**Program Change Request**

**New Program Proposal**

Date Submitted: 08/09/18 3:13 pm

Viewing: **BS-NRSC-MCB : Neuroscience - BS , Molecular and Cellular Neuroscience**

Last edit: 11/08/18 8:27 pm

Changes proposed by: christinefarris

<table>
<thead>
<tr>
<th>Contact(s)</th>
<th>Name</th>
<th>E-mail</th>
<th>Phone</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Thomas McKnight</td>
<td><a href="mailto:mcknight@bio.tamu.edu">mcknight@bio.tamu.edu</a></td>
<td>(979) 845-3896</td>
</tr>
</tbody>
</table>

Academic level: Undergraduate  
Effective Term: 2019-2020  
Department: Biology  
College: Science  
Program type: Degree w/Concentration  
Degree designation: BS - Bachelor of Science  
With a major in: Neuroscience (NRSC)  
Associated Program: Neuroscience (NRSC)  
With a concentration in: Molecular & Cellular Neuroscience (MCB)

Catalog Program Title: Neuroscience - BS , Molecular and Cellular Neuroscience  
CIP and Fund code: 261501

**Rationale for Proposal**

Neuroscience is the study of the nervous system and its impact on behavior and cognitive functions. This interdisciplinary field integrates several disciplines, including psychology, psychiatry, biology, chemistry, and physics. It is the interdisciplinary nature of neuroscience that requires the participation of multiple units, including the Department of Biology, the Department of Psychological & Brain Sciences, and the College of Veterinary Medicine and Biomedical Sciences in collaboration with Neuroscience and Experimental Therapeutics (NEXT) in offering this degree, as well as the Texas A&M Institute for Neuroscience. Nationwide, there is increasing interest in neuroscience programs and training. Students completing a BS in Neuroscience will be well prepared for graduate study, as well as to enter entry-level healthcare and technical occupations.

Program hours: 120  
Is this program eligible for financial aid? Yes  
Program delivery mode: On-campus  
Has program funding been finalized at the department or college level? Yes  
Will new costs for the first five years of the program be under $2 million? Yes

**Catalog Program Requirements**

In Workflow:
1. BIOL Department Head  
2. Curricular Services Review  
3. SC Committee Preparer UG  
4. SC Committee Chair UG  
5. SC College Dean UG  
6. Provost  
7. UCC Preparer  
8. UCC Chair  
9. Faculty Senate Preparer  
10. Faculty Senate  
11. Provost II  
12. President  
13. External Approval  
14. Curricular Services

Approval Path:
1. 07/06/18 1:29 pm  
   Thomas McKnight (tdmcknight): Approved for BIOL Department Head  
2. 07/16/18 6:15 pm  
   Angel Mario Carrizales (carri1214): Rollback to Initiator  
3. 07/27/18 4:00 pm  
   Thomas McKnight (tdmcknight): Approved for BIOL Department Head  
4. 08/09/18 2:44 pm  
   Angel Mario Carrizales (carri1214): Rollback to Initiator  
5. 08/14/18 5:59 pm  
   Thomas McKnight (tdmcknight): Approved for BIOL Department Head  
6. 08/15/18 3:37 pm  
   Angel Mario Carrizales (carri1214): Approved for Curricular Services Review  
7. 08/16/18 10:02 am  
   Sara Thigpin (sarathigpin): Approved for SC Committee Preparer UG  
8. 08/16/18 2:29 pm  
   Lucas Macri (lmacri): Approved for SC Committee Chair UG  
9. 08/16/18 2:29 pm  
   Lucas Macri (lmacri):  
10. 08/16/18 2:29 pm  
   Lucas Macri (lmacri):  
11. 08/16/18 2:29 pm  
   Lucas Macri (lmacri):  
12. 08/16/18 2:29 pm  
   Lucas Macri (lmacri):
### Plan of Study Grid

#### First Year

<table>
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<th>Semester</th>
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<th>Course Title</th>
<th>Credits</th>
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<td>or Engineering Mathematics I</td>
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<td></td>
<td>VIBS 101</td>
<td>Neuroscience 101 2</td>
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<td>CHEM 237</td>
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#### Third Year

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<td>BIOL 434</td>
<td>Regulatory and Behavioral Neuroscience</td>
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<td>Language, philosophy and culture</td>
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#### Fourth Year

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https://nextcatalog.tamu.edu/courseleaf/approve/?role=Faculty%20Senate
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<td>NRSC 450/VIBS 450 Mammalian Functional Neuroanatomy</td>
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<td></td>
<td>POLS 206 American National Government</td>
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<td></td>
<td>Total Semester Credit Hours</td>
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</table>

1 Course must be completed by start of fifth full semester.
2 Must have a “C” or better.
3 Select any approved course in area from core curriculum list.
4 Select from BIOL 430; NRSC 300-499
5 Any course except AGLS 101; BIMS 101; BIOL 107; BIOL 113; BIOL 206; BUSN 100; CAEN 100-499; CAEX 100-499; CHEM 106; CHEM 116; HORT 101; MATH 102; STLC 100-499; WFSC 101. Only one KINE 199 can be used as a Free Elective.

Additional information
This is an interdisciplinary degree with concentrations housed in units (see BS-NRSC-BCN & BS-NRSC-TPC). Changes to this degree require a consensus document signed (can be electronic signature) by all participating units (BIOL, PBSI, CoVM, TAMIN).

Required Proposal Forms
- MOU Addendum signed.pdf
- Intent and MOU signed.pdf
- Library Support Letter.pdf
- FundinTool BS-NRSC.xlsx.xlsx
- Updated Intent signed.pdf
- NRSC Consensus-signed.pdf
- NRSC Degree-Evaluation MCB-1.docx
- NRSC THECB Certification 9-27-18.DOCx
- NRSC BOR-Agenda-Item-9-27-18.docx
- NRSC NewBachMastersForm 9-27-18.docx
- Neuroscience Appendices.pdf

Reviewer Comments
Angel Mario Carrizales (carri1214) (07/16/18 6:15 pm): Rollback: The submitted degree evaluation template and the proposed catalog program requirements do not match. Please address the following and resubmit. 1) CHEM 119, CHEM 120, STAT 303 and VIBS 101 are not listed in the template but appear in the catalog. 2) The evaluation list requirements that are not listed (e.g. “Must make a C or better” on ENGL 104). 3) NRSC 111 is listed in the template but not in the catalog. 4) The initiator and proposal’s point of contact have been emailed additional notes and recommendations for the degree evaluation template.

Angel Mario Carrizales (carri1214) (08/09/18 2:44 pm): Rollback: NRSC 111 is listed on the template however, it is not included in the plan of study grid nor on the THECB forms attached. Is this meant to be VIBS 101/NRSC 101?

Angel Mario Carrizales (carri1214) (08/15/18 3:36 pm): Initial concerns addressed, Note VIBS 101 is a new course being proposed. Per the course proposal it will be cross-listed with NRSC 101.

Deena McConnell (djm) (08/17/18 5:20 pm): Sent comments on the Agenda Item, System Proposal Form and THECB Certification Form to Dr. McKnight, Dr. Lench and Dr. Crouch. We need to ensure that all three concentrations have the same final versions of these documents.

Deena McConnell (djm) (09/24/18 4:03 pm): Comments sent to each department on the agenda item and system proposal form (same comments as sent on 8/17/18); advised by Biology that revisions are being worked on and should be completed soon.

Deena McConnell (djm) (09/27/18 4:44 pm): Agenda Item and THECB documents revised with input from each of the three departments administering the three concentrations. All agreed to and approved the revised documents. Originally submitted documents were replaced with the updated documents, all dated 9-27-18.

Sandra Williams (sandra-williams) (11/05/18 3:23 pm): UCC approved November 2018.
Texas Higher Education Coordinating Board - General Academic Institution - Program Funding Estimation Tool

Instructions
Insert the credit hours projected to be taken for all students per semester into the appropriate field. Select the discipline and level from the drop-down menus. The spreadsheet will estimate the total amounts.

Assumptions
1. Calculations are based on hours taken, not Full-Time Student Equivalent (FTSE) or headcount. This model accounts for credit hours taken at different academic levels, across various disciplines, and at different loads during the fall, spring, and summer.
2. Hours used to calculate formula funding are based on the summer and fall of even numbered years and the spring of odd numbered years. For example, summer and fall 2010 and spring 2011 (Base Year 2011) are used to allocated funds for both.
3. The program's formula funding forecast will include hours from the various disciplines that a student must take to complete the degree, not just hours from the named discipline of the program.
4. The level of the hours funded is the level of the course or the student's enrollment classification, whichever is lowest.
5. The program's new cost to the state is the funding rate reduced by the institution's estimated statutory tuition.
6. Funding is not generated for the first two years the program generates semester credit hours.
7. The funding rate is held constant into future years.
8. This model's information and assumptions are subject to change, and the estimates are not a guarantee of funding.

<table>
<thead>
<tr>
<th></th>
<th>FY 2019</th>
<th>FY 2020</th>
<th>FY 2021</th>
<th>FY 2022</th>
<th>FY 2023</th>
<th>FY 2024</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Total Student-Based Funding</strong></td>
<td>$10,664,419</td>
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<tr>
<td><strong>Student Fees (TEC, Chapters 51, 54, and 55)</strong></td>
<td>$3,080,536</td>
<td>$270,118</td>
<td>$528,957</td>
<td>$667,875</td>
<td>$806,793</td>
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<td><strong>Board Authorized Tuition</strong></td>
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<td>$ -</td>
<td>$281,250</td>
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Note: The table above converts the table below from calendar year to fiscal year. The general revenue presented above represents the estimated allocated portion based on the "Base Year." See assumption 2.
### Starting (Calendar) Year

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<th>Starting Semester</th>
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<th>2020</th>
<th>Base Year</th>
<th>Base Year</th>
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<td>$ 493,056</td>
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<td>Designated Tuition (TEC, Section 54.0513)</td>
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### Course List: Discipline Level

#### Liberal Arts
- Undergraduate Lower Level
- Undergraduate Upper Level

#### Science
- Undergraduate Lower Level
- Undergraduate Upper Level

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<td>Liberal Arts</td>
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### Estimated Formula Funding: Discipline Level

#### Liberal Arts
- Undergraduate Lower Level
- Undergraduate Upper Level

#### Science
- Undergraduate Lower Level
- Undergraduate Upper Level

### Student Fees

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MEMORANDUM

October 25, 2017

TO: Dr. Carol A. Fierke, Provost and Executive Vice President
THROUGH: Dr. Pamela R. Matthews, Dean of Liberal Arts
Dr. Meigan C. Aronson, Dean of Science
FROM: Heather C. Lench, Head of Psychological & Brain Sciences
Thomas D. McKnight, Head of Biology

SUBJECT: Proposed Bachelor of Science in Neuroscience

The Departments of Psychological & Brain Sciences (PBSI) and Biology (BIOL) propose to jointly develop and offer a Bachelor of Science degree in Neuroscience. This degree will be placed at the University level, with two initial concentrations, one in Biology and one in Psychological & Brain Sciences. As the first step in accomplishing this, we are submitting this degree planning memo, which contains the information requested in the degree proposal memo from your office dated December 6, 2016. Additional information regarding our agreement to develop this interdisciplinary degree can be found in the attached memorandum of understanding. We will be happy to discuss any further details you may need.

Title of the degree – Neuroscience

Level of the degree – Undergraduate, Bachelor of Science

CIP code – 26.1501

Date of submission – Tentatively planned for fall 2017

Description – The BS in Neuroscience degree will be housed at the University level. The core courses for this degree will include a set of courses comprising core curriculum courses required by the university, prerequisites for medical school, and basic science and psychology courses that will prepare them for upper-level electives. They will also complete a first-year seminar in neuroscience to orient them to the major and their course of study. For the concentration of the degree administered by the Department of Biology, students will choose from menus of courses focused on biological processes as well as specialized courses focused on neurobiology and neuroscience. For the concentration of the degree administered by the Department of Psychological & Brain Sciences, students will choose from menus of courses focused on psychological processes as well as specialized courses focused on neuropsychology, neuroassessment, and neuroscience. The majority of the courses associated with these concentrations already exist in the respective departments, as both departments include faculty members
with expertise in neuroscience. The development of this interdisciplinary degree will permit students to specialize in programs related to neuroscience, and we anticipate will foster collaborative instruction and research between participating departments. There is extensive interest in neuroscience across campus, and therefore other units will be interested in adding concentrations to this degree in the future as they develop resources and courses in this area.

Justification – Neuroscience is the study of the nervous system and its impact on behavior and cognitive functions. This interdisciplinary field integrates several disciplines, including psychology, psychiatry, biology, chemistry, and physics. It is the interdisciplinary nature of neuroscience that requires the participation of both the Departments of Psychological and Brain Sciences and Biology in offering this degree.

Nationwide, there is increasing interest in neuroscience programs and training. In part this interest is driven by changes in the employment market. The Bureau of Labor Statistics estimates, for the period of 2014-2024, an increase of 7.4% in demand for life, physical, and social science occupations, and a 16.4% increase in demand for healthcare practitioners and technical occupations. Together, this represents an increase of about 1.5M jobs. Peer and aspirant peer universities have developed successful undergraduate programs in neuroscience. At the University of Texas, neuroscience was first offered as an undergraduate degree in fall 2013. They had 209 neuroscience majors that first year and nearly 500 students two years later in 2015. UCLA currently has 779 neuroscience majors, and, as of Winter 2017, the University of Michigan had about 600 students enrolled in their Neuroscience program that is jointly administered by Psychology and Biology (as well as over 500 students enrolled in the neuroscience program administered solely within Psychology). We predict our program will reach similar numbers within a few years of its inception. Students completing a BS in Neuroscience will be well prepared for graduate study, as well as to enter entry-level healthcare and technical occupations.

5-year costs – Cost for initially developing and implementing the BS in Neuroscience will be minimal. A few upper-level neuroscience courses may be developed, and can be accomplished with existing facilities and faculty. Both departments will advertise and promote the new degree during its first few years. If student enrollments reach the projected levels by the 4th or 5th year of the program, participating units will require additional support for undergraduate advising to handle increased student headcount (approximately $45,000 per year, per advisor FTE). At higher levels of enrollment, we would also need to recruit faculty and instructional support for courses and research opportunities for majors (estimated at $150,000 in salary per unit in years 4 and 5). The same increase in student enrollment that would require the extra advisor, faculty, and instructional support should generate sufficient revenue to offset the salaries. Faculty members hired in neuroscience are also highly competitive for federal grant support and would contribute to emerging expertise in this area at Texas A&M University.

Implementation date – We plan to implement the program as soon as possible, and ideally in Fall of 2019.
Development of an Undergraduate Major in Neuroscience
at Texas A&M University

Memorandum of Understanding (10/25/17)

This memorandum describes the understanding of the agreement for a Bachelor of Science degree in Neuroscience at Texas A&M University. This is intended to be an interdisciplinary degree and the structure of the degree outlined below is designed to support the participating units and foster collaborations across units. This initial agreement is between the Department of Biology (BIOL), the Department of Psychological and Brain Sciences (PBSI), the College of Veterinary Medicine and Biomedical Sciences (CVM), and the Texas A&M Institute for Neuroscience (TAMIN) (hereafter, referred to as "participants"). Any additions or changes to this agreement, including new participating departments, will necessitate an update of this MOU. The purpose of any update to this MOU is to put in writing the agreement reached among participating units.

1. The participants agree to develop an interdisciplinary Bachelor of Science degree in Neuroscience. This degree will be submitted for review through CARS in FY2018 and forwarded to the Texas Higher Education Coordinating Board as soon as possible after university approval.

2. This degree will initially be administered as one B.S. degree housed at the University level, with two concentrations - one in Biology and one in Psychological & Brain Sciences. The specific names of the concentrations will be determined, but are likely to be "Molecular and Cellular Neuroscience" and "Behavioral and Cognitive Neuroscience" respectively. The College of Veterinary Medicine and Biomedical Sciences intends to develop an additional concentration for the degree shortly thereafter. The additional concentration in CVM is likely to be "Translational and Preclinical Neuroscience." Additional concentrations can be added by other interested units that contribute to undergraduate education in neuroscience. These concentrations would follow the typical routing and approval process for any proposed program, which requires support from any potentially affected units. As part of this initial MOU, the participants agree to support the future development of concentrations of the degree in the Colleges of Medicine, Veterinary Medicine and Biomedical Sciences, and Engineering.

3. Participating units will be responsible for the administration of the concentration associated with their unit (e.g., Biology will administer the degree for students associated with the BIOL concentration). This will include advising, scheduling courses, providing learning and research opportunities, assessing learning outcomes, and encouraging timely graduation.
4. Recruitment events and opportunities for engagement outside the classroom (e.g., speaker series, undergraduate research conferences, graduation events, community outreach) will be jointly administered by the participating units whenever possible.

5. The participating units will be credited with students registered in their respective concentrations as students pursuing a major degree, and in any counts of number of students per faculty member (e.g., Veterinary Medicine and Biomedical Sciences is credited with students pursuing a Neuroscience degree in their concentration).

6. The prefix for the degree will be determined in collaboration with the Registrar’s office, with the intent of avoiding confusion with students or administration with existing degrees.

7. This is intended to be an interdisciplinary degree that trains students in the foundations of neuroscience techniques and brain function, and that fosters collaboration among units. Accordingly, the curriculum for this degree will be jointly determined, as follows:
   - The recommended core curriculum for this degree will be developed by a TAMIN-designated committee with the expertise required in foundational knowledge in neuroscience across disciplines. Each concentration of the degree will be developed by committees designated within each respective participating unit and informed by the recommended core curriculum.
   - The initially participating units will coordinate to forward the degree plans simultaneously. Other participating units and TAMIN should be included in the CARS system notification list for each concentration of the degree.
   - The degree will include a first year seminar, coordinated by TAMIN, that involves advisors and/or faculty involved in neuroscience across campus. If TAMIN is not able to provide this support, then each unit will take turns coordinating this seminar. The seminar should serve to orient students to the degree, make students aware of their options for study and career paths, and foster a sense of community and joint participation.
   - Upper-level neuroscience students will be encouraged to participate in TAMIN seminars and other activities, as appropriate.
   - During initial curriculum development and as new courses are proposed for the degree, units and faculty will consider the development of co-taught courses across units as part of the curriculum. These arrangements require pre approval by each Department Head or Assistant Dean, as appropriate.
   - Each concentration in the degree should include opportunities that give students hands-on experiences with neuroscience techniques and methodology. These opportunities could include research experiences in faculty labs that can count as part of the degree electives or laboratory components of NRSC courses.
8. Each participating unit is responsible for managing enrollment in their respective concentration based on available instructors and resources to support the degree. Each participating unit is responsible for setting requirements and prerequisites for students in or seeking to enter their respective concentration.

Thomas D. McKnight  
Head, Department of Biology

Heather C. Lench  
Head, Department of Psychological & Brain Sciences

Elizabeth Crouch  
Assistant Dean for Undergraduate Education, College of Veterinary Medicine & Biomedical Sciences

Meigan C. Aronson  
Dean, College of Science

Pamela R. Matthews  
Dean, College of Liberal Arts

Eleanor M. Green  
Carl B. King Dean of Veterinary Medicine  
College of Veterinary Medicine & Biomedical Sciences

Michael Smotherman,  
Chair, Texas A&M Institute of Neuroscience
10 April 2018

AMENDMENT TO:
Development of an Undergraduate Major in Neuroscience
at Texas A&M University
Memorandum of Understanding (10/25/17)

This document is an amendment to the memorandum of understanding (MOU) dated 25 October 2017 that outlines procedures and policies necessary to establish and govern the interdepartmental BS degree in neuroscience at Texas A&M University.

The purpose of this amendment is to clarify procedures that will be taken whenever any participating unit wishes to change the curriculum in their respective concentration. The clarification documented below restores and simplifies language that appeared in early drafts of the MOU but was removed from the final version.

The first bullet point under section 7 of the MOU currently reads

- The recommended core curriculum for this degree will be developed by a TAMIN-designated committee with the expertise required in foundational knowledge in neuroscience across disciplines. Each concentration of the degree will be developed by committees designated within each respective participating unit and informed by the recommended core curriculum.

This bullet point will now read

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Butler Hall 100
3258 TAMU
College Station, Texas 77843-3258
Tel. 979.845.7747  Fax. 979.845.2891
www.bio.tamu.edu
capstone course). Any major changes must have the consensus of the heads of all participating departments and the chair of TAMIN.

This amendment is ratified by signatures below from the currently participating units. Additional units wishing to develop a new concentration within the NRSC BS will be required to recognize and ratify this amendment to the original memorandum of understanding.

Michael Smotherman  
Chair of the Texas A&M Institute of Neuroscience

Heather C. Lench  
Head of Psychological and Brain Sciences

Thomas D. McKnight  
Head of Biology
## B.S. in Neuroscience – Draft 4/23/18

<table>
<thead>
<tr>
<th>Degree Code</th>
<th>Molecular &amp; Cellular Neuroscience</th>
<th>Behavioral &amp; Cognitive Neuroscience</th>
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<tr>
<td></td>
<td>NRSC-BIOL Biology</td>
<td>NRSC-PBSI Psychological &amp; Brain Sciences</td>
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<tr>
<td>Initiating Department</td>
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<tr>
<td>Foundations in Neuroscience (5 credit hours)</td>
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<tr>
<td></td>
<td>• NRSC 101 – overview seminar &amp; orientation (1 credit)</td>
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<td></td>
<td>• NRSC 102 – continuation of above (1 credit)</td>
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<tr>
<td></td>
<td>• NRSC/PSYC 335 Physiological Psychology (3 credits)</td>
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<td>Supporting Foundational Coursework (30 credit hours)</td>
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<td>Social Science Elective: PSYC 107 – Introduction to Psychology (3 credits)</td>
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<td>Supporting Life &amp; Physical Sciences:</td>
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<tr>
<td></td>
<td>• BIOL 111 – Introductory Biology I (4 credits, lecture + lab)</td>
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<td>• BIOL 112 – Introductory Biology II (4 credits, lecture + lab)</td>
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<td></td>
<td>• CHEM 101 – Fundamentals of Chemistry I (3 credits)</td>
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<td>• STAT 302 – Statistical Methods (3 credits)</td>
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<td></td>
<td>• CHEM 227 – Org Chem (3 credits)</td>
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<td>• CHEM 228 – Org Chem II (3 credits)</td>
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<td>• BIOL 213 – Mol Cell Bio (3 credits)</td>
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<td>• BIOL 413 Cell Biology (3 Credits)</td>
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<td>• BICH 410 – Comp Biochem (3 credits)</td>
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<td>• NRSC/BIOL 338 – Animal Phys (4 credits)</td>
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<td>• NRSC/BIOL 434 Neurobio (3 credits)</td>
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<td>• NRSC/PSYC 320 – Sens &amp; Perc (3 credits)</td>
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<td>• NRSC/PSYC 332 – Learn &amp; Mem (3 credits)</td>
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<tr>
<td>Core Curriculum Coursework (30-32 credit hours)</td>
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<td></td>
<td>• Eng 104 (3 credits)</td>
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<td></td>
<td>• Comm elective (3 credits)</td>
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<td>• Math 147 or 151 (3-4 credits)</td>
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<td>• Math 148 or 152 (3-4 credits)</td>
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<td>• POLS 207 (3 credits)</td>
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<tr>
<td>Creative Arts (3 credits)</td>
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<td>• Lang, Phil, Culture (3 credits)</td>
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<td>• Any 300- or 400-level NRSC course</td>
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<tr>
<td></td>
<td>• BIOL 430 Biological Imaging (3 credits)</td>
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<td>• BIOL 4xx – Neural Dev (3 credits)</td>
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| Free Electives (9-11 credit hours) | Select from course lists |

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<tr>
<td>NRSC/PSYC 333 – Bio of D/o (3 credits)</td>
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<td>NRSC/PSYC 350 – Cog Neuro (3 credits)</td>
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<td>NRSC/PSYC 340 – Psyc of Learn (3 credits)</td>
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<tr>
<td>NRSC/PSYC 360 Health Psych (3 credits)</td>
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<td>NRSC/PSYC 311 Animal Beh (3 credits)</td>
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<tr>
<td>NRSC/PSYC 336 Drugs &amp; Beh (3 credits)</td>
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<th>Select from:</th>
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<td>• Choose additional from Menu A</td>
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<tr>
<td>• Any 300- or 400-level NRSC course</td>
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<tr>
<td>• NRSC/PSYC 331 Social Neuro (3 credits)</td>
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<tr>
<td>• NRSC 289 Special Topics in... (1-4 credits)</td>
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<td>• PSYC 484 (3 credits; can be repeated)</td>
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<td>• PSYC 485 (3 credits; can be repeated)</td>
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<td>• PSYC 491 (3 credits; can be repeated)</td>
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<td>• NRSC 485 (3 credits; can be repeated)</td>
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<td>• NRSC 491 (3 credits; can be repeated)</td>
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Total hours 120
Dear Dr. Lench:

The Texas A&M University Libraries can readily support the proposed new BS program in Neuroscience. This program will not require additional library resources, because the library has steadily acquired materials in Neuroscience and related fields as part of its aggressive growth campaign.

