAs as Biomarkers of Exposure and Effect in Fetal Alcohol Spectrum Disorders Award Number: M1502914</td>
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<td>MicroRNAs as Biomarkers of Exposure and Effect in Fetal Alcohol Spectrum Disorders Award Number: M1502914</td>
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<td>Analysis of the effects of aversive experience on non-defensive behaviors and underlying neural circuits Award Number: M1500304</td>
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<td>The Control On Neural Transmission By Glycosylation Award Number: M1100189</td>
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<td>Acquisition of Goods and Services Award Number: M1402918</td>
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<td>Development of Molecular diagnostics tools for detection of Cry1AC/Cry1AB(B+) resistance in bollworm (H.zea) Award Number: M1502272</td>
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<td>Neurosteroid Treatment for OP Intoxication Award Number: M1303496</td>
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<td>Genetic Analysis of Inner Ear Development in ZebrafishAward Number: M1301297</td>
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<td>Screening for melanoma genes using natural hybrid incompatibilitiesAward Number: M1500841</td>
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<td>LTREB: Social, Environmental, and Evolutionary Dynamics of Replicated Hybrid Zones in Swordtails (Teleostei: Xiphophorus) of Mexico's Sierra Madre OrientalAward Number: M1401540</td>
<td>TAMU-Biology</td>
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<td>DISSERTATION RESEARCH: Neurogenetic Framework of Condition-dependent Mate ChoiceAward Number: M1503411</td>
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<td>Usage of Song in Acoustic Monitoring of an East African Bat (Student: Grace Smarch)Award Number: M1402187</td>
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<td>Epigenetics of the Aging Astrocyte: Implications for Stroke</td>
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<td>TARCC Investigator Grant Program (&quot;IGP&quot;) Award Number: M1500876</td>
<td>TAMHSC-Hsc-Vice President For Research</td>
<td>Texas Council on Alzheimer's Disease and Related Disorder</td>
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<td>Formation and maintenance of neuromuscular synapses</td>
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<td>SJPA - Toussaint: High Grade Glioma: Biology and Therapeutic Targets Award Number: M1301145</td>
<td>Neuroscience &amp; Experimental Therapeutics</td>
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<td>Pharmacogenetic Manipulation of Dopamine D1 Receptor-Expressing Medium Spiny Neurons in the Dorsomedial Striatum and Alcohol Consumption</td>
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<td>Ethanol and Glutamatergic Transmission in the Dorsal Striatum</td>
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<td>Ethanol and Glutamatergic Transmission in the Dorsal Striatum</td>
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<td>PEPTIDE THERAPY FOR NEUROLOGICAL DISEASES Assessment of the effects of CAP peptide agonists and antagonists on animal models of multiple sclerosis and epilepsy</td>
<td>AL-RSRCH-Veterinary Integrative Biosciences</td>
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<td>A Computational Neuroscience Approach to Frontal Compensation in Decisionmaking</td>
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<td>TAMU-Poultry Science</td>
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<td>His Fat Made Him Do It: Modulation of Drosophila Courtship Behavior By An Adipose-Expressed Gene Product Award Number: M1100164</td>
<td>TAMRF-Tamu-Biology</td>
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<td>AL-RSRCH-Veterinary Integrative Biosciences</td>
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<td>Biogenic Amines Regulate Cholangiocarcinoma Cell Growth Award Number: M1000806</td>
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<td>Circadian Clocks and Neuroprotection in Response to Stroke during Reproductive Aging Award Number: M1401057</td>
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<td>Graduate Student Expenses (Liu, Guo, Nunez, Chen, Banerjee, Wan, Goncalves) Award Number: M1000574</td>
<td>TAMU-Biology</td>
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<td>Improved Definition and Prediction of Huntington’s disease motor-onset using Advanced Statistical Models Award Number: M1400522</td>
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<td>Probenecid: A potential treatment to reduce cell death, and chronic pain, after spinal cord injury</td>
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<td>EFRI-PSBR: Microalgae Lab-ON-Chip Photobioreactor Platform for Genetic</td>
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<td>IDBR: TYPE A - Microfluidic Fungal Transformation System for Ultra High-</td>
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<td>Microfluidic Platforms for High-Throughput Screening of Microbes Utilizing</td>
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<td>DP ARF Ultrasound for Monitoring Muscle Degeneration In Duchenne Muscular</td>
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<td>MGS3 as a therapeutic protein for treatment of muscular dystrophy</td>
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<td>Laminin-111 protein therapy for Duchenne Muscular Dystrophy</td>
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<td>Myostatin Inhibition In Dmd Dogs By Gene Transfer</td>
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<td>Integrated Partnership for Education, Training and Mentoring to the Doctorate</td>
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<td>PILOT SAFETY STUDY FOR MIR-124 AND A PHASE I/II TRIAL OF MIR-124 IN DOGS WITH MALIGNANT GIOMAS</td>
<td>AL-RSRCH-Vet-Small Animal Med &amp; Surg</td>
<td>University of Texas-M.D. Anderson Cancer Center</td>
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<td>Identification of Novel Small Molecules for Cns Myelin Repair</td>
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<td>Taming the Stability Challenge of Analog and Mixed-Signal Systems: Theory, Analysis and Design</td>
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<td>Software biomechanics: Does the interaction design of software impact ergonomic risk factors?</td>
<td>TAMHSC-Environmental And Occupational Health</td>
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<td>Early Identification of Affected Children and Risk Factors for FASD in Ukraine</td>
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* Texas A&M Institute for Neuroscience
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<th>Start Date</th>
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<td>Development of molecular diagnostics tools for detection of Cry1Ac/Cry1Ab (Bt) resistance in bollworm (H. zeae) Award Number: M1401675</td>
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<td>LTREB: Social, Environmental, and Evolutionary Dynamics of Replicated Hybrid Zones in Swordtails (Teleostei: Xiphophorus) of Mexico’s Sierra Madre Oriental Award Number: M1401540</td>
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<td>Epigenetics of the Aging Astrocyte: Implications for Stroke</td>
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<td>Ethanol and Glutamatergic Transmission in the Dorsal Striatum</td>
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<td>A Computational Neuroscience Approach to Frontal Compensation in Decisionmaking</td>
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