The Libraries maintain subscriptions to top tier journals in the field along with access to key indices and databases like Neurosciences Abstracts, BIOSIS, Web of Science, Scopus, MEDLINE, PubMed, PsycINFO, PsycARTICLES, PsycCRITIQUES, Psychology and Behavioral Sciences Collection, Academic Search Ultimate, and Animal Behavior Abstracts.


Hundreds of print and e-books, media items and streaming media are added to the collection every year through individual purchases or included in various e-book packages and streaming services. PsycBOOKS, EBSCO eBook Collection and Hathitrust Digital Library are available along with streaming video and sound resources such as PsycTHERAPY, Counseling and Therapy in Video, SAGE Video, Kanopy Streaming Video, and Anatomy.tv. Additionally, over 30,000 titles in various video/file formats in the Media and Reserves unit can be studied either in the library or checked out.

The library has a collaborative approach to purchasing key resources in a field of study by encouraging and supporting faculty and student recommendations for new resources. Recommendations for databases, scholarly journals, and monographs can be made through the assigned library subject librarian or the library’s online “Suggest a Purchase” form. The annual library materials expenditures are over $14.5 million for journal and database subscriptions and over $2 million for print and e-book purchases. Overall, the University Libraries’ collection includes 1,712
databases, approximately 121,838 unique serial titles, and over 5.6 million volumes, which will adequately support the proposed BS program in Neuroscience.

Texas A&M University Libraries is a member of the Association of Research Libraries (ARL). This distinct membership is based on TAMU Libraries distinct collections, commitment to servicing the scholarly community, and leadership. In addition, TAMU Libraries currently holds membership in the Greater Western Alliance (GWLA), which allows our campus users access to the holdings of 37 other research libraries located across the United States. Another important consortium membership includes the Center for Research Libraries (CRL) whose mission is to foster and advance scholarly inquiry by granting members access to its five million newspapers, journals, dissertations, and digital resources.

To summarize, the Texas A&M University Libraries is committed to supporting a new BS program in Neuroscience.

Sincerely yours,

[Signature]

David Carlson
Dean of University Libraries
10 April 2018

AMENDMENT TO:
Development of an Undergraduate Major in Neuroscience
at Texas A&M University
Memorandum of Understanding (10/25/17)

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Michael Smotherman
Chair of the Texas A&M Institute of Neuroscience

Heather C. Lench
Head of Psychological and Brain Sciences

Thomas D. McKnight
Head of Biology

Elizabeth Crouch
Assoc. Dean College of Vet Med & Biomedical Sciences
APPENDIX A

Course Descriptions
and
Prescribed Sequence of Courses
Course Descriptions and Prescribed Sequence of Courses

**BI CH 410 Comprehensive Biochemistry I**  Structure, function and chemistry of proteins and carbohydrates; kinetics, mechanisms and regulation of enzymes; metabolism of carbohydrates. Not open to biochemistry or genetics majors.

**BI CH 411 Comprehensive Biochemistry II**  A continuation of BI CH 410. Structure, function, chemistry and metabolism of lipids and nucleic acids; cellular metabolism viewed from the standpoint of energetics and control mechanisms; interrelationships of metabolic pathways. Not open to biochemistry or genetics majors.

**BI CH 431/GENE 431 Molecular Genetics**  Molecular basis for inheritance; gene structure and function, chromosomal organization, replication and repair of DNA, transcription and translation, the genetic code, regulation of gene expression, genetic differentiation and genetic manipulations.

**BI OL 111 Introductory Biology I**  First half of an introductory two-semester survey of contemporary biology that covers the chemical basis of life, structure and biology of the cell, molecular biology and genetics; includes laboratory that reinforces and provides supplemental information related to the lecture topics.

**BI OL 112 Introductory Biology II**  The second half of an introductory two-semester survey of contemporary biology that covers evolution, history of life, diversity and form and function of organisms; includes laboratory that reinforces and provides supplemental information related to the lecture topics.

**BI OL 213 Molecular Cell Biology**  Explores the molecular basis of cell structure, function and evolution; gene regulation, cell division cycle, cancer, immunity, differentiation, multicellularity and photosynthesis; may not take concurrently with, or after the completion of BI OL 413.

**BI OL 413 Cell Biology**  Structure, function, and biogenesis of cells and their components; interpretation of dynamic processes of cells, including protein trafficking, motility, signaling and proliferation.

**BI OL 423 Cell Biology Laboratory**  Modern methods of study of cell structure and cell function.

**BI OL 430 Biological Imaging**  Still and video photography and photomicrography, computer-based digital image analysis and processing of biological images; theory and principles of light and electron microscopy including transmission and scanning electron microscopy; optical contrast methods for light microscopy including phase contrast, DIC, polarizing light and confocal laser scanning microscopy.

**BI OL 435 Laboratory for Regulatory and Behavioral Neuroscience**  Study of modern methods and tools used to investigate nervous system structure and function.

**CHEM 119**  Introduction to modern theories of atomic structure and chemical bonding; chemical reactions; stoichiometry; states of matter; solutions; equilibrium; acids and bases; coordination chemistry; methods and techniques of chemical experimentation; qualitative and semiquantitative procedures applied to investigative situations.

**CHEM 120**  Theory and applications of oxidation-reductions systems; thermodynamics and kinetics; complex equilibria and solubility product; nuclear chemistry; descriptive inorganic and organic chemistry; introduction to analytical and synthetic methods and to quantitative techniques to both inorganic and organic compounds with emphasis on an investigative approach.
CHEM 227 Organic Chemistry I  Organic Chemistry I. Introduction to chemistry of compounds of carbon; general principles and their application to various industrial and biological processes.

CHEM 228 Organic Chemistry II  Organic Chemistry II. Continuation of CHEM 227.

CHEM 237 Organic Chemistry Laboratory  Organic Chemistry Laboratory. Operations and techniques of elementary organic chemistry laboratory; preparation, reactions and properties of representative organic compounds.

CHEM 238 Organic Chemistry Laboratory  Organic Chemistry Laboratory. Continuation of CHEM 237.

GENE 302 Principles of Genetics  Mechanisms of inheritance, stressing the conservation of fundamental genetic processes throughout evolution, from bacteria to humans; mutations and phenotypes, Mendelian genetics, population genetics and evolution, and complex inheritance. Course designed for biochemistry, genetics and all majors in biology. Only one of the following will satisfy the requirements for a degree: GENE 301, GENE 302, GENE 315 and GENE 320/BIMS 320.

GENE 320/ BIMS 320 Biomedical Genetics  Fundamental genetic principles as applied to biomedical science; Mendelian inheritance, linkage and genetic mapping, mutagenesis and pedigree analysis; molecular basis of gene function and inherited disease; gene therapy and genetic counseling. Only one of the following will satisfy the requirements for a degree: GENE 301, GENE 302, GENE 315 or GENE 320/BIMS 320.

KINE 406 Motor Learning and Skill Performance  Learning in psychomotor domain; motor learning theories, physiological bases of skill behavior, motor and skill learning, state of performer and application of instructional techniques in motor learning and skill performance.

NRSC/ VIBS 101 Neuroscience 101  An introductory survey of neuroscience for freshmen undergraduate students on the basic neuroscience core ideas and neurological disorders.

NRSC/ PSYC 235 Introduction to Behavioral & Cognitive Neuroscience  Physiological bases of sensation, motor functions, emotion, motivation and complex psychological processes.

NRSC/ VIBS 277 Introduction to Neuroscience  Neuroscience from the molecular to system levels; fundamental principles and knowledge of neuroscience; current research information on neuroscience.

NRSC 311/ PSYC 311 Psychology of Animal Behavior  Problems, principles, and methods of animal psychology; animal learning, motivation, discriminative processes and abnormal, social and instinctual behaviors.

NRSC 320/ PSYC 320 Sensation-Perception  Review of sensory physiology, sensory and perceptual phenomena and the major perceptual theories; current research in the field.

NRSC 332/ PSYC 332 Neuroscience of Learning and Memory  Brain mechanisms of learning and memory from molecular to behavioral levels; synaptic plasticity, model systems, multiple memory systems, diseases of learning and memory.

NRSC 333/ PSYC 333 Biology of Psychological Disorders  Neurobiology and clinical explanation of molecular mechanisms underlying psychiatric disorders and their drug treatments; depression and bipolar, anxiety disorders, mood disorders, psychosis and schizophrenia.
NRSC 336/PSYC 336 Drugs and Behavior Physiological, pharmacological and behavioral effects of psychoactive drugs, including short-term and long-term effects of psychoactive drugs, properties of addictive drugs, etiology of addiction, and treatments of drug addiction and withdrawal.

NRSC 340/PSYC 340 Psychology of Learning Survey of significant concepts, experimental methods and principles of learning.

NRSC 350/PSYC 350 Cognitive Neuroscience Research in cognitive neuroscience; methodological advances that enable the study of the human brain safely in the laboratory; complex aspects of the mind like emotion, social behavior and consciousness.

NRSC 360/PSYC 360 Health Psychology and Behavioral Medicine Health psychology emphasizing behavioral and lifestyle factors in health and illness, prevention and modification of health-compromising behaviors, health care utilization, and psychological management of chronic disorders and psychological management of chronic disorders and terminal illnesses.

NRSC 401/VIBS 401 Developmental Neurotoxicology Effects of exposure to toxic substances on the developing nervous system; content to include mechanisms of toxicity of substances potentially devastating to the developing nervous system including lead, mercury and other heavy metals, alcohol, nicotine (smoking), pesticides, flame retardants, and others.

NRSC 407/VIBS 407 Core Ideas in Neuroscience General overview of selected core ideas across the full spectrum of neuroscience.

NRSC 434/BIOL 434 Regulatory and Behavioral Neuroscience Cell biology and biophysics of neurons; functional organization of the vertebrate nervous system; physiological basis of behavior.

NRSC 440/PSYC 440 Hormones and Behavior Principles of hormones and the endocrine system; relationships among hormones, the nervous system and a variety of behaviors in vertebrates including humans.

NRSC 450/VIBS 450 Mammalian Functional Neuroanatomy Functional morphology of the domestic animal and human brain using gross specimens, microscopic sections, interactive computer-, DVD-, and video-assisted instructional programs supplemented with clinical case studies.

NRSC 485 Directed Studies Directed readings or research problems in selected areas designed to supplement existing course offerings conducted under the direction of a member of the Faculty of Neuroscience. May be repeated for credit.

NRSC 489 Special Topics in... Selected topics in an identified area of neuroscience. May be repeated for credit.

NRSC 491 Research Research conducted under the direction of a member of the Faculty of Neuroscience. May be repeated for credit.


PHYS 202 College Physics Continuation of PHYS 201. Fundamentals of classical electricity and light; introduction to contemporary physics.

PHYS 218 Mechanics Mechanics for students in science and engineering.
PHYS 208 Electricity and Optics  Continuation of PHYS 218. Electricity, magnetism, and introduction to optics. Primarily for students in science and engineering.

PSYC 471 Research Writing in Neuroscience  Processes of sharing research findings and technical reports within neuroscience to a professional or general audience; includes written assignments to develop writing skills related to conveying research findings.

PSYC 475 Communicating Neuroscience Concepts  Processes of sharing neuroscience ideas and concepts to a professional or general audience; includes written assignments to develop writing skills related to conveying topics.

PSYC 484 Field Experiences  Participation in an approved mental health, mental retardation, school, industrial or other approved setting; field experiences supervised by an appropriate professor within an area of student interest; course requirements vary with the setting, the supervising professor and the needs of the individual student. May be repeated for credit.

PSYC 485 Directed Studies  Directed readings or research problems in selected areas designed to supplement existing course offerings. May be repeated for credit.

PSYC 491 Research  Research conducted under the supervision of a chosen faculty member in the department of psychology; involves discussion and presentation of student research projects. May be repeated for credit.

STAT 302 Statistical Methods  Intended for undergraduates in the biological sciences. Introduction to concepts of random sampling and statistical inference; estimation and testing hypotheses of means and variances; analysis of variance; regression analysis; chi-square tests.

VIBS 343 Histology  Normal tissues of vertebrates including histogenesis of some; histogenesis and organography of mammalian tissues.

VIBS 408 Neuroscience and Religion  Emphasis on the biology of the human mind in the context of religious implications.

VIBS 422 Endocrine Toxicology  Impacts of endocrine toxicology on endocrine system; prevalence, environmental and occupational use and disposal of environmental endocrine disrupting chemicals (EDCs); structure, toxicokinetics and mechanism of action of EDCs; effects of EDCs on the development and function, disorders and diseases of the endocrine and reproductive organs.

VIBS 424/VTPP 424 Biomedical Neuroendocrinology and Endocrine Disorders  Neuroendocrine (hypothalamus-pituitary) control of puberty, menstruation, ovulation, pregnancy, labor, lactation, female reproductive cycles, male reproductive functions, thyroid and parathyroid, adrenal and kidney, diabetes, obesity, sleep, memory, learning and aging and their endocrine disorders; overview on biosynthesis, transport and signaling of peptide and neuropeptide hormones, steroids and prostaglandins.

VIBS 443 Biology of Mammalian Cells and Tissues  Molecular phenomena placed in context with tissues, organs and organ systems; cell and tissue structures visualized by light microscopy and electron micrographs for functional relationships; clinical correlations reveal relevance of histology in specific disease states; conceptual thinking exercises facilitate problem solving skills.

VIBS 447 Neurophysiology of Music  Exploration of the heritability and genetics of musical talent, the physiology and physics of hearing, and the neurophysiology of processing sound using primarily German and Austrian compositions. Must be taken on a satisfactory/unsatisfactory basis.
**VIBS 485 Directed Studies**  Directed individual study of a selected problem in veterinary anatomy (with emphasis on neuroscience, cell biology, reproduction, developmental biology, marine mammal anatomy) approved by instructor or selected problems in veterinary public health (with emphasis on food safety, toxicology, epidemiology, informatics, zoonoses).

**Prerequisites:** Junior or senior classification and approval of instructor.

**VTPP 323 Physiology of Domestic Animals**  Physiology essential to understanding of life processes. For students in agriculture and related fields.

**VTPP 425 Pharmacology Credits 3. 3 Lecture Hours.**  Introduction to pharmacokinetics and pharmacodynamics; survey of major pharmaceutical classes; uses, mechanisms of action and adverse reactions of selected agents.
### Sequence of courses for the NRSC-BCN Track

<table>
<thead>
<tr>
<th>First Year Fall</th>
<th>Semester Credit Hours</th>
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<tbody>
<tr>
<td>BIOL 111</td>
<td>Introductory Biology I</td>
</tr>
<tr>
<td>CHEM 119</td>
<td>Fundamentals of Chemistry I</td>
</tr>
<tr>
<td>NRSC227</td>
<td>Introduction to Neuroscience</td>
</tr>
<tr>
<td>PSYC 107</td>
<td>Introduction to Psychology</td>
</tr>
<tr>
<td>NRSC 101</td>
<td>Neuroscience 101</td>
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<th>First Year Spring</th>
<th>Semester Credit Hours</th>
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<tbody>
<tr>
<td>BIOL 112</td>
<td>Introductory Biology II</td>
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<tr>
<td>CHEM 120</td>
<td>Fundamentals of Chemistry II</td>
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<td>MATH 147</td>
<td>Calculus I for Biological Sciences</td>
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<tr>
<td>NRSC 235</td>
<td>Introduction to Behavioral and Cognitive Neuroscience</td>
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<td>American history elective</td>
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<td>Government/Political Science elective</td>
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<td>Concentration elective$^3$</td>
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<tr>
<td>Government/Political Science elective</td>
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<td>Concentration elective$^3$</td>
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<td>Concentration elective$^3$</td>
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<tr>
<td>Language, philosophy and culture elective</td>
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<td>Concentration elective$^3$</td>
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<td>Semester Credit Hours</td>
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</table>

| Total Semester Credit Hours            | 120                   |

1 Students seeking teacher certification must take HIST 105 and HIST 106. Other students may choose HIST 105 and HIST 106 or any 6 hours of American history courses (3 hours may be in Texas history).

2 Students successfully completing the required four semesters of upper-level ROTC courses may substitute these courses for 3 hours of American history and 3 hours of government/political science.

3 Two courses in the major must be designated as writing intensive.

4 Select from any 100-499 course not used elsewhere. Only one KINE 199 may be used as a general elective.
**Sequence of courses for the NRSC-MCB Track**

<table>
<thead>
<tr>
<th>First Year Fall</th>
<th>Semester Credit Hours</th>
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<tbody>
<tr>
<td>BIOL 111 Introductory Biology I</td>
<td>4</td>
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<tr>
<td>CHEM 119 Fundamentals of Chemistry I</td>
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<tr>
<td>MATH 147 Calculus I for Biological Sciences</td>
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<tr>
<td>PSYC 107 Introduction to Psychology</td>
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<tr>
<td>NRSC 101 Neuroscience 101</td>
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<table>
<thead>
<tr>
<th>First Year Spring</th>
<th>Semester Credit Hours</th>
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</thead>
<tbody>
<tr>
<td>BIOL 112 Introductory Biology II</td>
<td>4</td>
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<tr>
<td>CHEM 120 Fundamentals of Chemistry II</td>
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<tr>
<td>MATH 148 Calculus II for Biological Sciences</td>
<td>4</td>
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<tr>
<td>NRSC 235 Introduction to Behavioral and Cognitive Neuroscience</td>
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<td><strong>Semester Credit Hours</strong></td>
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<thead>
<tr>
<th>Second Year Fall</th>
<th>Semester Credit Hours</th>
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<tbody>
<tr>
<td>BIOL 213 Molecular Cell Biology</td>
<td>3</td>
</tr>
<tr>
<td>CHEM 227 Organic Chemistry I</td>
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<td>&amp; CHEM 237 Organic Chemistry Laboratory I</td>
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<tr>
<td>PHYS 201 College Physics I</td>
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</tr>
<tr>
<td>American history elective 1,2</td>
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<thead>
<tr>
<th>Second Year Spring</th>
<th>Semester Credit Hours</th>
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</thead>
<tbody>
<tr>
<td>NRSC 227 Introduction to Neuroscience</td>
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<tr>
<td>CHEM 228 Organic Chemistry II</td>
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<tr>
<td>&amp; CHEM 238 Organic Chemistry Laboratory</td>
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<tr>
<td>PHYS 202 College Physics II</td>
<td>4</td>
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<tr>
<td>American history elective 1,2</td>
<td>3</td>
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<tr>
<td><strong>Semester Credit Hours</strong></td>
<td><strong>14</strong></td>
</tr>
</tbody>
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2 Students successfully completing the required four semesters of upper-level ROTC courses may substitute these courses for 3 hours of American history and 3 hours of government/political science.

The following are Common Body of Knowledge courses and must be completed prior to the start of 5th full semester: BIOL 111, BIOL 112, BIOL 213, CHEM 119, CHEM 120, CHEM 227 & CHEM 237, CHEM 228 & CHEM 238, MATH 147, MATH 148.

<table>
<thead>
<tr>
<th>Third Year Fall</th>
<th>Semester Credit Hours</th>
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<tbody>
<tr>
<td>BICH 410 Comprehensive Biochemistry I</td>
<td>3</td>
</tr>
<tr>
<td>NRSC 434 Neurobiology</td>
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<tr>
<td>Course Code</td>
<td>Course Title</td>
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<tr>
<td>STAT 302</td>
<td>Statistical Methods</td>
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<tr>
<td></td>
<td>Communication Elective</td>
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<tr>
<td>Elective</td>
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<td>Semester Credit Hours</td>
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Third Year Spring

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<tr>
<th>Course Code</th>
<th>Course Title</th>
<th>Semester Credit Hours</th>
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<tbody>
<tr>
<td>BICH 411</td>
<td>Comprehensive Biochemistry II</td>
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</tr>
<tr>
<td>NRSC 435</td>
<td>Neurobiology Laboratory</td>
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<td></td>
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<tr>
<td>Elective</td>
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<tr>
<td>POLS 206</td>
<td>American National Government</td>
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Fourth Year Fall

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<th>Course Title</th>
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<tbody>
<tr>
<td>BIOL 413</td>
<td>Cell Biology</td>
<td>3</td>
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<tr>
<td>POLS 207</td>
<td>Texas State and Local Government</td>
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<tr>
<td>NRSC 450</td>
<td>Functional Neuroanatomy</td>
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<td>Elective</td>
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<tr>
<td></td>
<td>Language, philosophy and culture elective</td>
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<td>Semester Credit Hours</td>
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Fourth Year Spring

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<th>Course Title</th>
<th>Semester Credit Hours</th>
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<tbody>
<tr>
<td>BIOL 388</td>
<td>Animal Physiology</td>
<td>4</td>
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<td>Any BIOL or NRSC course</td>
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<td>Creative arts elective</td>
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<tr>
<td>BIOL 430</td>
<td>Biological Imaging</td>
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<td>Elective</td>
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<tr>
<td></td>
<td>Semester Credit Hours</td>
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</table>

Total Semester Credit Hours

120

3 Two courses in the major must be designated as writing intensive.

4 Select from any 100-499 course not used elsewhere. (Except AGLS 101, BIMS 101; BIOL 101, BIOL 107, BIOL 113, BIOL 206; BUSN 100; CAEN 101-499; CHEM 106, CHEM 116; HORT 101; MATH 102; STLC 100-499; WFSC 101.) Only one KINE 199 may be used as a general elective.

5 Students successfully completing the required four semesters of upper-level ROTC courses may substitute these courses for 3 hours of American history and 3 hours of government/political science.

Sequence of courses for the NRSC-TPC Track

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<th>Course Code</th>
<th>Course Title</th>
<th>Semester Credit Hours</th>
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<td>BIOL 111</td>
<td>Introductory Biology I</td>
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</tr>
<tr>
<td>CHEM 119</td>
<td>Fundamentals of Chemistry I</td>
<td>4</td>
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<tr>
<td>MATH 147</td>
<td>Calculus I for Biological Sciences</td>
<td>4</td>
</tr>
<tr>
<td>NRSC 101</td>
<td>Neuroscience 101</td>
<td>1</td>
</tr>
<tr>
<td>American history elective</td>
<td>3</td>
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<tr>
<td>Semester Credit Hours</td>
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<tr>
<td><strong>First Year Spring</strong></td>
<td>Semester Credit Hours</td>
<td></td>
</tr>
<tr>
<td>BIOL 112 Introductory Biology II</td>
<td>4</td>
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<tr>
<td>CHEM 120 Fundamentals of Chemistry II</td>
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<tr>
<td>Communication elective</td>
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<td>PHYS 202 College Physics II</td>
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<td>NRSC 235 Introduction to Behavioral and Cognitive Neuroscience</td>
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<th>Course Title</th>
<th>Credit Hours</th>
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<tr>
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<td>3</td>
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<td>Neurobiology</td>
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<td>Free Elective</td>
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<td>GENE 302</td>
<td>Principles of Genetics</td>
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<td>POLS 207</td>
<td>Texas State and Local Government</td>
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<td>NRSC 450</td>
<td>Functional Neuroanatomy</td>
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<td>Prescribed concentration elective</td>
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<td>Language, philosophy and culture elective</td>
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<td>Cultural Discourse elective</td>
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<td>Total Semester Credit Hours</td>
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3 Two courses in the major must be designated as writing intensive.

4 Select from any 100-499 course not used elsewhere. (Except AGLS 101, BIMS 101, BIOL 101, BIOL 107, BIOL 113, BIOL 206; BUSN 100; CAEN 101-499; CHEM 106, CHEM 116; HORT 101; MATH 102; STLC 100-499; WFSC 101.) Only one KINE 199 may be used as a general elective.

5 Students successfully completing the required four semesters of upper-level ROTC courses may substitute these courses for 3 hours of American history and 3 hours of government/political science.
APPENDIX B

Five-Year Faculty Recruiting Plan
Hiring Schedule
Across concentrations, we project the need for one total additional faculty member in Year 4 of the program, with the specific area to be determined by student enrollment across the concentrations, or targeting an interdisciplinary neuroscience hire. Therefore the ad below is an example of what would be used in October of Year 3 to recruit this faculty member, with the exact hiring unit and expertise to be determined.

**FACULTY POSITION IN NEUROSCIENCE - TEXAS A&M UNIVERSITY.** The Department of XXX at Texas A&M University invites applications for a tenure-track faculty position in neuroscience. The position will be at the rank of Assistant Professor, with an anticipated start date of Fall, XXX. We are interested in scholars conducting research in any area related to neuroscience, and value innovation and excellence in methodological, quantitative, and computational approaches. This position will complement a world-class core of neuroscience researchers, many of whom participate in cross-cutting research concentrations across the university. Applicants should have an outstanding record of research achievement, evidence of or potential for extramural research funding, and a strong commitment to undergraduate and graduate education. The successful candidate will teach undergraduate and graduate courses in their area of expertise. A Ph.D. in neuroscience or a closely related field is required.

The Department of XXX is a community of scholars committed to generating scientific discoveries in the discipline, providing rigorous undergraduate and graduate education, and engaging in outreach about neuroscience and the application of science. We offer doctoral programs in xxx. We currently have xxx full-time faculty, over xxx graduate students, and almost xxx undergraduate majors. Our research laboratories are housed in xxx. We maintain collaborations with multiple local agencies, including the Texas A&M Institute for Preclinical Studies, the Texas A&M Institute for Neuroscience, and xxx. Texas A&M University is a land, sea, and space grant institution that holds the distinction of classification as an R1 Doctoral University (Highest Research Activity), and faculty benefit from the resources and support associated with this designation. A recipient of an NSF ADVANCE award to promote equity, Texas A&M University is an Equal Opportunity/Affirmative Action/Veterans/Disability Employer committed to diversity and broadening participation in higher education, and has a policy of being responsive to the needs of dual-career couples. The Department is interested in candidates who, through their research, teaching, and/or service, will contribute to the breadth and excellence of the academic community, as well as the educational needs of the population of Texas and the global community.

To apply, please email a letter of intent, curriculum vitae, statements on research and teaching, and 3 sample research publications to xxx. The search committee will begin reviewing applications xxx and will continue to review new applications until the position is filled.
APPENDIX C

Institution’s Policy on Faculty Teaching Load
Institution’s Policy on Faculty Teaching Load

Teaching Load is set at the University level. The full policy is available at: https://dars.tamu.edu/files/workload-policy

Minimum Workload Requirement
The minimum workload requirement for faculty members paid 100% from Faculty Salaries is 9 teaching credits, counting classroom and equivalent teaching credits. The workload requirement is proportionately less for less than full-time appointments. For Graduate Assistant’s Teaching, the minimum workload standard is set by the academic unit reporting the workload.
APPENDIX D

Itemized List of Capital Equipment Purchases During the Past Five Years
## Itemized List of Capital Equipment Purchases During the Past Five Years

Capital Equipment Purchased by Participating Units in Past 5 Years

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<th>Dept</th>
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1 "Equipment" has the meaning established in the Texas Administrative Code §252.7(3) as items and components whose cost are over $5,000 and have a useful life of at least one year.
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<tr>
<th>Dept</th>
<th>Description</th>
<th>Acq Date</th>
<th>Total Cost</th>
<th>Serial #</th>
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**Teaching Materials:**

- PSYC OACS-30 SHUTTLE BOX SLAVE $5,415.48
- PSYC VIBRATOME ULTRAPRO LITE CRYOSTAT $16,311.00
- PSYC GAS ANESTHESIA VALUE PACKAGE FOR TH $5,233.00
- PSYC PATHFINDER MAZE SYSTEM OPEN & CLOSE $6,645.00
- PSYC VIDEOMEX-ONE COMPLETE SYSTEM PLUS W $20,138.75
- PSYC MODEL 942 DUAL SMALL-ANIMAL STEREOT $11,425.00
- PSYC LEICA CM3050S CRYOSTAT, 2000 MODEL, $19,685.00
- PSYC BIOPAC MP150 SYSTEM FOR WINDOWS $5,295.00
- PSYC MP-150 DATA ACQUISITION SYSTEM, AS $5,303.17
- PSYC 63 LITER BENCHTOP AUTOCLAVE WITH: F $6,895.00
- PSYC EYELINK COTR UNIT, CAMARA, HOST PC, $25,602.50
- PSYC DUAL SMALL ANIMAL STEREOtaxic INSTR $11,570.00
- PSYC STIMULATOR, SOTERIX 1X1 TES $8,295.73
- PSYC NEW BRUNSWICK INNOVA U -80 FREEZER $9,500.14
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APPENDIX E

Librarian’s Statement
Of
Adequate Resources
Dear Dr. Lench:

The Texas A&M University Libraries can readily support the proposed new BS program in Neuroscience. This program will not require additional library resources, because the library has steadily acquired materials in Neuroscience and related fields as part of its aggressive growth campaign.

The Libraries maintain subscriptions to top tier journals in the field along with access to key indices and databases like Neurosciences Abstracts, BIOSIS, Web of Science, Scopus, MEDLINE, PubMed, PsycINFO, PsycARTICLES, PsycCRITIQUES, Psychology and Behavioral Sciences Collection, Academic Search Ultimate, and Animal Behavior Abstracts.


Hundreds of print and e-books, media items and streaming media are added to the collection every year through individual purchases or included in various e-book packages and streaming services. PsycBOOKS, EBSCO eBook Collection and Hathitrust Digital Library are available along with streaming video and sound resources such as PsycTHERAPY, Counseling and Therapy in Video, SAGE Video, Kanopy Streaming Video, and Anatomy.tv. Additionally, over 30,000 titles in various video/file formats in the Media and Reserves unit can be studied either in the library or checked out.

The library has a collaborative approach to purchasing key resources in a field of study by encouraging and supporting faculty and student recommendations for new resources. Recommendations for databases, scholarly journals, and monographs can be made through the assigned library subject librarian or the library’s online “Suggest a Purchase” form. The annual library materials expenditures are over $14.5 million for journal and database subscriptions and over $2 million for print and e-book purchases. Overall, the University Libraries’ collection includes 1,712...
databases, approximately 121,838 unique serial titles, and over 5.6 million volumes, which will adequately support the proposed BS program in Neuroscience.

Texas A&M University Libraries is a member of the Association of Research Libraries (ARL). This distinct membership is based on TAMU Libraries distinct collections, commitment to servicing the scholarly community, and leadership. In addition, TAMU Libraries currently holds membership in the Greater Western Alliance (GWLA), which allows our campus users access to the holdings of 37 other research libraries located across the United States. Another important consortium membership includes the Center for Research Libraries (CRL) whose mission is to foster and advance scholarly inquiry by granting members access to its five million newspapers, journals, dissertations, and digital resources.

To summarize, the Texas A&M University Libraries is committed to supporting a new BS program in Neuroscience.

Sincerely yours,

David Carlson
Dean of University Libraries
APPENDIX F

Articulation Agreements
With
Partner Institutions
Not Applicable
APPENDIX G

Curricula Vitae
For
Core Faculty
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.)

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<td>2006</td>
<td>Social Science</td>
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<tr>
<td>Villanova University</td>
<td>M.S.</td>
<td>2009</td>
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<td>Ph.D.</td>
<td>2014</td>
<td>Psychological &amp; Brain Sciences</td>
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<tr>
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<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**

R01-DA013165 (PI: Yantis/Courtney) 3/1/2011-6/30/2016 Post Doc 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
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: JOE A. AROSH

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

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>May 1995</td>
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<tr>
<td>University of Montreal, Canada</td>
<td>Post-Doc</td>
<td>November 2004</td>
<td>Reproductive Molecular Endocrinology</td>
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A. PERSONAL STATEMENT:
My laboratory-based research programs are focused to understand molecular, cellular and epigenetic aspects of the pathogenies of endometriosis. The objective of this application are to determine global pain-gene signatures in lumbar DRG, spinal cord, and pain-related brain regions by RNA-Seq, establish their association with pain perception and growth of peritoneal endometriotic lesions and determine the therapeutic effects of pharmacological inhibition of EP2, EP4, and TLR4 on pain perception, tissue-specific pain gene-expression signatures, and growth of peritoneal endometriotic lesions. We have developed and characterized novel human immortalized endometriotic epithelial and stromal cells and transgenic endometriotic cells, and xenograft of fluorescence–labeled epithelial and stromal cells in immunocompromised mice. These models can be used as valuable tool to study molecular pathogenesis of endometriosis, experimental therapeutics, and functional genomics. We have developed strong collaboration with clinical investigators (OBGYN) to study the pathogenesis of endometriosis comprehensively in order to decrease the disease burden and pain and preserve the fertility in child-bearing age women.

Note: Corresponding Author (*)

B. POSITIONS AND HONORS:
CURRENT POSITION:
12/2004-08/2011: Assistant Professor, Department of Integrative Biosciences, Texas A&M University.
09/2011-08/2016: Associate Professor (tenured), Department of Integrative Biosciences, Texas A&M University.
09/2016-Present: Professor, Department of Integrative Biosciences, Texas A&M University.


FACULTIES AND CENTERS: (1) Faculty of Veterinary Integrative Biosciences, Texas A&M University, 2004-present; (2) Faculty of Reproductive Biology, Texas A&M University, 2004-present.


EDITORIAL BOARDS
1. Editorial Board Member, Faculty of 1000 Medicine, Global Leaders & Expert Knowledge. Diabetes & Endocrinology-Reproductive Endocrinology, Feb 2010- present. http://f1000medicine.com/
2. Editorial Board Member, Journal of Veterinary Science and Technology, May 2010-Present.
3. Associate Editor, Bioinfo Journals, Veterinary Science Research, June 2010-Present.

AWARDS AND HONORS
1. CIHR Merit Award, Canadian Institute of Health Research, Govt. of Canada, 2002 & 2003.
2. Dean’s Recognition of Excellence for Doctoral Research (Honor), Faculty of Medicine, Laval University, Quebec, Canada, 2004.
5. Outstanding Doctoral Research Fellow Merit Award, Physiology-Endocrinology Program, Faculty of Medicine, Laval University, Quebec, Canada, 2004.
7. USDA-NRI Merit Award- Society for the Study of Reproduction 2003, USDA-NRI.
10. Pfizer Award for Veterinary Research Excellence for Outstanding Achievement and Dedication in the field of Veterinary Medicine, 2012.

GRANT REVIEW COMMITTEES
2. United States Department of Agriculture (USDA) and Binational Agricultural Research and Development (BARD) USDA-BARD review panel, 2008, 2011, and 2013.
4. External Reviewer, Study Section, Natural Sciences and Engineering Research Council (NSERC 2009-2012) of Canada, and reviewed research grant proposals.

C. CONTRIBUTION TO SCIENCE


(II) Maternal-Fetal interactions at Establishment of Pregnancy in Ruminants. Understand the physiological roles of PGF2α and PGE2 in luteolysis and the maternal-fetal dialogue required for the establishment of pregnancy in ruminants. Understanding of the molecular and cellular mechanisms of these processes is necessary to ameliorate infertility/subfertility in ruminants.


(III) Chromium (CrVI) and Reproductive Functions: Drinking water contamination with CrVI in the United States is a growing problem due to increased usage of CrVI and improper disposal of chromium waste into the environment. Significant contamination with CrVI has been found in the drinking water sources of more than 30 cities in the United States. While the adverse effects of CrVI have been well studied in lung cancer, effects of Cr on reproductive health in women have received less attention. We (in collaboration with Dr. Sakhila K. Banu, Texas A&M University) determine the effects of CrVI ovarian development and functions.


3. Sivakumar KK, Stanley JA, Arosh JA*, Pepling ME, Burghardt RC, Banu SK. Prenatal exposure to chromium induces early reproductive senescence by increasing germ cell apoptosis and advancing germ cell cyst breakdown in the F1 offspring. Dev Biol. 2014 Apr 1; 388 (1):22-34. PMID: 24530425; PubMed Central PMCID: PMC3991725


(IV) Dioxin and Reproductive Functions: Dioxin exposure affects reproductive function in human and animals, and induces endometriosis.

The goal of this project is to investigate the role of the environmental toxicant “dioxin” as a trigger for the development of endometriosis. We (in collaboration with Dr. Kevin G. Osteen and Dr. Kaylon Bruner-Tran, Vanderbilt University) utilize both in vitro and in vivo experimental model systems to test the therapeutic value of resveratrol and prostaglandin signaling inhibitors to act as anti-inflammatory agents capable of protecting progesterone sensitivity in human endometrial cells following acute and chronic exposure to dioxin.

1. Bruner-Tran KL, Ding T, Yeoman KB, Archibong A, Arosh JA, Osteen KG. Developmental exposure of mice to dioxin promotes transgenerational testicular inflammation and an increased risk of preterm birth in


(V) Superovulation and Embryo Transfer. My early research involved with estrous synchronization and embryo transfer in ruminants.


Complete List of Published Work in My Bibliography:
Note: Articles published in Indian Journals are not acceptable through PubMed at present.

D. RESEARCH SUPPORT:

ONGOING RESEARCH SUPPORT
1. NIH/NICHD (1R01HD079625)
   Project title: Role of miR15a and miR34c in PGE2 Signaling in the Pathogenesis of Endometriosis
   Role: Principal Investigator
   Duration: 05/01/2015 - 04/30/2020
   The goal of this project is to study molecular, cellular, and epigenetic interactions between aspects of PGE2 signaling and proapoptotic miRNA in survival and growth of endometriosis.

   The Role of Intraluteal Prostaglandins in Luteolysis and Luteal Protection in Sheep
   Role: Principal Investigator
   Duration: 09/01/13-08/31/18
   The goal of this project is to understand PGF2α and PGE2 biosynthesis and signaling in luteal cells.

3.NIH/NIEHS (2 R01 ES014942-06)
   Project title: Dioxin Exposure and the Invasive Pathogenesis of Endometriosis
   Role: Co-Investigator (PI- Kevin Osteen, Vanderbilt University, Subcontract to Texas A&M)
   Duration: 9/1/2012 to 8/31/2018
   The goal of this project is to investigate the role of the environmental toxicant ‘dioxin’ as a trigger for the development of endometriosis. The project utilizes both in vitro and in vivo experimental model systems to test the therapeutic value of resveratrol and prostaglandin signaling inhibitors to act as anti-inflammatory agents capable of protecting progesterone sensitivity in human endometrial cells following acute and chronic exposure to dioxin.

4. NIH/NIEHS-1R01ES025234-01
   Project title: Mechanism of Prenatal Chromium-VI Exposure and Germ Cell Apoptosis in the Ovary.
   Role: Co-Investigator (PI: Dr. Sakhila K. Banu)
   Duration: 02/01/2016 to 01/31/2021
   The objective of this project is to understand molecular and cellular mechanisms through which gestational exposure to CrVI accelerates oocyte nest break down, apoptosis of germ cells and somatic cells, impairs oocyte and early embryonic development, and fetal growth through altering epigenetic pathways in F1 progeny.

COMPLETED RESEARCH SUPPORT
1. Source: NIH/NICHD (1R21HD065138)
   Project title: Molecular Basis of Treating Endometriosis by Prostaglandin E2 Receptor Inhibitors
   Role: Principal Investigator
   Duration: 09/30/2011 to 08/31/2014
The goal of this project is to study molecular and cellular aspects of PGE2 signaling in apoptosis, immunomodulation, and estrogen biosynthesis in the pathogenesis of endometriosis.

2. Source: NIH/NICHD (1R21HD066248)
Project title: Prostaglandin E2 Signaling in Growth and Pains of Endometriosis
Role: Principal Investigator
Duration: 10/01/2011 to 09/30/2014
The goal of this project is to study molecular and cellular aspects of PGE2 signaling in pains of Endometriosis

3. Source: NIH/NIEHS (1R21ES020561)
Project title: Chromium VI-induced toxicity on female reproductive function.
Role: Co-Investigator (PI: SK Banu)
Duration: 08/16/2011 to 07/31/2014
The goal of this project is to study molecular and cellular aspects of chromium (VI)-induced follicular atresia and granulosa cell apoptosis.

NAME: Banu, Sakhila

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

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 Montreal, Canada</td>
<td>Postdoctoral</td>
<td>02/2004</td>
<td>Endocrine Oncology</td>
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A. Personal Statement

My long-term goals are two-fold: 1) to understand the molecular mechanism of prenatal CrVI exposure on placental and fetal development, ovarian and uterine function, and pregnancy outcome, and; 2) to understand the protective effects of various natural and synthetic antioxidants (such as edaravone, glutathione, vitamin C and resveratrol) against the deleterious effects of heavy-metals, CrVI in particular. Current research in my lab is focused on the study of reproductive and developmental toxicity of CrVI. Drinking water contamination with CrVI in the United States is a growing problem due to increased usage of CrVI and improper disposal of Cr waste into the environment. Significant contamination with CrVI has been found in the drinking water sources of all the states in the U.S. Effects of Cr on reproductive health in women and development in children have received less attention. Epidemiological data document that women exposed to Cr in environmental or occupational settings suffer from infertility, gynecological problems, congenital malformation of fetuses, neonatal mortality, and premature abortions with increased levels of Cr in their blood, urine and placenta. Cr can bind directly to DNA and nuclear proteins, cause DNA strand breaks and mutations, alter the balance between reactive oxygen...
species (ROS) and antioxidants, and activate several cell signaling pathways. Therefore, my current research objective is to determine molecular pathways and identify target genes/proteins by which Cr alters prenatal development and organogenesis of female reproductive system in the offspring. The goal of the current research project is to understand how gestational exposure to CrVI affects placental development and function and the subsequent health of the F1 progeny by modulating oxidative stress, inflammatory and epigenetic interactive pathways. I have assembled a multidisciplinary research team to dissect the key pathways that potentially cause placental dysfunction due to gestational CrVI exposure. The outcome of this project is expected to fill the gap in knowledge between gestational exposure to CrVI and female reproductive function during adult life.

**B. Positions and Honors**

**Positions and Employment**

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<td>2001-2003</td>
<td>Post-Doctoral Fellow, Department of Obstetrics &amp; Gynecology, Laval University, Canada</td>
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<tr>
<td>2003-2004</td>
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<td>Research Assistant Professor, Department of Veterinary Integrative Biosciences, Texas A&amp;M University, College Station, TX</td>
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<td>2008-2012</td>
<td>Clinical Assistant Professor, Dept. of Integrative Biosciences, College of Veterinary Medicine &amp; Biomedical Sciences, Texas A&amp;M University, College Station, TX</td>
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<tr>
<td>2009-2012</td>
<td>Honorary Visiting Professor, University of Madras, India</td>
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<td>2012-2017</td>
<td>Assistant Professor, Department of Veterinary Integrative Biosciences, Texas A&amp;M University, College Station, TX</td>
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**Other Recent Experience and Professional Memberships**

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<tr>
<td>1994-2017</td>
<td>Life-time member, Society for Reproductive Biology and Comparative Endocrinology</td>
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<td>2001-2005</td>
<td>Member, Society for Study of Reproduction, Ad Hoc reviewer for more than 40 journals, including: Archives of Environmental and Occupational Health; American Journal of Obstetrics &amp; Gynecology; Bimetal; BBA-Mol Cell Research; Biology of Reproduction; Domestic Animal Endocrinology; Ecotoxicology and Environmental Safety; Environmental Toxicology &amp; Pharmacology; Fertility &amp; Sterility; Indian Journal of Animal Reproduction; Indian Journal of Animal Sciences; International Journal of Cancer; Journal of Applied Toxicology; Journal of Biological Chemistry; Journal of Endocrinology &amp; Reproduction; Journal of Food and Chemical Toxicology; Molecular Reproduction and Development; Neurotoxicology; PLoS One; Regulatory Toxicology and Pharmacology; Reproductive Toxicology; Science, Toxicological and Environmental Chemistry; Toxicology; Applied Pharmacology; and Toxicology Mechanisms and Methods</td>
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<td>External examiner, PhD theses, University of Madras, Ad Hoc reviewer, NIH Review Committee, Systemic Injury by Environmental Exposure study section</td>
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<tr>
<td>2013-2017</td>
<td>External examiner, PhD theses, Bharathidasan University, Ad Hoc reviewer, NIH Review Committee, Cellular, Molecular and Integrative Reproduction study section</td>
</tr>
<tr>
<td>2011-2017</td>
<td>Reviewer, Republic of Italy, Ministry of Health, Department of Public Health and Innovation, Member, Endocrine Society, Society of Toxicology, and Society for the Study of Reproduction</td>
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**Honors**

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<tr>
<td>1993</td>
<td>Senior Research Fellowship, Lady Tata Memorial Trust &amp; Hospitals, India</td>
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<td>1997</td>
<td>Senior Research Fellowship, Council of Scientific and Industrial Research (CSIR), Government of India</td>
</tr>
<tr>
<td>2002</td>
<td>Canadian Institute of Health Research (CIHR) Merit Award, Government of Canada</td>
</tr>
<tr>
<td>2002</td>
<td>Trainee Award Finalist, Society for the Study of Reproduction (SSR)</td>
</tr>
<tr>
<td>2002</td>
<td>Trainee Merit Award, USDA-NRI</td>
</tr>
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<td>2003</td>
<td>Larry Ewing Memorial Award, SSR</td>
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<td>2003</td>
<td>CSIR Merit Award, Government of Canada</td>
</tr>
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<td>2003</td>
<td>Trainee Travel Award, SSR</td>
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C. Contributions to Science

1. **Elucidating the mechanisms of how exposure to CrVI during pregnancy disrupts placental development:** Epidemiological data suggests that exposure of pregnant women to CrVI through occupational settings increases Cr levels in their blood, urine, and umbilical cord, resulting in pre-term labor. Our data suggests that CrVI increases apoptosis of the trophoblasts and down regulates the cell survival proteins. CrVI also increases oxidative stress and decreases antioxidants levels in the placenta and AOX expressions in the trophoblast cells.
   
   

2. **Determining that CrVI exposure causes premature ovarian failure and developing a model of embryonic/fetal whole ovarian culture:** For the first time my laboratory showed that prenatal exposure to CrVI increases germ cell death and lead to premature ovarian failure (POF). Women with balanced translocations between the long arm of the X chromosome and an autosome frequently suffer POF. The translocation disrupts five genes, including Xpnpep-2, encoded in the human X chromosome in POF women. We were the first to report that prenatal exposure to CrVI alters the expression pattern of protein encoded by Xpnpep2. The translocation disrupts Xpnpep2 during fetal and postnatal ovarian development, resulting in accumulation of collagen in the ovarian follicular compartments leading to oocyte death and follicle degeneration. Thus, we reported that prenatal exposure to CrVI causes POF by altering Xpnpep2. We also developed an embryonic/fetal whole ovarian culture model to evaluate the effects of EDCs/drugs on fetal ovarian development. This is a novel and innovative tool that can be used for high throughput analysis of other EDCs and heavy metals across the mammalian species.
   
   
   

3. **Demonstrating that postnatal exposure to CrVI through mother's milk increases follicle atresia and disrupts steroidogenesis:** Drinking water contamination with CrVI in the United States is a growing concern due to increased usage of CrVI and improper disposal of chromium waste into the environment. According to September 2016 EWG data, CrVI contamination has been detected in almost every state in the U.S. Epidemiological data document that women exposed to Cr in environmental or occupational settings suffer from infertility and various gynecological problems, congenital malformation of fetuses, neonatal mortality, and premature abortions. Therefore, my lab is focused to determine molecular pathways and identify target genes/proteins by which heavy metals alter prenatal development and organogenesis of female reproductive system in the offspring, as well as postnatal ovarian development and functions of the offspring transgenerationally. To the best of my knowledge, my lab is the only lab in the nation that is currently working on CrVI toxicity and the ovary/female fertility. For the first time my laboratory reported that lactational exposure of Cr impairs follicular development in F1 offspring of exposed rats. CrVI delayed follicle development by decreasing granulosa cell proliferation and by altering cell cycle regulatory proteins, accelerating follicular atresia through ROS, p53 and ERK1/2 pathways, and by altering sub-cellular localization of p53, ERK and mitochondrial pro-apoptotic proteins. In addition, we identified that increased oxidative stress and decreased antioxidant levels in the blood and the ovary as the key mechanisms behind of CrVI-induced reproductive toxicity in F1 offspring, exposed to CrVI through the mothers.


4. Identifying antioxidants and nutrioxidants that protect against CrVI-induced reproductive toxicity: Apart from understanding the mechanism of reproductive toxicity of CrVI, we also for the first time identified intervention strategies to mitigate or inhibit CrVI toxicity and protect the ovary. In this line, we have identified vitamin C, edaravone, and resveratrol as potential candidates to protect the ovary from heavy metal toxicity.


5. Determining how sex steroids regulate TSH-induced thyroid growth from birth to adult age: There is a gender bias in the incidence of thyroid diseases and cancers, which predominates in females. In order to understand the underlying mechanism, in collaboration with my PhD mentor (Dr. Michael M. Aruldhas), I studied the changes in sex steroid profiles, estrogen and androgen receptors, thyroid gland development and corresponding changes in thyroid stimulating hormone (TSH) and TSH-receptors in male and female rats from age postnatal day 1 through 150, both in vivo and in vitro models. Key finding is that sex steroids have a differential and temporal mitogenic role in regulating normal thyroid growth in a gender-specific manner, by regulating the levels of TSH and TSH receptors.


Complete List of Published Work in MyBibliography

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

Ongoing Research Support
1R01ES025234-01  Banu (PI)  02/01/16 - 01/31/21
NIH/NIEHS
Mechanism of Prenatal Chromium-VI Exposure and Germ Cell Apoptosis in the Ovary
The objective of this project is to understand molecular and cellular mechanisms through which gestational exposure to CrVI accelerates oocyte nest break down, apoptosis of germ cells and somatic cells, impairs oocyte and early embryonic development, and fetal growth through altering epigenetic pathways in F1 progeny.
Role: Principle Investigator
The objective is to determine the factors regulating intraluteal PGF2a and PGE2 biosynthesis and signaling during luteolysis and establishment of pregnancy using sheep as a ruminant model.

Role: Co-Investigator

Role of miR15a and miR34c in PGE2 Signaling in the Pathogenesis of Endometriosis

The goal of this project is to study molecular, cellular, and epigenetic interactions between aspects of PGE2 signaling and proapoptotic miRNA in survival and growth of endometriosis.

Role: Co-Investigator
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>
</tr>
<tr>
<td>University of Michigan</td>
<td>PHD</td>
<td>08/2012</td>
<td>Psychology</td>
</tr>
<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 Clinical
2016 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
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: 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>
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<tr>
<td>California State University, San Bernardino, CA.</td>
<td>M.A.</td>
<td>05/1995</td>
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<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, PLC, FosB; 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.


Complete list of published work in MyBibliography:

**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.
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: Dulin, Jennifer Natalie

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

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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<td>B.S.</td>
<td>05/2005</td>
<td>Biochemistry</td>
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<tr>
<td>University of Texas Health Science Center, Houston, TX</td>
<td>Ph.D.</td>
<td>05/2012</td>
<td>Neuroscience</td>
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<tr>
<td>University of California, San Diego</td>
<td>Postdoctoral</td>
<td>09/2017</td>
<td>Neuroscience</td>
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Positions and Honors

*Positions and Employment*

2017-2017 Assistant Professor, Dept. of Biology, Texas A&M University
2012-2017 Postdoctoral Fellow, University of California, San Diego
2008-2012 Graduate Student, University of Texas Health Science Center – Houston
2007-2008 Research Assistant II, MD Anderson Cancer Center
2005-2006 Research Assistant, UT Southwestern Medical Center
2002-2005 Research Assistant, Texas A&M University

*Other Experience and Professional Membership*

2018-2017 Texas Brain & Spine Institute
2017-2017 Texas A&M Institute for Neuroscience (TAMIN)
2017-2017 Mission Connect (TIRR Foundation)
2015-2015 Coordinator & speaker, Mini-symposium “Transcriptomics Approaches to Neural Repair”, Society for Neuroscience 2015, Chicago, IL
2012-2015 Mission Connect, student member
2010-2012 American Association for the Advancement of Science, student member
2009 6th NINDS Spinal Cord Injury Research Training Program, Ohio State University
2008-2010 Society for Neuroscience
2008-2012 National Neurotrauma Society
2008-2010 Women in Neurotrauma Research

*Honors and Awards*

2018 Invited speaker, TIRR Foundation Board meeting, Houston, TX
2018 Invited speaker, Department of Neuroscience, Thomas Jefferson University, Philadelphia
2018 Southeastern Conference (SEC) Faculty Travel Grant
2018 ADVANCE Science Scholar, Texas A&M University
2017 Mission Connect Research Grant
2017 TIRR Foundation Scholar, Texas A&M University
2017 Veterans Administration Career Development Award CDA-2 (declined)
2017 Invited speaker, Interdisciplinary Center for Neurosciences (IZN) Lecture Series, Heidelberg University, Germany
2014 Invited speaker, HeadNorth Neural Injury and Regeneration Symposium, UCSD

*Peer-Reviewed Journal Articles*


Review Articles


Book Chapter

Other

Research Support
Current Support:
Neural Stem Cells for Mitigating Pain after Spinal Cord Injury
Craig H. Neilson Foundation
07/31/18 – 07/30/20

PI (Dulin, Jennifer)
Major Goals: To test the hypothesis that spinal cord neural stem cells grafted into sites of spinal cord injury will restore inhibitory inputs onto dorsal horn pain-processing neurons at and below the site of injury, thereby attenuating hyperactive nociceptive signaling.
Total Direct Costs: $271,941

Connectivity Mapping of Neural Stem Cells for Restoring Locomotor Function
Paralyzed Veterans of America Research Foundation
04/01/18 – 03/31/20

PI (Dulin, Jennifer)
Major Goals: To address the gap in our current understanding of the mechanisms by which spinal cord neural stem cell grafts can promote motor functional recovery following spinal cord injury.
Total Direct Costs: $134,721

Chemogenetic Silencing of Nociceptors to Enhance Motor and Sensory Outcomes following Spinal Cord Injury
TIRR Foundation
12/01/17 – 12/31/19

PI (Dulin, Jennifer)
Major Goals: To test the hypothesis that silencing nociceptor activity during the acute phase of spinal cord injury will (1) attenuate the development of enhanced mechanical reactivity and pain-associated behaviors, and (2) enhance recovery of motor function.
Total Direct Costs: $50,000

Pending Support:
Generating a Neuron Interaction Network for Neural Progenitor Cell Grafts in the Injured Spinal Cord
NIH Director’s New Innovator Award Program (DP2) 08/15/19 – 06/30/24
PI (Dulin, Jennifer)
Pending merit review 03/2019
Major Goals: To generate an unbiased, cell type-specific connectivity map and functional interaction network for neural progenitor cell transplants within the injured adult spinal cord.
Total Direct Costs: $1,392,052
Role: PI

Peptide Strategies to Increase Axon Regeneration by Targeting Stress Granule Function
NINDS IGNITE R61/R33 07/01/19 – 06/30/22
Pending merit review 03/2019
Major goals: To demonstrate in vivo efficacy of a novel stress granule-targeting peptide on peripheral and central axon regeneration.
Total Direct Costs: $200,528
Role: Co-Investigator (PI: Twiss, Jeffery)

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.
**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>SHOSHANA EITAN</th>
<th>POSITION TITLE</th>
<th>ASSOCIATE PROFESSOR</th>
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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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<td>Open University, Tel-Aviv, Israel</td>
<td>BA</td>
<td>1987-90</td>
<td>Biology</td>
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<td>Weizmann Institute of Science, Rehovot, Israel</td>
<td>MsC</td>
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<td>Neurobiology</td>
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<tr>
<td>Weizmann Institute of Science, Rehovot, Israel</td>
<td>PhD</td>
<td>1992–97</td>
<td>Neurobiology</td>
</tr>
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<td>Norman Cousin Center of Psychoneuroimmunology, UCLA, Los Angeles, CA</td>
<td>Trainee</td>
<td>1997-00</td>
<td>Neurobiology</td>
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<tr>
<td>Neuropsychiatric Institute, UCLA, Los Angeles, CA</td>
<td>Post-Doctorate</td>
<td>2000-02</td>
<td>Neuropharmacology</td>
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<tr>
<td>Neuropsychiatric Institute, UCLA, Los Angeles, CA</td>
<td>Assistant Researcher</td>
<td>2002-05</td>
<td>Neuropharmacology</td>
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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)
RESEARCH AND PROFESSIONAL EXPERIENCE (CONTINUED).

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


outgrowth”, *PLoS ONE* 2011, 6(3):e18439


**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)  
9/08-8/08  
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
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 Microbiology. 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* 2014;10.7554/eLife.01730


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>
<th>Completion Date MM/YYYY</th>
<th>FIELD OF STUDY</th>
</tr>
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<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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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 (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 unravels the functional mechanism that underlies the learning (Joyner 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 Metaplasticity 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).


A full list of my publications can be found at:
D. Research Support

Completed

R01 NS069537 (PI: Ferguson; co-I: 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; co-I: 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, co-I: 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; co-I: 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; co-I: 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
Lawrence R. Griffing
Associate Professor, Biology, TAMU

(a) Professional Preparation

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<td>University of Utah</td>
<td>Biology</td>
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<tr>
<td>Stanford University</td>
<td>Biology</td>
<td>Ph.D.</td>
<td>1981</td>
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<tr>
<td>Oregon State University</td>
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<td>1982</td>
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<td>University of Saskatchewan</td>
<td></td>
<td>Post-doc</td>
<td>1984</td>
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<tr>
<td>Plant Biotechnology Institute, National Research Council of Canada</td>
<td></td>
<td>Post-doc</td>
<td>1986</td>
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(b) Appointments

2001-2006 Associate Director (Biology), ITS Center for Teaching and Learning, Texas A&M, College Station, TX
1994-1995 Program Director, Cell Organization, BIO, National Science Foundation, Arlington, VA
1991-present Associate Professor, Department of Biology, Texas A&M, College Station, TX
1986-1991 Assistant Professor, Department of Biology, Texas A&M, College Station, TX

(c) Selected Peer Reviewed Publications and Monographs.


Griffing LR (2011) Laser stimulation of the chloroplast/endoplasmic reticulum nexus in tobacco transiently produces protein aggregates (boluses) within the endoplasmic reticulum and stimulates local ER remodeling. Mol Plant 4:886-95 [Full text](http://www.plantphys.net/content/4/6/886)

d) Current graduate students:

Krishna Kumar: Pathway of sterol uptake in plant cells.
Sara Maynard: Effects of SERCA inhibitors on the calcium wave triggered by ER-chloroplast photostimulation.
e) Current semester (Fall 2017) undergraduate researchers
   Connelly, Meghan: Effect of lanthanum and gadolinium on calcium wave triggered by ER-chloroplast photostimulation.
   Mulroy, Ian, and Puckett, Nathaniel: Virtual reality representations of internal structures of the plant endoplasmic reticulum
   Prabhakar, Sarah, and Singh, Sujay; Quantitative action spectra of blue light photoresponse at the ER-chloroplast junction.
   Barnett, Abby: Effect of external sterols on plant growth and development.

f) Recent invited oral presentations
   Society for Experimental Biology, Goteborg, Sweden, July 5-8, 2017. The calcium wave produced by photostimulation of the ER-Chloroplast nexus.
   Society for Experimental Biology: Brighton, UK, July 4-8, 2016: The nature of the association of the ER with the cytoskeleton during plasmolysis.

g) Recent poster and oral presentations:

h) Current teaching:
   BIOL 112 Introductory Biology (Spring 2017)
   BIOL 430 Biological Imaging (4 credits, Spring 2017 and 2018)
   BIOL 423 Advanced Lab in Cell Biology (2 credits, Fall 2018).
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.)

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<td>Indiana University</td>
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<tr>
<td>Brandeis University</td>
<td>Postdoctoral</td>
<td>08/1991</td>
<td>Neurogenetics</td>
</tr>
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</table>

A. Personal Statement

I have >25 years experience using Drosophila as a model system for molecular clocks research. As a postdoctoral 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**

<table>
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<tr>
<th>Years</th>
<th>Position and Title</th>
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<tr>
<td>1991-1995</td>
<td>Assistant Professor, Department of Biology and the Center for Advanced Invertebrate Molecular Sciences, Texas A&amp;M University.</td>
</tr>
<tr>
<td>1995-2000</td>
<td>Associate Professor, Department of Biology and Biochemistry, University of Houston.</td>
</tr>
<tr>
<td>2000-2005</td>
<td>Professor, Department of Biology and Biochemistry, University of Houston.</td>
</tr>
<tr>
<td>2004-2005</td>
<td>John and Rebecca Moores Professor, Department of Biology and Biochemistry, University of Houston.</td>
</tr>
<tr>
<td>1996-2005</td>
<td>Adjunct Professor, Department of Biology, Texas A&amp;M University.</td>
</tr>
<tr>
<td>2005-2008</td>
<td>John W. Lyons ’59 Chair and Professor, Department of Biology, Texas A&amp;M University.</td>
</tr>
<tr>
<td>2005-2008</td>
<td>Adjunct Professor, Department of Biology and Biochemistry, University of Houston.</td>
</tr>
<tr>
<td>2006-2009</td>
<td>Member, Faculty of Genetics, Texas A&amp;M University.</td>
</tr>
<tr>
<td>2006-2009</td>
<td>Member, Texas A&amp;M Institute of Neuroscience, Texas A&amp;M University.</td>
</tr>
<tr>
<td>2008-</td>
<td>John W. Lyons Jr. ’59 Chair and Distinguished Professor, Department of Biology, Texas A&amp;M University.</td>
</tr>
</tbody>
</table>

**Other Experience and Professional Memberships**
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 1 \( \Sigma^\text{TM} \) (PDP1\( \Sigma^\text{TM} \)) 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
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

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/Rand 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
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.


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 vasulogenesis (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 vasulogenesis, 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 $\rightarrow$ VEGFR2.

**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 posttranslational 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.

Texas A&M Institute for Neuroscience
Discovering a new bioactive peptide, peptide Lv, and its role in neural function and angiogenesis.

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).

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-onset 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.

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-onset 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.

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**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.

- **R01 EY017452-01A1** 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

Revised 6/08
BIOGRAPHICAL SKETCH

Name: Jianrong Li, Ph.D.  Position/Title: Associate Professor

<table>
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<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE</th>
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<td>B.S.</td>
<td>1988</td>
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<td>1991</td>
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<td>University of Hawaii</td>
<td>Ph.D.</td>
<td>1997</td>
<td>Biochemistry</td>
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<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>2000-2005</td>
<td></td>
<td>Neurobiology</td>
</tr>
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</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

Revised 6/08
Publications 2012-2017:

Original Research Reports:


Invited reviews and book chapters:


**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. 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>
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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>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></td>
<td>2007-2009</td>
<td>Chemistry</td>
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</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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<td>McGill University, Montreal, QC</td>
<td>AB</td>
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<td>MFA</td>
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<td>MA</td>
<td>08/2009</td>
<td>Clinical Psychology</td>
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<tr>
<td>Stony Brook University, Stony Brook, NY</td>
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<td>08/2013</td>
<td>Clinical Psychology</td>
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<tr>
<td>University of Illinois at Chicago, Chicago, IL</td>
<td>Other training</td>
<td>06/2013</td>
<td>Predoctoral Clinical Psychology Internship</td>
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<tr>
<td>University of Illinois at Chicago, Chicago, IL</td>
<td>NIH training grant</td>
<td>06/2015</td>
<td>T32 Postdoctoral Research Fellow</td>
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</table>

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 increasing 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 - Assistant Professor, Texas A&M University, College Station, TX

Other Experience and Professional Memberships
2007 - Member, Society for Psychophysiological Research
2008 - 2012 Member, American Psychological Association
2013 - Member, Society for a Science of Clinical Psychology
2013 - Member, Anxiety and Depression Association of America
2017 - Member, Society for Affective Science

Honors
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 - Assistant Professor, Texas A&M University, College Station, TX

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-
elicted 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.

a. MacNamara A, Proudfit GH. Cognitive load and emotional processing in generalized anxiety disorder:
  PMID: 24933276; PubMed Central PMCID: PMC4122583.

b. MacNamara A, Schmidt J, Zelinsky GJ, Hajcak G. Electrocortical and ocular indices of attention to
  fearful and neutral faces presented under high and low working memory load. Biol Psychol. 2012

c. MacNamara A, Ferri J, Hajcak G. Working memory load reduces the late positive potential and this
  PMID: 21556695.

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.

a. MacNamara A, Post D, Kennedy AE, Rabinak CA, Phan KL. Electrocortical processing of social signals
  PMID: 24025760.

b. MacNamara A, Hajcak G. Distinct electrocortical and behavioral evidence for increased attention to

c. MacNamara A, Hajcak G. Anxiety and spatial attention moderate the electrocortical response to

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 (vIPFC) 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

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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<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
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<th>FIELD OF STUDY</th>
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<td>University of Illinois – Urbana-Champaign</td>
<td>BS</td>
<td>1989</td>
<td>Psychology</td>
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<td>University of Southern California, Los Angeles, CA</td>
<td>MS</td>
<td>1991</td>
<td>Neurobiology</td>
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<td>University of Southern California, Los Angeles, CA</td>
<td>PhD</td>
<td>1993</td>
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<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
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.


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
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 amnesics 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 this 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

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<td>B.S.</td>
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<td>Psychology &amp; Neuroscience</td>
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<tr>
<td>Northwestern University, Evanston, IL</td>
<td>Ph.D.</td>
<td>06/2012</td>
<td>Psychology &amp; Neuroscience</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
Professional Memberships

American Pain Society

Vice Chair, Pain and Disparities SIG, 2016 – present
Secretary, Pain and Disparities SIG, 2012 – 2016
Board Member, Early-Career Advisory Group, 2014 – present
Consultant, Early-Career Forum Planning Committee – 2016

Association for Psychological Science

Cognitive Neuroscience Society

International Association for the Study of Pain

Social and Affective Neuroscience Society

Society for Personality and Social Psychology

Society for Neuroscience

Society for Social Neuroscience

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.


*denotes student mentee

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 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 lower 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/) and inclusion in the outreach focused arm of the Pain Research Forum (June 2016 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: CoInvestigator, Dissertation (2011-2013)
**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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<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 reaggregate 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.


Complete List of Published Work in MyBibliography:

http://www.ncbi.nlm.nih.gov/sites/myncbi/1hABQzv4cxSx/bibliography/48171577/public/?sort=date&direction=ascending

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

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<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
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<tr>
<td>Nazareth College of Rochester</td>
<td>B.S.</td>
<td>05/1982</td>
<td>Psychology major/Biology minor</td>
</tr>
<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>
</tr>
<tr>
<td>Texas A&amp;M University</td>
<td>Postdoc</td>
<td>05/1993</td>
<td>Clinical Psychology</td>
</tr>
<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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</tbody>
</table>

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. Though 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
2001 Associate Professor, Department of Psychology, Texas A&M University
2005 Professor, Department of Psychology, Texas A&M University
2003-08 Director, Recovery of Function Graduate Training Program in Neuroscience
2007- Texas Brain & Spine Institute – Basic Research Faculty

**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 dPAG, 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-
authors (*) were placed in postdocs and faculty positions. Notably, Tamara King (U New England, Assoc. Prof.) extended this early work to develop an associative learning measure of tonic pain (conditioned place preference task) with Drs. Frank Porreca and Howard Fields. Adam Ferguson (UCSF, Assist Prof.) investigates spinal plasticity and recovery of function using a spinal instrumental learning paradigm.


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, Theiler's virus induced demyelination. This project involves a collaboration with CJR Welsh, a neuroimmunologist with expertise in Thelier'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 surraspinal 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**  
**6/01/15-5/31/17**  
NIH/NIAAA  
Effect of Alcohol Withdrawal on Pain Sensitization  
National Research Service Award, Individual Predoctoral Fellowship  
Role: Sponsor for Dokyoung Sophia You

**One Health Grant**  
**5/31/15-6/1/18**  
Texas A&M University  
Mechanisms mediating pain sensitization following sexual assault  
Role: PI

**2 T32 OD011083-06**  
**7/18/2015-3/31/2020**  
NIH  
Institutional Training Grant for Comparative Biomedical Research Training for Veterinarians.  
Role: Training faculty
Completed Research Support:

R21 DA034285-01 (PI: Ditre; coI: Meagher) 8/01/12-03/31/16 NIH/NIDA
Effects of Smoking Abstinence on Pain Reactivity: A Human Experimental Model

R21 HD066248-01A1 (PI: Arosh; coI: Meagher) 10/01/11-07/31/15 NIH/NICHD
Prostaglandin E2 Signaling in Growth and Pain of Endometriosis

TBSI Seed Grant (PI: Meagher) 6/01/12-8/31/16 St. Joseph’s Hospital
Effects of psychogenic stress on pain after spine surgery: the moderating/mediating role of cytokines.

T32OD011083-05 (PI: Kier) 3/1/10-5/15/15 OD/NIH
Role: Training Faculty, “Comparative Biomedical Research Training for Veterinarians”

R01NS060822 (PI: Meagher) 12/1/07-1/31/14 NIH/NINDS
Role of social stress-induced cytokines in exacerbating an animal model of MS

NIH/T32 MH65728-01 (PI: Gonzalez-Lima) 7/1/07-6/30/11 NIH/NIMH
Behavioral Neuroscience Minority Training Grant
Role: Executive Committee and training faculty

R01AG07805 (PI: Griffith; coI: Meagher) 10/1/05-8/31/08 NIH/NIA
Physiology of cholinergic basal forebrain neurons

NSF Graduate Fellowship 2005-2008 NSF
Role: Sponsor for Elisabeth Good-Vichaya, “Exaggerated pain states in Theiler’s virus infection”

R01NS39569 (PI: Welsh; coI: Meagher) 4/01/02-03/31/07 NIH/NINDS
Stress effects on an animal model of autoimmune disease

F31NS50476-2 9/01/04-8/31/06 NIH/NINDS
National Research Service Award, Individual Predoctoral Fellowship
Role: Sponsor for Robin Johnson, “Social stress induced inflammation: Role of cytokines”

NSF Graduate Fellowship 2001-2004 NSF
Role: Sponsor for Robin Johnson, “The impact of social stress on Theiler’s virus infection”.

RG3128 (PI: Welsh; co-PI: Meagher) 9/01/99-08/31/2003 National Multiple Sclerosis Society
The effects of stress on the neuropathogenesis of Theiler’s virus infection

R01MH54557 (PI: Grau; col: Meagher) 12/01/96-11/31/00 NIH/NIMH
Sensitization: Behavioral properties & neural mechanisms

R01MH60157 (PI: Grau; col: Meagher) 7/1/92-6/4/96 NIH/NIMH
Spinal plasticity and pain: mechanisms and function

R03Mh48994 (PI: Grau; col: Meagher) 7/01/92-6/30/94 NIH/MIMH
Spinal Plasticity and Pain: Mechanisms and Function

BNS 881981 (PI: Grau; col: Meagher) 3/89-8/91 NSF
Conditioned changes in pain reactivity: The variables determining the direction and the form of the conditioned response
Justin Moscarello, PhD
Assistant Professor
Department of Psychology Institute for Neuroscience
Texas A&M University
jmm31@tamu.edu

Education & Training

Degrees

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<th>Year</th>
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<th>Institution</th>
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<td>2010</td>
<td>PhD Psychology</td>
<td>University of California, Santa Barbara</td>
<td>Dissertation: The role of the medial prefrontal cortex and nucleus accumbens in motivation and reinforcement</td>
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<td>2003</td>
<td>BA Physical Anthropology</td>
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Academic Honors

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<td>2009</td>
<td>Harry J. Carlisle Award for Outstanding Graduate Student</td>
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<td>2006</td>
<td>Advanced to PhD candidate with distinction</td>
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<td>2003</td>
<td>Graduated magna cum laude and with distinction in major</td>
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Positions & Employment

Faculty

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<td>2017</td>
<td>Assistant Professor</td>
<td>Department of Psychology Institute for Neuroscience Texas A&amp;M University</td>
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Research

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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>
</tr>
<tr>
<td>2003-04</td>
<td>Laboratory Technician</td>
<td>Ettenberg Lab, Department of Psychological &amp; Brain Sciences, UCSB</td>
</tr>
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</table>
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.
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.)

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<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>
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<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>
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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
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.
**NAME:** PETER P. NGHIEM  
**POSITION TITLE:** ASSISTANT PROFESSOR (TENURE-TRACK), 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.)

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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>Texas A &amp; M University (TAMU)</td>
<td>DVM</td>
<td>05/2008</td>
<td>Medicine &amp; Surgery</td>
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<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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<tr>
<td>Children's National Medical Center (CNMC)</td>
<td>Fellowship</td>
<td>08/2010</td>
<td>Vet Neurology</td>
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<tr>
<td>CNMC</td>
<td>F32 NRSA</td>
<td>08/2013</td>
<td>Molecular Medicine</td>
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<td>The George Washington University (GWU)</td>
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<td>01/2014</td>
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<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-NIAMS 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.
Piercy RJ, Kornegay JN. Whole genome sequencing reveals a 7 base-pair deletion in DMD exon 42 in a
   (*Corresponding author)
Clinical progression of X-linked muscular dystrophy in two German shorthaired pointers. J Am Vet Med

   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.
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: 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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<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>
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<tr>
<td>University of Colorado Boulder, Boulder,</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.


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
Texas A&M Institute for Neuroscience
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 neuroscience, 10-year survey, 1996-2006).

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

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 meeting Washington, D.C. Fall, 2002

National Science Foundation
Behavioral Neuroscience and Endocrinology Grant Review Panel meeting Washington, D.C. Spring, 2003

National Science Foundation
Behavioral Neuroscience and Endocrinology Grant Review Panel meeting Washington, D.C. Fall, 2003

National Science Foundation
Behavioral Neuroscience and Endocrinology Grant Review 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 D.C. 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 (external grant

United States- Israel Binational Science Foundation Grant Program (external

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

Journal of Neuroscience
Behavioral Brain Research
Brain Research
European Journal of Pharmacology
Hormones and Behavior
European Journal of Neuroscience
Psychopharmacology
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, University of Texas, 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 Graduate

Texas A&M Institute for Neuroscience 477
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JOURNAL PUBLICATIONS (REFEREED) – LAST FIVE YEARS


BOOK CHAPTERS


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>DEGREE (if applicable)</th>
<th>Completion Date</th>
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<td>06/2002</td>
<td>Biopsychology</td>
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<td>University of Pennsylvania</td>
<td>Ph.D.</td>
<td>12/2008</td>
<td>Neuroscience</td>
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<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.


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
F32 DA031519-02  Smith, Rachel J (PI)  04/15/11-01/24/13
Molecular Mechanisms of Cocaine-Induced Alterations in Accumbens AMPA Receptors
Role: PI

F31 DA019733-01  Smith, Rachel J (PI)  09/01/05-08/31/07
Involvement of protracted withdrawal in morphine relapse
Role: PI
**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>1989</td>
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<td>University of Maine</td>
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<td>Zoology</td>
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<td>University of California, Los Angeles</td>
<td>Ph.D.</td>
<td>1998</td>
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<td>Postdoc</td>
<td>1999-2004</td>
<td>Neurosci</td>
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</table>

**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
2010- Associate Professor, Department of Biology, Texas A&M University
2004-2009 Assistant Professor, Department of Biology, Texas A&M University
2000-2004 Postdoctoral Researcher, Department of Physiological Science, UCLA.
2002 Grass Fellow, Woods Hole Marine Biological Laboratory

Other Relevant Professional Experience
2011- Chair, Graduate Program Texas A&M Institute for Neuroscience
2011- Graduate Advisor, Texas A&M Institute for Neuroscience
2012- Environmental Consultant, City of San Antonio (Bat Expert)
2016- Chair, Institutional Animal Care and Use Committee, Texas A&M

Honors
1992 University of Maine, Outstanding Graduate Student Research Fellowship
1997 Edith Hyde Memorial Scholarship in Physiology (UCLA)
1998 UCLA Dissertation Year Fellowship
2000-2003 NIH-NRSA F32 Post-doctoral Fellowship (NIDCD)
2000 Capranica Foundation Award in Neuroethology (Honorable Mention)
2002 Woods Hole Marine Biological Laboratory Young Investigator Award

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) 9/192015-8/31/2017
“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.
Micah J. Waltz
Texas A&M University
College of Veterinary Medicine and Biomedical Sciences Department of Veterinary Integrative Biosciences
660 Raymond Stotzer Parkway, VIDI 4458 College Station, Texas 77843-4458 mwaltz@cvm.tamu.edu
979-862-8152

EDUCATION
Ph.D. in Biomedical Sciences Texas A&M, College of Veterinary Medicine and Biomedical Sciences, College Station, Texas, 77843-4458
2015 – Emphasis: Epidemiology
Present Co-chairs: C. Budke, B. Gastel

M.S. in Biomedical Sciences West Virginia University, School of Medicine, Morgantown, WV
2008 - 2014
Emphasis: Cellular and Integrative Physiology
Certification: Certificate in University Teaching
- A 15 cr. hour degree certificate with core coursework focusing on general education as well as discipline-specific pedagogy.

B.S. in Zoology Idaho State University, School of Arts and Sciences, Pocatello, ID 2004-2008
Advisor: C. Anderson

ACADEMIC POSITIONS
Lecturer Texas A&M, College of Veterinary Medicine and Biomedical Sciences, College Station, Texas, 77843-4458
Fall 2016 - University Libraries, Medical Sciences Library Present Joint Appointment, Educational Programs Coordinator
Fall 2014 – College of Veterinary Medicine and Biomedical Sciences, Present Department of Veterinary Integrative Biosciences

TEACHING EXPERIENCE
Course Instructor Texas A&M University, College of Veterinary Medicine and Biomedical Sciences, Spring 2015 - College Station, TX
Present Veterinary Integrative Biosciences 310: Biomedical Writing
Face-to-face and online sections

Course Instructor Texas A&M University, College of Veterinary Medicine and Biomedical Sciences, College Station, TX
Fall 2014 - Present Veterinary Integrative Biosciences 311: Biomedical Explorations through Narrative
Face-to-face and online sections

Guest Lecturer Texas A&M University, College of Veterinary Medicine and Biomedical Sciences, College Station, TX
Spring 2015 - Spring 2017 Veterinary Integrative Biosciences 650: Introduction to Graduate Education in the Veterinary Medicine and Biomedical Sciences Environment

STUDY ABROAD EXPERIENCE
Course Instructor Texas A&M University, College of Veterinary Medicine and Biomedical Sciences, Spring 2015 - College Station, TX
Present Study Abroad Program, AIB, Bonn, Germany
Veterinary Integrative Biosciences 447: Neurophysiology of Music

PREVIOUS COURSES TAUGHT
Course Instructor West Virginia University, Eberly School of Arts and Sciences, Morgantown, WV
Lab Manager Biology 340: Invertebrate Zoology
Fall 2013

Guest Lecturer West Virginia University, Eberly School of Arts and Sciences, Morgantown, WV Spring 2012 - West Virginia University, School of Medicine, Morgantown, WV
Physiology 241:Elementary Physiology Physiology 441: Mechanism of Body Function

Teaching Assistant West Virginia University, Eberly School of Arts and Sciences, Morgantown, WV
Fall 2011 BIOL 115: Introductory Biology for Majors labs
Spring 2014  BIOL 117: Introductory Physiology for Majors labs

RESEARCH EXPERIENCE

Educational Research  Texas A&M, College of Veterinary Medicine and Biomedical Sciences, Fall 2016 - University Libraries, Medical Sciences Library
Present  Research task force on accreditation-driven information management competencies in health professions’ curricula.

Program Effectiveness  West Virginia University, School of Medicine, Morgantown, WV
Evaluation  Evaluation of online component of undergraduate course in a Physiology Classroom
Spring 2014

Educational Research, West Virginia University, School of Arts and Sciences, Morgantown, WV Fall 2013
Curriculum Development and Evaluation for BIOL 340: Invertebrate Zoology

Research Assistant  West Virginia University, School of Arts and Sciences, Morgantown WV 2011-2013
S. Farris/J. Belanger labs, Neuroanatomy/Neuroethology

Research Assistant  National Institute of Occupational Health (NIOSH), Morgantown WV
2009-2011  A. Shvedova lab, Pulmonary Toxicology, Dept. of Physiology and Pharmacology

PEER-REVIEWED PUBLICATIONS


EDITOR-REVIEWED PUBLICATIONS

2017 Moberly HK, Bankston S, Waltz MJ. An evidence-based approach to teaching the critical evaluation of scientific literature. In: Mallon, M; Bradley, C; husman, R; Hays, L; and Belanger, J; eds. The Grounded Instruction Librarian: Participating in the Scholarship of Teaching and Learning. (accepted)

NON-PEER-REVIEWED PUBLICATIONS

2016 Waltz MJ. International Programs at the CVM. CVM Today, 18(1): 34-35


PRESENTATIONS/ABSTRACTS

2018 Waltz MJ, Moberly HK, Meador A, Carrigan EE. Identifying information-related competencies to align educational support Qualitative and Quantitative Methods in Libraries, 05/22/18-05/25/18; Crete, Greece.

2017 Waltz MJ, Moberly HK, Budke CM. De-mythifying observational study design: modelling deliberate library collaboration to support competency-based curricula.
European Association for Health Information and Libraries, 06/12/17-06/16/17; Dublin, Ireland.

2012  Waltz MJ, Belanger JH
Sound Reception in Crabs: Keeping an ear to the ground and a leg to the wind.
Society for Neuroscience, 10/13/12-10/17/12; New Orleans LA, USA.

2011  Waltz MJ, Murray A, Kisin E, Tkach A, Shvedova A
“Osteopontin and TGF-β1 release in response to single walled carbon nanotube exposure.”
Society of Toxicology, 03/06/11-03/10/11; Washington D.C., USA.

COMMITTEES/SERVICE
2016  Texas A&M University, College of Veterinary Medicine, College Station, TX Staff Awards Committee

2015 - Texas A&M University, College of Veterinary Medicine, College Station, TX Present Scholarships and Award Committee

PROFESSIONAL DEVELOPMENT
2018  Texas A&M University, Student Services
  Green Dot Bystander Training

2018  Texas A&M University, Provost’s Office
  Hello2 Teaching Certificate
  In progress

2015 -  Texas A&M University, Professional Programs Office

2016  Aggie Allies Facilitator Training

2015  Texas A&M University, Professional Programs Office
  Aggie Allies Training

2015  Texas A&M University, Department of Instructional Technology Services
  Flipping the Classroom: Faculty Institute

2015  Texas A&M University, Department of Instructional Technology Services
  Professional Certification in Online Teaching

2011  West Virginia University
  Summer Institute for Undergraduate Scientific Teaching

PROFESSIONAL ORGANIZATIONS/AFFILIATIONS
2016 – Evidence Based Veterinarian Medicine Association Present

2015 – Texas Veterinary Medical Association Present

2012 – American Physiological Society Present
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.)

<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 London, U.K.</td>
<td>B.Sc.</td>
<td>06/1976</td>
<td>Microbiology</td>
</tr>
<tr>
<td>King’s College Hospital, U.K.</td>
<td>Postdoc</td>
<td>1979-1981</td>
<td>Autoimmune liver</td>
</tr>
<tr>
<td>Dept. of Pathology, Cambridge, U.K.</td>
<td>Postdoc</td>
<td>1982-1985</td>
<td>Rheumatoid arthritis</td>
</tr>
<tr>
<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
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 enterobactericae; (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
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 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</th>
<th>MM/YY</th>
<th>FIELD OF STUDY</th>
</tr>
</thead>
<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>
</tbody>
</table>

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 sialytransferase 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 an 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>
<thead>
<tr>
<th>Year</th>
<th>Position and Honors</th>
</tr>
</thead>
<tbody>
<tr>
<td>1987-1991</td>
<td>Postdoctoral Fellow, Department of Zoology and Genetics, Iowa State University</td>
</tr>
<tr>
<td>1991-1997</td>
<td>Assistant Professor, Department of Biology, Texas A&amp;M University</td>
</tr>
<tr>
<td>1997-2011</td>
<td>Associate Professor, Department of Biology, Texas A&amp;M University</td>
</tr>
<tr>
<td>2001-2007</td>
<td>Chair, Faculty of Neuroscience, Texas A&amp;M University</td>
</tr>
<tr>
<td>2003-Present</td>
<td>Associate Dean for Graduate Studies, College of Science, Texas A&amp;M</td>
</tr>
<tr>
<td>2011-Present</td>
<td>Professor, Department of Biology and Neuroscience, 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>
</tr>
</tbody>
</table>

Other Experience and Professional Memberships

<table>
<thead>
<tr>
<th>Year</th>
<th>Position and Honors</th>
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<tbody>
<tr>
<td>1987-Present</td>
<td>Member, Society for Neuroscience</td>
</tr>
<tr>
<td>1999-Present</td>
<td>Director, Cellular and Molecular Imaging Facility, Texas A&amp;M University</td>
</tr>
<tr>
<td>2001-Present</td>
<td>Member, Center for Biological Clocks Research, Texas A&amp;M University</td>
</tr>
<tr>
<td>2003-Present</td>
<td>Chair, College of Science, Graduate Instruction Committee</td>
</tr>
<tr>
<td>2003-Present</td>
<td>Member, TAMU Graduate Operations Committee</td>
</tr>
<tr>
<td>2005-Present</td>
<td>Member, Society for Research on Biological Rhythms</td>
</tr>
<tr>
<td>2005-Present</td>
<td>Research Associate, Texas Brain and Spine Institute</td>
</tr>
<tr>
<td>2006-Present</td>
<td>NSF LSAMP Bridge to the Doctorate Program, TAMU Advisory Group</td>
</tr>
<tr>
<td>2009-2011</td>
<td>Member, Biosafety Advisory Committee, Texas A&amp;M VP for Research</td>
</tr>
<tr>
<td>2011-2015</td>
<td>Chair, Texas A&amp;M University Graduate Council</td>
</tr>
</tbody>
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Honors

<table>
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<tr>
<th>Year</th>
<th>Award Description</th>
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</thead>
<tbody>
<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>
</tbody>
</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 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**

<table>
<thead>
<tr>
<th>Project ID</th>
<th>PI/Co-I</th>
<th>Start/End Date</th>
<th>Funding Agency</th>
<th>Project Title</th>
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<tr>
<td>R01 NS075534</td>
<td>V. Panin (PI)</td>
<td>08/01/2011-07/31/2016</td>
<td>NIH/NINDS</td>
<td>The Control of Neural Transmission by Glycosylation</td>
</tr>
<tr>
<td>P01 NS39546</td>
<td>D. Bell-Pedersen (PI)</td>
<td>07/01/2006-06/30/2011</td>
<td>NIH/NINDS</td>
<td>Coordination of Circadian Physiology of Diverse Species</td>
</tr>
<tr>
<td>P01 NS39546</td>
<td>V. Cassone (PI)</td>
<td>12/01/1999-06/30/2006</td>
<td>NIH/NINDS</td>
<td>Coordination of Circadian Physiology of Diverse Species</td>
</tr>
</tbody>
</table>
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 H

Curricula Vitae
For
Support Faculty
APPENDIX H  Curricula Vitae for Support Faculty

Gerianne M. Alexander
Professor and Associate Dean of Research and Graduate Programs

POSITIONS:

- Associate Dean, Texas A&M University/College of Liberal Arts 2012-present.
- Professor, Texas A&M University/Department of Psychology 2011-present.
- Associate Professor, Texas A&M University/Department of Psychology 2005-2011.
- Assistant Professor, Texas A&M University/Department of Psychology 2002-2005.
- Research Associate, Yale Child Study Center 1999-2002.
- Research Associate, Yale/Department of Anesthesiology 1998-1999.
- Assistant Professor, University of New Orleans/Department of Psychology 1993-1998.

EDUCATION:

- Ph.D. (Clinical Psychology) McGill University 1991
- B.A. (First Class Honors) St. Francis Xavier U. 1984
- B.A.Mus (First Class Honors) St. Francis Xavier U. 1981

SELECT HONORS/AWARDS:


GRANTS AS PI, co-PI, or co-I:

- "Age Discrimination in Hiring: Eye-tracking during the Resume Review process", Sloan Foundation (Co-PI), 2012-2013. Total award of $116,000.
- "Eye-tracking Studies of Gender Development", National Science Foundation (PI), 2006-2011. Total award of $481,000.
SELECT PUBLICATIONS (of 61 journal articles, 3 book chapters):


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: Gregg C. Allen, Ph.D.

POSITION TITLE: Associate Professor (instructional)

eRA COMMONS USER NAME: alleng

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>
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<tbody>
<tr>
<td>University of Oregon, Eugene, OR</td>
<td>B.S.</td>
<td>1991-1995</td>
<td>Biology</td>
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<tr>
<td>Texas A&amp;M University, College Station, TX</td>
<td>Ph.D.</td>
<td>1996-2001</td>
<td>Medical Sci.- Neurobiology</td>
</tr>
<tr>
<td>Texas A&amp;M University, College Station, TX</td>
<td>Postdoctoral</td>
<td>2002-2005</td>
<td>Neuroscience</td>
</tr>
<tr>
<td>Vanderbilt University, Nashville, TN</td>
<td>Postdoctoral</td>
<td>2005-2009</td>
<td>Neuroscience</td>
</tr>
</tbody>
</table>

A. Positions and Honors.

Positions and Employment

1993-1995 Undergraduate Research Assistant, Dept of Biology, University of Oregon
1996-2001 Graduate Research Assistant, Dept of Anatomy & Neurobiology, Texas A&M University
2002-2005 Postdoctoral Fellow, Dept of Anatomy & Neurobiology, Texas A&M University
2005-2009 Postdoctoral Research Associate, Dept of Biological Sciences, Vanderbilt University
2009-2017 Assistant Professor, Dept of Neuro & Exp Therapeutics, Texas A&M University 2017-present Associate Professor, Dept of Neuro & Exp Therapeutics, Texas A&M University

Research Awards

1998 1st Place, Presentation Award, Texas A&M Univ. Society for Neuroscience
1999 Graduate Teaching Award, Texas A&M Univ. Medical Gross Anatomy & Neuroscience
1999 3rd Place, Presentation Award, Texas A&M Univ. Graduate Student Organization
1999 3rd Place, Presentation Award, Texas A&M Univ. Society for Neuroscience
2000 Graduate Travel Award, Society for Neuroscience
2000 1st Place, Presentation Award, Texas A&M Univ. Society for Neuroscience
2001 1st Place, Presentation Award, Texas A&M Univ. Graduate Student Organization
2001 Keystone Symposia Scholarship, National Institute of Mental Health
2001 Research Grant, Texas A&M Univ. Office of Graduate Studies 2001 Training Fellowship, Texas A&M Univ. Circadian Clocks Program
2005 – 2009 Fellow, Vanderbilt University Scientist-Educator Program
2006 – 2009 Fellow, Institutional Research & Academic Career Development Award (IRACDA)
NIH 5K12 GM068543-04

B. Selected peer-reviewed publications (in chronological order).


c. Research Support.

Completed Research Support

PO1 NS 039546 Earnest (PI) 01/02-08/05
NINDS
Analysis of Clock and Clock Controlled Genes in Immortalized SCN Cells
This project examined the roles of clock genes in circadian rhythm generation in the hypothalamic SCN. Role: Postdoctoral Research Associate

RO1 AA 013242 Earnest (PI) 01/02-08/05
NIAAA
Developmental Alcohol and Circadian Clock Function
This project examined long-term effects of early postnatal alcohol exposure upon the circadian timing system in adult rats.
Role: Postdoctoral Research Associate

RO1 MH 060147 Earnest (PI) 01/02-08/05 NIMH
Role of BDNF in the Photic Control of Circadian Rhythms
This project examined the role of brain-derived neurotrophic factor (BDNF) and its TrkB tyrosine kinase receptor in the photic regulation of SCN circadian function.

Role: Postdoctoral Research Associate

R01 EY09256-16 McMahon (PI) 4/04-3/09. NIH/NEI
Mechanisms of Retinal Synaptic Plasticity
This project investigates the mechanisms by which glutamatergic and electrical synapses are modulated by light adapting signals in retinal neurons and circuits.

Role: Postdoctoral Research Associate
NAME: Christen Elizabeth Boudreau, DVM, PhD, DACVIM (Neurology)

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

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>
<th>Completion Date MM/YYYY</th>
<th>FIELD OF STUDY</th>
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<tr>
<td>Baylor College of Medicine</td>
<td>PhD</td>
<td>10/2001</td>
<td>Neuroscience</td>
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<tr>
<td>Texas A&amp;M University</td>
<td>DVM</td>
<td>5/2010</td>
<td>Veterinary medicine</td>
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<tr>
<td>University of California, Davis</td>
<td>Diplomate</td>
<td>8/2014</td>
<td>ACVIM (Neurology)</td>
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</tbody>
</table>

A. Personal Statement
My veterinary career has to date been divided between clinical practice, clinically oriented research, and training of new veterinarians. As a clinician and teacher at a tertiary referral hospital, I have the opportunity to see regularly those complex or unusual cases that challenge me to find ways to improve our skill as a profession in the diagnosis and treatment of veterinary neurological disease. Directly or indirectly, my experience with these patients guides my research interests, which focus on areas where current understanding of disease or available diagnostics and treatment are inadequate to meet the needs of my patients. These studies include evaluation of the quality of existing diagnostic instruments, exploration of novel relationships between neurological and systemic disease processes, examination of the basic biology of poorly understood diseases, and novel therapeutic approaches to difficult clinical problems. Because of the relative rarity of some of these neurological diseases, I most often collaborate with veterinary neurological specialists at partner institutions on these projects. Because of the translational potential of some of my work, I also have membership in and collaborations with research groups from human medicine that work on similar problems. In my role as a mentor to clinical year students and veterinary house officers, I can provide young veterinarians with opportunities to begin careers in research and stimulate their interest in the important role of clinician-scientists in veterinary medicine.

B. Positions and Honors
List in chronological order, previous positions first, concluding with your present position. List any honors. Include present membership on any Federal Government public advisory committee.

Positions and Employment
2016 – present  Department of Small Animal Clinical Sciences  Assistant Professor  
Texas A&M University, College Station, TX
2014 – 2016
Department of Small Animal Clinical Sciences  Clinical Assistant Professor
Texas A&M University, College Station, TX

2011 – 2014
Department of Surgical and Radiological Services  Resident in Neurology/Neurosurgery
University of California, Davis

2010 – 2011
Veterinary Medical Center
The Ohio State University Rotating Small Animal Intern

Honors

2016  Juan Carlos Robles Emanuelli Teaching Award
Texas A&M University, College Station, TX

2016  Outstanding Young Faculty Research Award
Texas A&M University, College Station, TX

2013  Resident Research Award
American College of Veterinary Internal Medicine Forum

C. Contribution to Science:

Major Contributions

1. Investigation of clinical correlation of MRI findings with risk factors and outcome in small animal veterinary patients
   b. Kerwin SC, Levine JM, Budke CM, Griffin JF 4th, Boudreau CE. Putative cerebral microbleeds in dogs undergoing magnetic resonance imaging of the head: a retrospective study of demographics, clinical associations, and relationship to case outcome. JVIM 2017 PMID: 28556471

2. Investigation of biochemical and clinical issues in naturally occurring spinal cord injury in dogs
   a. Russell RL, Levine JM; Jeffery ND, Young C; Mondragon A, Lee B; Boudreau CE; Welsh CJ; Levine GJ. Arachidonic acid pathway alterations in cerebrospinal fluid of dogs with naturally occurring spinal cord injury. BMC Neuroscience 2016 (PMID 27287721)
3. Pioneering efforts in cellular and molecular characterization of canine and feline gliomas
   c. Rissi DR, Porter BF, Boudreau CE, Krimer PM, Miller AD. Immunohistochemical characterization of the inflammatory cell population in feline glioma. Journal of Comparative Pathology 2018 (PMID 29729717)

Complete List of Published Work in My Bibliography:

D. Research Support (recent)

Ongoing Research Support:
MicroRNA profiling in canine glioma. Boudreau (PI). Ginn Fund ($10,000), funded 8/2016
Goal: Next-generation sequencing of microRNA in canine gliomas
Role: Mentor-principal investigator

Genomic and Immunological Canine Glioma Characterization. dePinho (PI) NCI P30 CCSG ($500,000), funded 8/2016
Goal: Genomic and Immunological Canine Glioma
Role: Investigator

Completed Research Support During the Last Three Years:
None
BIOGRAPHICAL SKETCH

NAME: Candice Brinkmeyer-Langford

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

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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>Texas A&amp;M University</td>
<td>BS</td>
<td>12/2002</td>
<td>Genetics</td>
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<tr>
<td>Texas A&amp;M University</td>
<td>PHD</td>
<td>12/2006</td>
<td>Genetics/Genomics</td>
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<tr>
<td>Texas A&amp;M University</td>
<td>Postdoctoral Fellow</td>
<td>12/2009</td>
<td>Immunogenetics</td>
</tr>
<tr>
<td>Texas A&amp;M University</td>
<td>Postdoctoral Fellow</td>
<td>12/2010</td>
<td>Neuroimmunology, genetics</td>
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</table>

A. Personal Statement
My role in the proposed project is PD/PI. I have assembled a research team that includes collaborators with expertise complementary to my own. My background is in the field of genomics; I am proficient at many of the techniques used in genomics-related research that will be important for the proposed work. My first postdoctoral appointment broadened my knowledge of the immune system and how underlying genetic variations contribute to variability in the immune response. During this time I received my first federal funding (a USDA postdoctoral grant) to study the structure and organization of the major histocompatibility complexes (MHC) of horses and cattle. I was able to associate some of these variations with health conditions including variable responses to infections. My second postdoctoral experience (with C. Jane Welsh, Co-I on the proposed project) gave me the opportunity to work with the Theiler's virus mouse model of multiple sclerosis. I gained experience with using mouse models, including scoring mice for neurological deficiencies. As a junior faculty member, I regularly participate in career-development activities and workshops, and I benefit from the guidance of NIH-funded, fully tenured faculty mentors (Drs. C. Jane Welsh and Farida Sohrabji).

Being diagnosed with multiple sclerosis at the start of my graduate studies has played an integral role in my career choices, including the decision to remain at Texas A&M University. I enjoy the complete support of my department and the university and have found the environment at Texas A&M to be rich with diversity – in faculty, research interests, and opportunities, owing largely to its size and status as a top-tier research university. Therefore, I am confident in my ability to succeed in an independent academic research career. In summary, I have a record of productive and diverse research projects in areas that are highly relevant to the study of complex conditions such as those to be studied in the proposed work. My experience and expertise, together with those of the co-investigators and collaborators, make me confident I can lead the proposed project.

B. Positions and Honors Positions and Employment
2010 - 2012  Assistant Research Scientist, TEXAS A&M UNIVERSITY
2011 - 2012  Assistant Research Scientist, TEXAS A&M AGRILIFE RESEARCH
2013 - Research Assistant Professor, TEXAS A&M UNIVERSITY

Other Experience and Professional Memberships
1999 - 2000  Member, Phi Theta Kappa National Honor Society
2003 - 2006  Member, Texas A&M College of Veterinary Medicine Graduate Student Association 2003 - 2006  Member, Texas A&M University Graduate Student Association
2004 - Member, Texas Genetics Society
2005 - 2005  Representative, Texas A&M University Graduate Student Council 2006 - 2009  Member, International Equine Genome Consortium
2007 - 2008  Member, Center for Animal Biotechnology and Genomics
2008 - Member, Texas A&M Institute for Genome Sciences and Society (formerly Whole Systems Genomics Institute)
2009 - Member, Texas A&M Institute for Neuroscience
2011 - 2011  Member, Texas A&M Center for the Integration of Research, Teaching & Learning (TAMU-CIRTL) Post-Doctoral Professional Development program workshops
2011 - 2012  Participant, Center for Integrated Research, Teaching and Learning Coffee Hour series
2013 - Adjunct member, Texas A&M Graduate Faculty
2013 - Member, Society for Neuroscience - Texas A&M Chapter 2014 - Ad hoc reviewer, Disease Models and Mechanisms
2014 - Ad hoc reviewer, Association Francaise contre les Myopathies (AFM - French muscular dystrophy association, funding agency)
2014 - Ad hoc reviewer, Neurotoxicology
2014 - Chair, Texas A&M Dept. of Veterinary Integrative Biosciences “Specific Aims Group” Faculty Proposal Review Committee
2014 - 2014  Member, Texas A&M University Summer Bioinformatics Workshop 2015 Ad hoc reviewer, Canine Genetics and Epidemiology
2015 - Ad hoc reviewer, Animal Genetics
2015 - Chair - monthly meetings, Texas A&M College of Veterinary Medicine Neuroscience Faculty
2015 - 2015
2015 - Ad hoc reviewer, Bioinformatics and Biology Insights
2016 - Ad hoc reviewer, BMC Musculoskeletal Disorders
2017 - Ad hoc reviewer, G3
2017 - Ad hoc reviewer, Scientific Reports
2017 - Ad hoc pilot project reviewer for CTEHR “P30 Centers Pilot Grant Reviewer Consortium”: Michigan Center on Lifestage Environmental Exposures and Disease (M-LEEaD) Center

Honors
2002  Distinguished Student Award, Texas A&M College of Agriculture and Life Sciences 2006  Academic Excellence Award, Assoc. of Former Students Memorial Scholarship 2006  Best Graduate Student Platform Presentation, Texas Genetics Society Meeting
2007  University Distinguished Graduate Student Award, Texas A&M Assoc. of Former Students 2007  Graduate Award, Texas A&M Veterinary Faculty Auxiliary
2014  Invited participant, NINDS Grant Writing Workshop for Diverse Researchers
2014  Invited participant, Texas A&M ADVANCE Center for Women Faculty Roadmap for a Successful Academic Career Workshop
2015  Invited poster presentation, Population-Based Rodent Resources for Environmental Health Sciences Meeting

C. Contributions to Science
1. Development of high-resolution comparative maps of the equine genome. Before whole-genome sequence data was available for horses, high-resolution gene maps provided information about genome synteny,
evolution, and chromosome structure. Our group constructed high-resolution radiation hybrid and bacterial artificial chromosome (BAC) maps that formed the skeleton upon which sequence data was eventually organized. These maps provided new insights into the organization of the horse genome and facilitated the construction of comparative maps across species.


2. Identification and characterization of polymorphisms and haplotype structure of the equine and taurine leucocyte antigen complexes. The ability of an organism to identify and destroy foreign substances and to distinguish between self and non-self is regulated by genes within the MHC, which encode proteins involved in the initial stages of acquired and innate immune responses. Consequently, genetic variations among genes of the MHC have been frequently associated with predispositions to autoimmune diseases and susceptibilities to various pathogens in humans and animals.


3. Characterizing contributions of the MHC to autoimmune and neurological conditions. The MHC contributes to susceptibility to autoimmune conditions in humans and animals. These conditions are often further affected by environmental exposures. We evaluated the contributions of genetic and environmental components of autoimmune diseases in mice and horses.


4. **Identification of genetic modifiers of inflammatory and degenerative disease.** Chronic inflammation and degeneration are hallmarks of many conditions in humans and animals. Dogs share a phenotypic and immunological similarity to humans for some of these diseases, including Duchenne muscular dystrophy (DMD).

5. **Development of novel genomic tools for exploring gene expression variability.** Our findings have resulted in useful tools for exploring gene expression variability with greater precision and depth, which will be utilized in the experiments proposed in this project.


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

**Ongoing Research Support**
R01 NS103934-01 Brinkmeyer-Langford (PI) 09/01/2017-08/31/2022 NINDS
Host genetic determinants of diversity in viral-induced neuropathology
The goal of this project is to determine how genetic background influences disease diversity following TMEV infection. The central hypothesis is that genetic background, as modeled by a new population-based mouse model, will differentially modify susceptibility to TMEV-induced diseases based upon genetic polymorphisms. The rationale for the proposed research is that a delineation of the genetic effects underlying the diverse outcomes of TMEV infection is likely to contribute new insights into the heterogeneity of virally induced human neurological conditions.
RD-83580201 Rusyn (PI) 06/01/2015-05/31/2019 EPA STAR
Cardiotoxicity Adverse Outcome Pathway: organotypic culture model and in vitro-to-in vivo extrapolation for high-throughput hazard, dose-response and variability assessments.
This Center will develop and validate a population-based human and mouse organotypic culture model for characterizing susceptibility and variability in cardiac toxicity.
Role: Quality Assurance Manager

T32 ES026568 Rusyn (PI) 04/01/2016-03/31/2021 NIH NIEHS
Regulatory Science in Environmental Health and Toxicology
This training program aims to strengthen training and research base at Texas A&M Interdisciplinary Faculty of Toxicology program and also to provide unique focus on regulatory science, a scientific discipline consisting of the development and application of scientific methods, tools, and approaches that are used to support regulatory and other policy objectives.
Role: Coordinator of Writing Skills program; Co-Mentor

U24 Rusyn (PI) 09/23/2016-08/31/2018 NIH NCATS
TEX-VAL: Texas A&M Tissue Chip Validation Center
This purpose of this award is to establish a Tissue Chip Validation Center at Texas A&M University (TEX-VAL Center).
Our goal is to provide resources, personnel and infrastructure for tissue chip validation using standardized methodologies and reference test compounds.
Role: Quality Assurance Manager

P42 ES027704 Rusyn (PI) 04/01/2017-03/31/2022 NIH/NIEHS
Comprehensive tools and models for addressing exposure to mixtures during environmental emergency-related contamination events
This Center brings together a team of scientists from biomedical, geosciences, data science and engineering disciplines to design comprehensive solutions for complex exposure- and hazard-related challenges. Our overall theme is to characterize and manage both existing and environmental emergency-created hazardous waste sites through the development of the tools that can be used by first responders, the impacted communities, and the government bodies involved in site management and cleanup.
Role: Administration Core; Quality Assurance Manager
Rebecca J. Brooker

**Assistant Professor**

**POSITIONS:**

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<thead>
<tr>
<th>Position</th>
<th>Institution</th>
<th>Dates</th>
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<tr>
<td>Assistant Professor</td>
<td>Texas A&amp;M University</td>
<td>8/2017 – present</td>
</tr>
<tr>
<td>Honorary Fellow</td>
<td>University of Wisconsin - Madison</td>
<td>8/2013 – present</td>
</tr>
<tr>
<td>Assistant Professor</td>
<td>Montana State University</td>
<td>8/2013 – 7/2017</td>
</tr>
<tr>
<td>Research Scientist</td>
<td>University of Wisconsin – Madison</td>
<td>4/2013 - 8/2013</td>
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**EDUCATION:**

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<tr>
<td>Ph.D.</td>
<td>The Pennsylvania State University</td>
<td>2011</td>
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<tr>
<td>M.S.</td>
<td>The Pennsylvania State University</td>
<td>2008</td>
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<tr>
<td>B.A.</td>
<td>Central College</td>
<td>2003</td>
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**SELECT HONORS/AWARDS:**

- *Faculty Award for Excellence, MSU Alumni Association & Bozeman Chamber of Commerce* (2017)
- *Association for Psychological Science Rising Star* (2015)

**GRANTS:**

Montana State University College of Nursing

- **Bio-energy for stress relief: Future directions**
  - Role: Co-Investigator (PI: Dr. Alice Running)
  - Amount: $4,192.00

The John Templeton Foundation: University of Virginia Genetics and Human Agency

- **How do Perceptions About Heritability Influence Complex Child-Parent Interactions and Character Development?**
  - Role: Co-Investigator (PI: Dr. Matthew Vess)
  - Dates: September 2016 – August 2019
  - Total Costs: $523,485

P20 GM104417

- **Center for Health Equity in Rural Montana**
  - Role: Pilot Project Leader (PI: Dr. Alexandra Adams)
  - Dates: September 2014-August 2017
  - Amount Awarded: $179,544

State of Montana Commissioner of Higher Education

- **The Synergistic Improvement in the Diagnosis and Treatment of Mental Illness, Dementia, & Chronic Pain**
  - Role: Primary Investigator, Project 1 (PI: Drs. Matt Byerly and Frances Lefcort)
SELECT PUBLICATIONS (of 24 journal articles):


Brooker, R.J., Buss, K.A., Lemery-Chalfant, K., Aksan, N., Davidson, R.J., & Goldsmith, H.H. (2013). The development of stranger fear in infancy and toddlerhood: Normative development,
individual differences, antecedents, and outcomes. Developmental Science, 16(6), 864-878. PMCID: PMC4129944.

SELECT PRESENTATIONS:

Brooker, R.J., & Canen, M.J. (2016). The ERN as a predictor of early fear: The role of context. Paper presented at the annual meeting of the Association for Psychological Science; Chicago, IL.


SIGNIFICANT TEACHING ACTIVITIES:

classroom teaching
Advanced Statistical Analysis, Montana State University
Temperament and Development, Montana State University

undergraduate independent research supervision
Rebekah Lindsey, McNair Scholars Program, Montana State University
Tanner Lineberry, McNair Scholars Program, Montana State University
Randi Phelps, Undergraduate Independent Study, Montana State University
Holly Howe, Undergraduate Independent Study, Montana State University
Lindsey Whitcomb, Undergraduate Independent Study, Montana State University

additional undergraduate supervision
Undergraduate senior thesis consultation (N = 3)
Undergraduate research assistants (8-10 per academic semester; 5-6 per summer term)

graduate student supervision
J. Patrick Begnoche, primary mentor, Montana State University Master’s Program
Mara Canen, primary mentor, Montana State University Master’s Program
Reema Najjar, primary mentor, Montana State University Master’s Program

MAJOR SERVICE ACTIVITIES:

ad-hoc reviewer

- Adoption Quarterly
- Behavior Genetics
- Biological Psychology
- Child Development
- Clinical Psychological Science
- Developmental Neuropsychology
- Developmental Psychobiology
- Developmental Psychology
- frontiers in Genetics (editorial board)
- Hormones and Behavior
- Infant and Child Development
- International Journal of Psychophysiology
- Twin Research and Human Genetics
- Journal of Research on Adolescence
- Journal of Experimental Child Psychology
- Psychoneuroendocrinology
• Emotion (Consulting Editor)
• International Journal of Developmental Neuroscience

• Sex Roles
1. Name: Diane E. Chico, Ph.D.
   Faculty Rank: Associate Professor

2. Current Position: Associate Professor (tenure track, primary: education), Department of Neuroscience & Experimental Therapeutics, Texas A&M University Health Science Center – College of Medicine; 02/2016-present

3. Education

<table>
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<tr>
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<th>Years Attended</th>
<th>Degrees</th>
<th>Field of Study</th>
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<tr>
<td>Saint Edward’s University, Austin, TX</td>
<td>1991-1995</td>
<td>B.S.</td>
<td>Major: Biology</td>
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<td></td>
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<td>Minor: Religious Studies</td>
</tr>
<tr>
<td>University of Texas Medical Branch (UTMB), Galveston, TX</td>
<td>1997-2002</td>
<td>Ph.D.</td>
<td>Cell Biology</td>
</tr>
</tbody>
</table>

4. Post-graduate Training: Postdoctoral Researcher, Department of Biochemistry, Albert Einstein College of Medicine of Yeshiva University (AECOM), Bronx, NY; 2002-2006

5. Professional Experience:
   05/2002-10/2002 Faculty Associate, Dept. of Anatomy & Neurosciences, UTMB
   11/2004-03/2006 Anatomy Instructor, Dept. of Anatomy & Structural Biology, AECOM
   05/2006-05/2009 Assistant Professor (non-tenure, research track), Dept. of Neuroscience & Cell Biology, UTMB
   06/2009-09/2014 Assistant Professor (non-tenure, educator track), Dept. of Neuroscience & Experimental Therapeutics, Texas A&M Health Science Center – College of Medicine
   09/2014-02/2016 Associate Professor (non-tenure, educator track), Dept. of Neuroscience & Experimental Therapeutics, Texas A&M Health Science Center – College of Medicine

6. Medical School Teaching Experience:
   Texas A&M University Health Science Center College of Medicine
   2009-present Medical Gross Anatomy
   2009-present Medical Histology
   2010-present Medical Neuroscience
   2014-2017 CSIE Facilitator, Foundations (M1) and Organ Systems (M2)
   Blocks

   School of Medicine, University of Texas Medical Branch, Galveston, TX
   1998-2001 Microanatomy (first-year course), Laboratory Teaching Assistant
   1999-2002 Gross Anatomy and Radiology (first-year course), Laboratory Teaching Assistant
   1999-2001 Gastrointestinal course (second-year course), Laboratory Teaching Assistant
   2006-2009 Gross Anatomy and Radiology (first-year course); Lecturer, laboratory instructor, PBL facilitator
   2006-2009 Molecules, Cells & Tissues (first-year course); Lecturer, laboratory instructor, PBL facilitator
   2006-2009 Neuroscience and Human Behavior (first-year course); Laboratory instructor, PBL facilitator
   2006-2009 Senior Selective in Anatomy (fourth-year course)
   2008-2009 Pathobiology and Host Defense (first-year course); PBL facilitator

   Albert Einstein College of Medicine, Bronx, NY
   2004-2006 Clinical and Developmental Anatomy (first-year course); Laboratory instructor

   Other Teaching Experience:
   2015-present Texas A&M University Master of Science in Athletic Training, Kinesiology, Laboratory instructor for cadaver labs (ATTR 662, fall – Clinical Diagnosis & Examination: Lower Extremity; ATTR 664, spring – Clinical Diagnosis & Examination: Upper Extremity)
2015-present  Joint Admissions Medical Program (JAMP) (college students), PBL facilitator, lecturer (anatomy and histology)
Neuroscience & Experimental Therapeutics (NEXT) 689 Special Topics Course: Advanced Teaching in the Anatomical Sciences (graduate course; primary instructor): 1 student per session
- 3-5 credit, 18 week course that runs concurrently with Medical Histology (MEID 605) and Medical Gross Anatomy (MEID 607) throughout the Fall semester. Graduate students act as teaching assistants to gain experience in both lecture and laboratory teaching environments. Students are mentored in completing a teaching portfolio and generating a lecture and/or project for medical education research.

7. Medical School Teaching Awards: Texas A&M University Health Science Center, College of Medicine
- 2013 Recipient, Teaching Award for Outstanding Achievement Core Principles of Medicine Professor in the first year medical curriculum, Class of 2016.
- 2014 Recipient, Teaching Award from the TAMU Association of Former Students’ University College-Level.
- 2014 Recipient, R. Kelly Hester Distinguished Teaching Award for Basic Science Education.
- 2015 Recipient, Teaching Award for Outstanding Achievement Student Dedication in the first year medical curriculum, Class of 2018.
- 2016 COM Nominee, Texas A&M University 2016 Presidential Professor for Teaching Excellence Award. 2016 Recipient, Most Inspirational Faculty Award, Class of 2020.
- 2017 Inducted into the Texas A&M University College of Medicine Academy of Distinguished Medical Educators.

8. Medical School Education Administration Experience: Texas A&M University College of Medicine
- 2010-present Medical Histology, co-discipline leader
- 2010-2011 Core Principles of Medicine I in Phase I, co-block leader 2012-2015 Phase I Sub-committee, co-phase leader
- appointed 6/2012 2012-2015 Core Principles of Medicine II in Phase I, co-block leader 2014-2018 COM Curriculum Committee, elected member
- 2014-2017 COM Curriculum Committee, Elected Chair; Past Chair (2017-2018)
- 2015-present Foundations of Medicine I in Pre-Clerkship Curriculum, co-block leader 2017 Foundations of Medicine II in Pre-Clerkship Curriculum, co-block leader
- 2018-present Pre-Clerkship Curriculum Subcommittee, co-leader (March-April 2018) and member

Medical School Education Administration Experience: University of Texas Medical Branch
- 2008 – 2009 UTMB Academic Review Committee, member

9. Institutional Service to the Texas A&M University College of Medicine
- 2010- Student Promotions Committee, appointed member
- 2012- Medical School Admissions Committee, guest interviewer (2012), appointed member (2013); interviewer 2014-2015 Pre-Clerkship Curriculum Reform Subcommittee, member
- 2014-2015 Pre-Clerkship Curriculum Implementation Group, Co-Chair, appointed 2014- Diversity Leadership Council, appointed member
- 2014- Graduate Faculty, Texas A&M University, member
- 2015; 2016 Association of Former Students’ of Texas A&M Award Selection Committee, College Level, appointed member
- 2016-2017 Engineering and Medicine (EnMed) Track Curriculum Steering Committee, member

Other Indices of Service
- 2013; 2015 Reviewer, Faculty Funding Awards by the Office of Medical Education, College of Medicine, Texas
- 2016 A&M University Health Science Center
2009 Academy of Distinguished Medical Educators (ADME) Education Grand Rounds Work Group, volunteer member
2018 Chair, Academy of Distinguished Medical Educators, Elected

10. Professional Development in Medical Education:
2007-2009 Scholars in Education cohort, University of Texas Medical Branch, Galveston, TX
   ▪ This faculty development program is an 18-month program for UTMB faculty who are interested in enhancing their skills as educators.

2010-2012 Leadership Education and Development (LEAD) Program cohort, sponsored by the Southern Group on Educational Affairs of AAMC (first cohort of program)
   ▪ This program was a 20-month program offered by SGEA for SGEA members interested in professional development of leadership skills in medical education. I was part of the first cohort of LEAD participants.

Awards Received for Professional Development | Educational Scholarship

2008 UTMB Academy of Master Teachers Junior Faculty Educational Development Award for research in “Enhancing Gross Anatomy Education”
2009 “Best Poster Presentation by a Medical Educator,” Medical Educational Scholarship Award, Southern Group of Educational Affairs of AAMC
2012 Professional Development Award from the Office of Faculty Development, Texas A&M Health Science Center, for travel to the Annual Meeting of the Southern Group on Educational Affairs
2013; Professional Development Grant ($250.00 in 2013 & 2014) from the Office of Faculty
2014 Development, Texas A&M Health Science Center, for completion of Levels I & II of the Modeling Excellence in Teaching Program
2015: Faculty Travel Award, Professional Development Grants from the Office of Faculty Development, 2016; Texas A&M University Health Science Center ($750 - $1500)
2017
2017 American Association of Anatomists Visiting Scholarship ($1000) for travel to the 2017 Harvard Medical School course, “Advanced Teaching Skills” (June 2-3, 2017)
   American Association of Anatomists Travel Grant ($350) for travel to the 2018 annual meeting of the American Association of Anatomists (April 21-25)

11. Research and Scholarly Activities
   Involvement in Medical Education Research: Selected presentations

My research focuses on measuring whether student learning and knowledge retention of basic sciences improve with the use of these instructional modalities. I evaluate the impact of innovative instructional approaches in histology on student academic performance, assess basic science enrichment in undergraduate medical education, and assess potential uses of mobile technology in medical education.

12. Publications in Medical Education
   A. Refereed Papers

   B. Abstract (*peer reviewed): Selected Presentations
   2. *Chico, D.E.; Hoesley, C.; Lewis, S.; Ryan, K. (2009). Integration of the Basic Sciences in the Clinical Years of Undergraduate Medical Education. Primary organizer and Moderator. GEA/GSA Small Group Discussion Session presented at the Annual Meeting of the Association of American Medical Colleges. Number of
attendees = 30.


13. Professional Service
   A. Manuscript Review – Journals Refereed
      2011- present  Medical Education Online, reviewer
      2013- present  Medical Science Educator, reviewer
      2015- present  MedEdPORTAL Publications, invited reviewer
   
   B. Professional and Scholarly Societies
      2015-present  American Association of Anatomists, regular member
      2013-present  International Association of Medical Science Educators, regular member 2006-present  Southern Group on Educational Affairs
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.)

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<tr>
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<th>DEGREE (if applicable)</th>
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<th>FIELD OF STUDY</th>
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<tbody>
<tr>
<td>University of Texas, Austin, Texas</td>
<td>B.A.</td>
<td>05/1998</td>
<td>Biology</td>
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<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>
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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 basall 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. GABA_A receptors as a target of early postnatal ethanol exposure. Much of my research career has focused on examining the GABA_A receptor as a target of ethanol. Ethanol is often viewed as a 'dirty drug' that impacts many targets and developmental processes. Since GABA_A 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 GABA_A 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&directio
n = 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, GABA\textsubscript{A}Rs 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

F32 Individual Postdoctoral Fellowship
NIH
Project Goal: Ethanol’s Effect on Basolateral Amygdala GABA Receptors (in Dr. McCool’s laboratory), but Chico
declined due to health problems and transitioning to postdoctoral position in Dr. Pearce’s laboratory.
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: 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>
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<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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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

1984-87 NIMH Postdoctoral Fellow (MH 09129), Department of Neurobiology and Anatomy, University of Rochester School of Medicine

1987-91 Faculty Scientist, Department of Neurobiology and Anatomy, University of Rochester School of Medicine

1991-94 Assistant Professor, Department of Neurobiology and Anatomy, University of Rochester School of Medicine

1991-94 Assistant Professor, Department of Human Anatomy and Medical Neurobiology, Texas A&M University Health Science Center, College of Medicine

2001-2005 Associate Professor, Department of Human Anatomy and Medical Neurobiology, Texas A&M University Health Science Center, College of Medicine
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 NIH: 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.


neurotrophic factor and its cognate receptor, TrkB, in the rat suprachiasmatic nucleus. Exp. Neurol.

c. Liang, F.-Q., Allen, G. and Earnest, D.J. (2000) Role of brain-derived neurotrophic factor in the


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 though 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.


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.

Chico
Completed (in the last 5 years):

#14GRNT18370013 Role: PI 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.
Sherecce Fields

Associate Professor

POSITIONS:
- Associate Professor, Texas A&M University, 2016 – present
- Assistant Professor, Texas A&M University, 2010 – 2016
- Visiting Researcher/Scholar, University of Texas, Austin, 2012 – 2013
- Postdoctoral Scientist/Pediatric Psychology Fellow, Nationwide Children's Hospital, 2008 – 2010

EDUCATION:
- PhD (Clinical Psychology) University of South Florida, 2008
- M.A. (Clinical Psychology) University of South Florida, 2004
- B.S. (Chemistry) Duke University, 1998

SELECT HONORS/AWARDS:
- Ray A. Rothrock '77 Fellow, College of Liberal Arts, Texas A&M University, 2016 – 2018
- SRNT Tobacco-Related Health Disparities Travel Award, 2014
- American Psychological Association Interdivisional Mentoring Award, 2012
- Texas A&M University ADVANCE Scholar, 2011-2013

GRANTS AS PI, co-PI, or co-I:
- “Integrating Biometric Responses to the Social Sciences,” Texas A&M University RDF grant (co-PI), 2016 – 2018. Total award $1,200,000.
- “Neural Basis of Habit Learning,” College of Liberal Arts SEED Grant (PI), 2012-2014. Total award $15,000.

SELECT PUBLICATIONS (15 most recent publications):


**SELECT PRESENTATIONS:**

**SIGNIFICANT TEACHING ACTIVITIES:**

Summer 2017 **Instructor** – (PSYC 304, PSYC 319) Psychology of Sport & History and Systems Department of Psychology Study Abroad Program in Bonn, Germany

Spring 2017 **Instructor** – (PSYC 407) Behavior Disorders in Children, Texas A&M University **Instructor** – (PSYC 637) Graduate Course in Clinical Intervention, Texas A&M University **Instructor** – (PSYC 613)

Graduate Practicum in Assessment, Texas A&M University

Fall 2016 **Instructor** – (PSYC 450) Clinical Psychology, Texas A&M University

**Graduate Student Supervision**

Sneha Wager (Fall 2010 – Summer 2016) – current postdoc at Brown University Medical Center

Krista Lange (Fall 2011 – Summer 2016) – current postdoc at Baylor College of Medicine

Ashley Ramos (Fall 2012 – Summer 2017) – current postdoc at Children’s National Medical Center

Michale Sferra (Fall 2012 – present)

Sara Dowd (Fall 2015 – present) – supported in part by Texas A&M University Vision 20/20 Fellowship

Timothy Regan (Fall 2016 – present)

Bethany Harris (Fall 2017 – present)

**Undergraduate Research Supervision**

Fall 2016 Research (PSY 485/491) – two students

Spring 2016 Research (PSY 485/491) – four students; one honors thesis mentored

Fall 2015 Research (PSY 485/491) – four students; one honors thesis mentored

Summer 2015 Research (PSY 485/491) – one student

Spring 2015 Research (PSY 485/491) – five students; two honors theses mentored

Fall 2014 Research (PSY 485/491) – four students

Summer 2014 Research (PSY 485/491) – one student

Spring 2014 Research (PSY 485/491) – six students; three honors theses mentored

Fall 2013 Research (PSY 485/491) – five students

Summer 2013 Research (PSY 485/491) – three students

Spring 2013 Research (PSY 485/491) – four students

Fall 2012 Research (PSY 485/491) – five students

Spring 2012 Research (PSY 485/491) – three students; one honors thesis mentored

Fall 2011 Research (PSY 485/491) – seven students

Summer 2011 Research (PSY 485/491) – three students

Spring 2011 Research (PSY 485/491) – two students

**MAJOR SERVICE ACTIVITIES:**

*National Service*

2016 – pres CPDD, Underrepresented Populations Committee

2012 – pres SRNT: Program Committee for Health Disparities Research Network
2011 – pres APA Div. 28: Committee on Minority Support in Psychopharm and Substance Abuse Research
2003 – 2005 American Psychological Association of Graduate Students
   Campus Representative for University of South Florida

Department Service
2017 – pres Curriculum Development Committee
2010 – 2014 Clinic Coordination Committee
2013 – 2014 Senior FMRI Search Committee Member 2013
   – 2014 Chair, Clinical Area Awards Committee
2014  Graduate Grant Summer Fellowship Committee
2014 – 2015 Clinical Area Representative, Graduate Studies Committee
2014-2015  Affective Science Search Committee Member
2014  TA training for Graduate Students

University Service
2016 – 2017 Reviewer, Honorary Degree Committee 2016-  
   pres  CIMPIR Tier One Program Research Mentor
2015  Reviewer, Aggies Commit
   Fellowship 2015 – pres Women’s Faculty Network,  
   Treasurer
2012 – 2015 Women’s Faculty Network, Steering Committee Member
2013  Faculty Ombudsman Search Committee
   Member
CURRICULUM VITAE
Tamy C. Frank-Cannon, DVM, PhD

PRESENT POSITION AND ADDRESS:
Title: Clinical Assistant Professor
Office: VICI 112
Phone: 979-862-1178
FAX: 979-847-8981
Email: tfrank-cannon@cvm.tamu.edu

EDUCATION:
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<th>Conferring Institution</th>
<th>Field</th>
<th>Year</th>
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<tbody>
<tr>
<td>PhD</td>
<td>Texas A&amp;M University</td>
<td>Veterinary Anatomy</td>
<td>2005</td>
</tr>
<tr>
<td></td>
<td>College Station, Texas</td>
<td>(neuroscience)</td>
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<tr>
<td>DVM</td>
<td>Texas A&amp;M University</td>
<td>Veterinary Medicine</td>
<td>1996</td>
</tr>
<tr>
<td></td>
<td>College Station, Texas</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BS</td>
<td>Texas A&amp;M University</td>
<td>Veterinary Science</td>
<td>1993</td>
</tr>
<tr>
<td></td>
<td>College Station, Texas</td>
<td></td>
<td></td>
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<tr>
<td>BS</td>
<td>Texas A&amp;M University</td>
<td>Zoology</td>
<td>1992</td>
</tr>
<tr>
<td></td>
<td>College Station, Texas</td>
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</table>

PROFESSIONAL EXPERIENCE AND ACADEMIC APPOINTMENTS:
2012 – present Texas A&M University College Station, TX
Clinical Assistant Professor
• Teach professional, graduate and undergraduate Gross Anatomy
• Teach professional and graduate Neuroanatomy
• Teach professional embryology/developmental anatomy
• Teach professional & clinical skills
• Director Gross Anatomy Teaching Lab (2009 – 2013)
• Director Gross Anatomy Simulation & Teaching Models Lab (2013 – present)

2008 – 2012 Texas A&M University College Station, TX
Lecturer
• Teach professional and undergraduate Gross anatomy
• Teach professional Neuroanatomy
• Director Gross Anatomy Teaching Lab (2009 – 2013)

2007 – 2008 UT Southwestern Medical Center Dallas, TX
Postdoctoral Researcher II
• Research Fellow – Neuroinflammation & Parkinson’s Disease

2005 – 2007 UT Southwestern Medical Center Dallas, TX
Postdoctoral Researcher I
• Research Fellow – Neuroinflammation & Parkinson’s Disease
2003 – 2005  Texas A&M University  
Postdoctoral Research Associate  
• Manage departmental tissue culture laboratory  
• Teach professional and undergraduate canine anatomy & professional neuroanatomy  

2002 – 2003  Texas A&M University  
Veterinary Clinical Associate  
• Teach professional and undergraduate canine anatomy & professional neuroanatomy  

2001 – 2002  Texas A&M University  
Graduate Teaching Assistant  
• Teach professional and undergraduate canine anatomy & professional neuroanatomy  

2000 – 2001  Texas A&M University  
Veterinary Clinical Associate  
• Teach professional and undergraduate canine gross anatomy  

1996 – 2000  Nolana Animal Hospital  
Associate Veterinarian  
• General practice, companion animal medicine & surgery  

1993 – 1995  Texas A&M University  
Gross Anatomy Tutor  
• Assist 1st year professional students in canine & large animal grossanatomy  

1993 – 1994  Texas A&M University  
Gross Anatomy Laboratory Assistant  
• Dissection projects & specimen preparation  
• Teaching assistant undergraduate canine gross anatomy  

**CLINICAL SPECIALTY/BOARD CERTIFICATION:**  
Accredited by the Texas Animal Health Commission, since 1996 - 2011  
Texas State Board of Veterinary Medical examiners, Active License #7944, since 1996  

**AWARDS AND HONORS:**  
Richard H. Davis Teaching Award, April 2011.  
Postdoctoral Training Certificate in Research, UT Southwestern Medical Center, Dallas, Texas, January 2008.

Society for Neuroscience, Postdoctoral Travel Award Fellow, November 2007.


American Association of Anatomist Student Travel Award, April 2006. Plum Endowed Scholarships in Veterinary Medicine, Summer & Fall 2005.

College of Veterinary Medicine Graduate Student Association Research Symposium, 3rd place Poster Presentation Award, April 26, 2005.

Fisher Institute Medical Research Award, April 15, 2005.

Texas A&M Faculty of Neuroscience, 2nd Place Graduate Student Poster Presentation Award, March 31, 2005.

Who’s Who Among Students in American Universities & Colleges, Texas A&M University, 2005.


Ethel Ashworth-Tsutsui Memorial Award for Research, November 9, 2004. Phi Zeta, Honor Society of Veterinary Medicine, inducted 2004.


Texas A&M Faculty of Neuroscience Travel Award, November 2003 & 2005.

American Association of Veterinary Anatomist, Graduate Student Competition, Oral Presentation, 1st Place, August 2, 2003.

Fisher Institute Medical Research Award, April 11, 2003.

Cajal Club, Nissl Body’s Graduate Student Poster Presentation Award, April 2002.

American Association of Anatomist, Graduate Student Travel Award, April 2002, & April 2005.

College of Veterinary Medicine Graduate Student Association Travel Award, April 2001, April 2002, April 2005.


TAMU Student Research Week, Graduate Student Research Award, First Place Biological Sciences, Division II, March 2001.


Bachelor of Science in Veterinary Science awarded Cum Laude, 1993. Bachelor of Science in Zoology awarded Cum Laude, 1992.


### COURSES:

#### Undergraduate:

<table>
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<th>Course Title</th>
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<th>Credit</th>
<th>% of Course</th>
<th>Formal Course</th>
<th>Contact Hr</th>
<th>Dates</th>
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<tbody>
<tr>
<td>VIBS 305 Biomedical Anatomy Assistant</td>
<td>TAMU</td>
<td>4</td>
<td>100%</td>
<td>2 lecture/wk</td>
<td>6 lab/wk</td>
<td>5/29 – 8/09/17</td>
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<tr>
<td>VIBS 305 Biomedical Anatomy Assistant</td>
<td>TAMU</td>
<td>4</td>
<td>100%</td>
<td>2 lecture/wk</td>
<td>6 lab/wk</td>
<td>6/2 – 8/11/15</td>
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<tr>
<td>VIBS 305 Biomedical Anatomy Assistant</td>
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<td>100%</td>
<td>2 lecture/wk</td>
<td>6 lab/wk</td>
<td>6/3 – 8/13/14</td>
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<tr>
<td>VIBS 305 Biomedical Anatomy Assistant</td>
<td>TAMU</td>
<td>4</td>
<td>100%</td>
<td>2 lecture/wk</td>
<td>6 lab/wk</td>
<td>6/3 – 8/14/13</td>
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<tr>
<td>VIBS 305 Biomedical Anatomy Coordinator</td>
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<td>2 lecture/wk</td>
<td>6 lab/wk</td>
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<td>VIBS 305 Biomedical Anatomy Assistant</td>
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<td>4</td>
<td>100%</td>
<td>2 lecture/wk</td>
<td>6 lab/wk</td>
<td>5/31 – 8/5/11</td>
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<td>VIBS 305 Biomedical Anatomy Assistant</td>
<td>TAMU</td>
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<td>100%</td>
<td>2 lecture/wk</td>
<td>6 lab/wk</td>
<td>6/1 – 8/6/10</td>
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<tr>
<td>VIBS 305 Biomedical Anatomy Coordinator</td>
<td>TAMU</td>
<td>4</td>
<td>100%</td>
<td>2 lecture/wk</td>
<td>6 lab/wk</td>
<td>6/2 – 8/11/09</td>
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### Professional:

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<tr>
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<th>Dates</th>
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<tbody>
<tr>
<td>VIBS 914 Professional &amp; Clinical Skills II Coordinator</td>
<td>TAMU</td>
<td>3</td>
<td>100%</td>
<td>1 lecture/wk</td>
<td>1/8 – 5/4/17</td>
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<tr>
<td>VTPPP 914 Professional &amp; Clinical Skills I Assistant</td>
<td>TAMU</td>
<td>3</td>
<td>75%</td>
<td>1 lecture/wk</td>
<td>8/21 – 12/08/17</td>
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<tr>
<td>VIBS 910 Animal Anatomy Assistant</td>
<td>TAMU</td>
<td>4</td>
<td>50%</td>
<td>2 lecture/wk</td>
<td>8/21 – 12/08/17</td>
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<tr>
<td>VIBS 913 Microscopic anatomy II – embryology Coordinator</td>
<td>TAMU</td>
<td>4</td>
<td>100%</td>
<td>1 lecture/wk</td>
<td>1/9– 5/5/017</td>
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<tr>
<td>VIBS 913 Microscopic anatomy II – neuroanatomy Assistant</td>
<td>TAMU</td>
<td>4</td>
<td>100%</td>
<td>1.5 lecture/wk</td>
<td>1/9– 5/5/017</td>
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<tr>
<td>VMID 913 Clinical correlates II Assistant</td>
<td>TAMU</td>
<td>4</td>
<td>-</td>
<td>8 lab/semester</td>
<td>1/9– 5/5/017</td>
</tr>
<tr>
<td>VIBS 913 Microscopic anatomy II – embryology Coordinator</td>
<td>TAMU</td>
<td>4</td>
<td>100%</td>
<td>1 lecture/wk</td>
<td>1/9– 5/5/016</td>
</tr>
<tr>
<td>VIBS 913 Microscopic anatomy II – neuroanatomy Assistant</td>
<td>TAMU</td>
<td>4</td>
<td>100%</td>
<td>1.5 lecture/wk</td>
<td>1/9– 5/5/016</td>
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<tr>
<td>VMID 913 Clinical correlates II Assistant (IV catheterization, suture, cystocentesis, feline neuter models; labs)</td>
<td>TAMU</td>
<td>4</td>
<td>-</td>
<td>8 lab/semester</td>
<td>1/9– 5/5/016</td>
</tr>
<tr>
<td>VMID 912 Clinical correlates I Assistant &amp; instruction (instrument handling, suturing, venipuncture, IV catheterization)</td>
<td>TAMU</td>
<td>4</td>
<td>-</td>
<td>8 lab/semester</td>
<td>8/22 – 12/09/16</td>
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<tr>
<td>VIBS 910 Veterinary gross anatomy I Assistant (assist in labs, attend lectures, live animal exams, proof &amp; grade exams)</td>
<td>TAMU</td>
<td>4</td>
<td>50%</td>
<td>2 lecture/wk</td>
<td>8/22 – 12/09/16</td>
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### Graduate:

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<tr>
<th>Course Title</th>
<th>Institution</th>
<th>Credit</th>
<th>% of Course</th>
<th>Formal Contact</th>
<th>Dates</th>
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</thead>
<tbody>
<tr>
<td>VIBS 601 Anatomy</td>
<td>TAMU</td>
<td>4</td>
<td>100%</td>
<td>2 lecture/wk</td>
<td>5/29 – 8/09/17</td>
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</table>
Coordinator (graduate specific lectures, all labs, exams, grading, and course management)

VIBS 603 TAMU 4 100% 2 lecture/wk 6 lab/wk 8/29 – 12/14/16
Cross listed with NRSC 603
Neuroanatomy

VIBS 685 TAMU 4 100% 2hr lecture/wk 8/31 – 12/16/15
Directed studies in Biomedical Anatomy
6hr lab/wk

Coordinator (lectures, labs, grading, and course management)

VIBS 603 TAMU 4 100% 2 lecture/wk 6 lab/wk 8/26 – 12/6/15
Cross listed with NRSC 603
Neuroanatomy

VIBS 601 TAMU 4 100% 2 lecture/wk 8 lab/wk 6/2 – 8/11/15
Anatomy
Coordinator (all lectures, labs, exams, grading, and course management)

VIBS 601 TAMU 4 100% 2 lecture/wk 8 lab/wk 6/3 – 8/14/14
Anatomy
Coordinator (all lectures, labs, exams, grading, and course management)

VIBS 603 TAMU 4 100% 2 lecture/wk 6 lab/wk 8/26 – 12/6/13
Cross listed with NRSC 603
Neuroanatomy
Coordinator (all lectures, labs, exams, grading, and course management)

VIBS 601 TAMU 4 100% 2 lecture/wk 8 lab/wk 6/3 – 8/14/13
Anatomy
Coordinator (all lectures, labs, exams, grading, and course management)

VIBS 603 TAMU 4 100% 4 lecture/wk 4 lab/wk 8/27 – 12/11/12
Cross listed with NRSC 603
Neuroanatomy
Co-coordinate lectures and labs held in conjunction with VIBS 450H, Coordinate graduate level lectures & labs.

VIBS 601 TAMU 4 100% 2 lecture/wk 8 lab/wk 5/29 – 8/8/12
Anatomy
Coordinator (all lectures, labs, exams, grading, and course management)

VIBS 603 TAMU 4 100% 4 lecture/wk 4 lab/wk 8/29 – 12/19/11
Cross listed with NRSC 603
Neuroanatomy
Co-coordinate lectures and labs held in conjunction with VIBS 450H, Coordinate graduate level lectures & labs.
Directed Studies: Biomedical Anatomy
Directed working with VIBS 305 lectures and lab dissection.

Cross listed with NRSC 689
Special Topics: Veterinary Ruminant Anatomy – guided discussions based lectures and coordinated lab dissection.

Co-ordinate lectures and labs held in conjunction with VIBS 450H, Coordinate graduate level lectures & labs.

STUDENTS:

Graduate 685 & Undergraduate 485 Students:

<table>
<thead>
<tr>
<th>Name</th>
<th>Degree</th>
<th>Major</th>
<th>Credit Hours</th>
<th>Semester</th>
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<tbody>
<tr>
<td>Nathan Earl</td>
<td>NTO-MS</td>
<td>BIMS</td>
<td>2</td>
<td>Summer II 2018</td>
</tr>
<tr>
<td>Madison Buchanan</td>
<td>BS</td>
<td>BIMS</td>
<td>2</td>
<td>Spring 2018</td>
</tr>
<tr>
<td>Amber Heard</td>
<td>BS</td>
<td>BIMS</td>
<td>3</td>
<td>Spring 2017</td>
</tr>
<tr>
<td>Amber Heard</td>
<td>BS</td>
<td>BIMS</td>
<td>3</td>
<td>Fall 2016</td>
</tr>
<tr>
<td>Austin Perryman</td>
<td>BS</td>
<td>BIMS</td>
<td>3</td>
<td>Spring 2016</td>
</tr>
<tr>
<td>Megan Davis</td>
<td>NTO-MS</td>
<td>BIMS</td>
<td>4</td>
<td>Fall 2015</td>
</tr>
<tr>
<td>Jennifer Garrett</td>
<td>NTO-MS</td>
<td>BIMS</td>
<td>4</td>
<td>Fall 2015</td>
</tr>
<tr>
<td>Jessica Mendonca-Gunn</td>
<td>NTO-MS</td>
<td>BIMS</td>
<td>4</td>
<td>Fall 2015</td>
</tr>
<tr>
<td>Tony Zhang</td>
<td>NTO-MS</td>
<td>BIMS</td>
<td>4</td>
<td>Summer 2015</td>
</tr>
<tr>
<td>Cameron Jacobs</td>
<td>NTO-MS</td>
<td>BIMS</td>
<td>4</td>
<td>Summer 2014</td>
</tr>
<tr>
<td>Gina Jamal</td>
<td>NTO-MS</td>
<td>BIMS</td>
<td>1</td>
<td>Spring 2014</td>
</tr>
<tr>
<td>Sonny Aguilar</td>
<td>NTO-MS</td>
<td>BIMS</td>
<td>3</td>
<td>Fall 2013</td>
</tr>
<tr>
<td>Sarah Davidson</td>
<td>BS</td>
<td>BIMS</td>
<td>3</td>
<td>Fall 2013</td>
</tr>
<tr>
<td>Gina Jamal</td>
<td>NTO-MS</td>
<td>BIMS</td>
<td>2</td>
<td>Fall 2013</td>
</tr>
<tr>
<td>Erica Malone</td>
<td>BS</td>
<td>BIMS</td>
<td>3</td>
<td>Fall 2013</td>
</tr>
<tr>
<td>Sarah Davidson</td>
<td>BS</td>
<td>BIMS</td>
<td>3</td>
<td>Summer 2013</td>
</tr>
<tr>
<td>Reid Alley</td>
<td>BS</td>
<td>BIMS</td>
<td>2</td>
<td>Spring 2013</td>
</tr>
<tr>
<td>Rosa Banuelos</td>
<td>BS</td>
<td>BIMS</td>
<td>1</td>
<td>Spring 2013</td>
</tr>
</tbody>
</table>
Sierra Dodson  
Kayla Matschek  
Krystal Walker  
Alejandro Barbosa  
Gary Chang  
Katherine Ketchum  
Adarsh Pakanti  
Stephanie Sullivan  
Carolyn Jacobellis  
Meredith Lynch  
Carlos Dominguez  
Cameron Lancarte  
Elyzabeth Peterson  
Stephanie Sullivan  
Kyllie Ryan-Hummel

**Graduate Students:**

<table>
<thead>
<tr>
<th>Name</th>
<th>Degree</th>
<th>Institution</th>
<th>Advisor or Committee Member</th>
<th>Dates</th>
</tr>
</thead>
<tbody>
<tr>
<td>David Andrews (NTO-MS)</td>
<td>TAMU</td>
<td>Co-Chair</td>
<td></td>
<td>2011-2012</td>
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<tr>
<td></td>
<td>Graduated DVM Texas A&amp;M University May 2017</td>
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<tr>
<td>Heather Johnson (NTO-MS)</td>
<td>TAMU</td>
<td>Chair</td>
<td></td>
<td>2012 – 2013</td>
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<tr>
<td></td>
<td>Graduated December 2013</td>
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</tr>
<tr>
<td>Laura Surratt (NTO-MS)</td>
<td>TAMU</td>
<td>Chair</td>
<td></td>
<td>2012 – 2014</td>
</tr>
<tr>
<td></td>
<td>Graduated May 2014</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Josh Hathcock (NTO-MS)</td>
<td>TAMU</td>
<td>Member</td>
<td></td>
<td>2012 – 2013</td>
</tr>
<tr>
<td></td>
<td>Graduated December 2013</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Jasmin Bui (NTO-MS)</td>
<td>TAMU</td>
<td>Chair</td>
<td></td>
<td>2013 – 2016</td>
</tr>
<tr>
<td></td>
<td>Graduated Spring 2016, working as medical scribe</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cameron Jacobs (NTO-MS)</td>
<td>TAMU</td>
<td>Chair</td>
<td></td>
<td>2013 – 2014</td>
</tr>
<tr>
<td></td>
<td>Graduated August 2014, Admitted to DVM program Oklahoma State University Fall 2014</td>
<td></td>
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<tr>
<td>Sarah Wiggins (NTO-MS)</td>
<td>TAMU</td>
<td>Chair</td>
<td></td>
<td>2013 – 2014</td>
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<tr>
<td></td>
<td>Admitted to UT Southwestern Physician Assistant School Jan. 2014, leave of absence NTO-MS program as of spring 2014.</td>
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<tr>
<td>Gina Jamal (NTO-MS)</td>
<td>TAMU</td>
<td>Chair</td>
<td></td>
<td>2013 – 2014</td>
</tr>
<tr>
<td></td>
<td>Graduated August 2014, Medical transcriptionist, Attended UT San Antonio Medical School</td>
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<tr>
<td>Arturo Pinon-Velasquez (NTO-MS)</td>
<td>TAMU</td>
<td>Chair</td>
<td></td>
<td>2013 – 2015</td>
</tr>
<tr>
<td></td>
<td>Graduated May 2015, attending TAMU, DVM program beginning Fall 2017</td>
<td></td>
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</tr>
<tr>
<td>Name</td>
<td>Degree</td>
<td>Institution</td>
<td>Committee Member</td>
<td>Dates</td>
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<tr>
<td>Brandon Poskevich (NTO-MS)</td>
<td>NTO-MS</td>
<td>TAMU</td>
<td>Chair</td>
<td>2014 – 2015</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Admitted to medical school for Fall 2015, withdrawn from NTO-MS program June 2015</td>
</tr>
<tr>
<td>Rafa Nizam (NTO-MS)</td>
<td>NTO-MS</td>
<td>TAMU</td>
<td>Member</td>
<td>2014 – 2015</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Graduated Summer 2015, Applying to dental school for fall 2017 admission</td>
</tr>
<tr>
<td>Tony Zhang (NTO-MS)</td>
<td>NTO-MS</td>
<td>TAMU</td>
<td>Member</td>
<td>2014 – 2015</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Graduated Summer 2015. Interviewed, dental school</td>
</tr>
<tr>
<td>Maisie Llewellyn (NTO-MS)</td>
<td>NTO-MS</td>
<td>TAMU</td>
<td>Member</td>
<td>2014 – 2015</td>
</tr>
<tr>
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<td></td>
<td></td>
<td></td>
<td>Graduated Fall 2015, attending TAMU, DVM program beginning Fall 2017</td>
</tr>
<tr>
<td>Jennifer Garrett (NTO-MS)</td>
<td>NTO-MS</td>
<td>TAMU</td>
<td>Chair</td>
<td>2014 – 2016</td>
</tr>
<tr>
<td></td>
<td></td>
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<td></td>
<td>Graduated Spring 2016</td>
</tr>
<tr>
<td>Megan Davis (NTO-MS)</td>
<td>NTO-MS</td>
<td>TAMU</td>
<td>Chair</td>
<td>2014 – 2016</td>
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<td>Graduate Spring 2016</td>
</tr>
<tr>
<td>Robert McQuitty (NTO-MS)</td>
<td>NTO-MS</td>
<td>TAMU</td>
<td>Member</td>
<td>2015 – 2016</td>
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<td>Graduated Fall 2016</td>
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<tr>
<td>Shelby Cruz (NTO-MS)</td>
<td>NTO-MS</td>
<td>TAMU</td>
<td>Member</td>
<td>2015 – 2017</td>
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<td></td>
<td></td>
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<td></td>
<td>Graduated Spring 2017</td>
</tr>
<tr>
<td>Landry Tucker (NTO-MS)</td>
<td>NTO-MS</td>
<td>TAMU</td>
<td>Chair</td>
<td>2016 – 2017</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Graduated Spring 2018, attending Physician Assistant school beginning Fall 2018</td>
</tr>
<tr>
<td>Edelicia Garza (NTO-MS)</td>
<td>NTO-MS</td>
<td>TAMU</td>
<td>Member</td>
<td>2017 – 2018</td>
</tr>
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<td></td>
<td></td>
<td>Graduated Spring 2018, working for bovine genetics and breeding company.</td>
</tr>
<tr>
<td>Brittany Crawford (NTO-MS)</td>
<td>NTO-MS</td>
<td>TAMU</td>
<td>Member</td>
<td>2017 – present</td>
</tr>
<tr>
<td>Sarah Koch (NTO-MS)</td>
<td>NTO-MS</td>
<td>TAMU</td>
<td>Chair</td>
<td>2017 – present</td>
</tr>
</tbody>
</table>

Residents/Interns/Postdoctoral Fellows:

<table>
<thead>
<tr>
<th>Name</th>
<th>Degree</th>
<th>Institution</th>
<th>Committee Member</th>
<th>Dates</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lauren LeBlanc, DVM</td>
<td>DVM</td>
<td>TAMU</td>
<td>Advisor</td>
<td>6/2012-4/2013</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Accepted Clinical Practice Position (companion animal &amp; avian) April 2013</td>
</tr>
</tbody>
</table>

**TEACHING PROGRAMS:**

**Anatomy Workshop 2011 & 2014:** coordinated and offered an intensive 6 week workshop in gross anatomy for incoming professional students. The workshop consists of 2 lectures a week and 5 labs a week covering all the major body systems including written and laboratory exams. In 2014 introduced the use of ExamSoft for anatomy written & laboratory exams.

**Youth Adventure Program: Veterinary Medicine 2010, 2013, 2015, 2016 & 2018:** Coordinated and supervised a week-long summer camp for Junior High and High School students interested in Veterinary Medicine. Program consist for seminars in a variety of veterinary fields include species specific medicine and various topics such as what it takes to get into vet school.
BIMS Summer Academy – Introduction to Biomedical Anatomy: Developed and offered a one week workshop to introduce 2+2 transfer students to the BIMS program to the subject of Biomedical Anatomy. Offered in August 2011 and May 2012, it included lecture-type discussions and laboratory dissections to give the student an overview of the volume of information covered and Biomedical Anatomy as well as experience in the some of the detail anatomy of structures.

RESEARCH/SCHOLARLY ACTIVITIES:


Development of equine external jugular vein model for intravenous blood collection, drug injection and catheter placement. Began instructional use in VMID 912 & 913 clinical correlates labs Fall 2013 and use has continued in Professional & Clinical Skills Courses (VTPP 914, VIBS 914, VSCS 914). Used in 4H Vet Science camp, YAP camp and Veterinary Technician continuing education training course.

Development of a skin and subcutaneous suture model. Began use in VMID 912 clinical correlates labs Spring 2013 and VMID 935 Surgery I labs Fall 2013. Use has continued in VSCS 914 and models are available to student in the course to purchase for practicing skills at home.


Development of subcutaneous injections models and suture models for instructional use in VMID 912 & 913 clinical correlates lab and continued use in VTPP 914.

Development of cystocentesis model & feline neuter model used in VMID 912 Clinical Correlates lab. Cytocentesis model continues to be used in VIBS 914.

Development of pericardiocentesis model used Fall of 2015 in 3VM cardiology medicine course lab (VSCS 954) and use at DVM continuing education wetlab courses.

Refinement of a published fundic exam model. Improved retinal appearance, support of lens and support of globe within canine model head. Used in VIBS 914 course for instruction and exams.

Development of skin punch biopsy model used in VIBS 914 course and a skin excision biopsy model used in VSCS 914.

Development of a caudal epidural model and tail vein for cattle.

Development of an articulated stifle joint model used in VIBS 305 by multiple instructors.

BIBLIOGRAPHY:

Publications in Refereed Journals:


Abstracts (last five years):


Frank-Cannon, Tamy C. Simulators in 1st year veterinary student clinical skills course. Poster presentation & Blitz oral presentation 2015 InVeST (International Veterinary Simulation in Teaching) Conference, Germany.


Frank-Cannon, Tamy C. Simulators in 1st year veterinary student clinical skills course. Poster presentation & Blitz oral presentation 2015 InVeST (International Veterinary Simulation in Teaching) Conference, Germany.

**Frank-Cannon, Tamy C.**, Blue, Alice. Intravenous injection and catheter placement teaching models. Poster presentation American Association of Veterinary Medical College 2014 Annual meeting.

**Non-Refereed Publications:** (optional)

*VIBS 305 Osteology CD. Tamy C. Frank-Cannon* and Michelle Pine. An in-house computer teaching aid for VIBS 305: Biomedical Anatomy printed and sold by CVM Creative Technologies.


**PATENTS AND RELATED DISCOVERY ACTIVITIES:**

Texas A&M University press release on Cerebellar Purkinje cell death in the α1A voltage- gated calcium channel mutant mouse, leaner (PhD dissertation). November 1, 2005

**SERVICE ACTIVITIES:**
**Clinical Service:**
Care & oversight of use of the animals procured through the Educational Memorial Program.

**Professional Organizations and Service:**
TAMU Faculty Senate, College of Veterinary Medicine & Biomedical sciences representative, 2014 to present.
CVM caucus leader 2018 - 2019


Texas A&M University, College of Veterinary Medicine, Graduate Student Association – member, 2000 to 2005; president, 2003 – 2004.


Phi Zeta Eta Chapter (TAMU) – member, 2004 to present, president elect 2016-17, president 2017-18

American Association of Veterinary Anatomists – member, 2001 to present.
Manuscript Review of Journals:
Veterinary Radiology & Ultrasound Impact factor 0.985
Manuscripts (1)
Journal of Neuroimmunology Impact factor 3.159
Manuscripts (1)
Neurotoxicology
Impact factor 1.282
Manuscripts (6)
Antioxidants & Redox Signaling: Impact factor 5.484
Reviewing editor, comprehensive review (1)
Anatotan, Histologia, Embryologia Impact factor 0.672
Manuscript reviews (2)
Neuroscience letters
Impact factor Manuscript reviews (1)
Neurobiology of aging
Impact factor Manuscript reviews (1)
Brain Research
Impact factor Manuscript reviews (3)
Oncotarget
Impact factor Manuscript reviews (1)

OTHER SCHOLARLY ACTIVITY:

Conference Presentations
October 20, 2017: Emergency & Critical Care Conference Pericardiocentesis Wetlab with Dr. Ashley Sauders.
  • CE wetlab

April 18-30, 2017: Veterinary Innovation Summit
Pericardiocentesis – The future of simulated learning & veterinary training.
  • oral presentation

September 13 – 16, 2015: InVeST (International Veteirnary Simulation in Teaching). Simulators in 1st year veterinary student clinical skills course.
  • Poster presentation & short oral presentation

February 27 – March 4, 2014: InVeST (International Veterinary Simulation in Teaching)
Intravenous Injection and catheter placement teaching models.
  • Poster presentation

March 13 – 15, 2014: AAVMC (American Association of Veterinary Medical Colleges) Annual Meeting
Intravenous Injection and catheter placement teaching models.
  • Poster presentation

Invited Presentations
Aggies Invent (April 7-9, 2017) – invited content expert in veterinary model making, veterinary medicine & education.
  • on site and available to the students participating in challenge to ask questions and get advice.

Four degrees & six letters – Done...So, Now What?
BIOGRAPHICAL SKETCH

NAME Cédric G. Geoffroy

POSITION TITLE: Assistant Professor (tenure-track)

<table>
<thead>
<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE</th>
<th>Completion Date</th>
<th>FIELD OF STUDY</th>
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<tbody>
<tr>
<td>University of Sciences of Montpellier, France</td>
<td>BS</td>
<td>06/2002</td>
<td>Cellular biology and Animal physiology</td>
</tr>
<tr>
<td>College of Engineering Applied Biology and Microbiology at Luminy, Marseille, France</td>
<td>Engineer</td>
<td>07/2004</td>
<td>Biotechnology and Healthcare Industries</td>
</tr>
<tr>
<td>University of Cambridge, UK</td>
<td>(MS)</td>
<td>07/2008</td>
<td>Neuroscience</td>
</tr>
<tr>
<td>University of California San Diego, La Jolla, CA</td>
<td>Postdoc</td>
<td>04/2013</td>
<td>Neuroscience</td>
</tr>
</tbody>
</table>

A. Personal Statement

For over 14 years, I have been studying the mechanisms of axon growth and repair after CNS injury. My background in molecular neurobiology and experience in CNS injury and repair research make me well suited to carry the work proposed in this application.

I obtained my PhD with Dr. Raineteau at the Cambridge Centre for Brain Repair where I developed methods for fate mapping of spinal cord stem cells and to promote neurogenesis with engineered transcription factors. This work provided me with a strong background in molecular biology, genetics and cellular biology. With the motivation to better understand the regenerative process in the spinal cord, I joined Dr. Zheng's laboratory at UC San Diego, where I determined the roles of neuron-intrinsic and extrinsic molecules in axon growth and functional recovery after spinal cord injury. I was the second author of a highly cited study demonstrating that genetically targeting one or multiple myelin-associated axon growth inhibitors (Nogo, MAG, OMgp) does not significantly enhance the regeneration of injured axons but alters the sprouting of uninjured axons (Lee, Geoffroy et al., Neuron, 2010). Next, I discovered that the neuron intrinsic growth state determines neuronal responsiveness to changes in the extrinsic environment (Geoffroy, et al., J Neurosci, 2015). More recently, I discovered an age-dependent decline in corticospinal axon regeneration in PTEN deleted mice (Geoffroy, et al., Cell Rep, 2016). Age is an important consideration in promoting axonal repair after CNS injuries.

B. Positions and Honors

Positions and Employment:
2014 - 2017 Assistant Project Scientist, Department of Neurosciences, UC San Diego
2017- present Assistant Professor, Texas A&M, Dept. of Neurosciences and Experimental Therapeutics Honors and Awards:
2004 Leonardo Da Vinci European grant
2006, 2007 Brain journal travel award
2006, 2007 Homerton College (University of Cambridge) research funds
2007 IBRO travel award (International Brain Research Organization)
2013 Wings for Life grant (with Dr. Zheng, key personnel – 2013-2015)

Other Experience and Professional Memberships:
2005- present Ad Hoc Reviewer for journals including Stem Cells, Journal of Neuroscience, E-life...
2007- present Member, Society for Neuroscience
2006 Invited speaker, NeuroNE, Marseille, France.
2006 Invited speaker, Brain Repair Centre Away Day, Cambridge Neuroscience, UK.
c. Contributions to Science

1) Engineering transcriptional factors for fate tracking of neural precursor cells in vitro and in vivo. I demonstrated that transient overexpression of two transcription factors in spinal cord-derived neural precursor cells leads to the generation of different neuronal populations and that their post-transcriptional inhibition was due to the sequestration of E-proteins by gliogenic transcription factors. I also developed ways to permanently label spinal cord stem cells for long-term fate tracking in vivo after a transient transfection, enabling the tracking of the transfected cells both in vitro and in vivo.


2) Understanding the role of myelin-associated axon growth inhibitors in CNS repair. I was the second author on a key paper from the Zheng lab assessing the role of myelin-associated axon growth inhibitors on axon regeneration and sprouting after injury. This study redefined “myelin inhibitors” as myelin-associated axon sprouting modulators and illustrates the critical need to understand the in vivo role of molecular targets before effective therapies can be developed.


3) Understanding the interaction between neuron intrinsic and extrinsic control of CNS axon repair. Following the discovery of PTEN/mTOR, an important question was how neuron-intrinsic and extrinsic pathways interact to regulate axonal repair. This study provided proof-of-principle evidence that neuronal growth state determines its responsiveness to changes in extrinsic influence and that combined manipulation of both intrinsic and extrinsic factors may further improve axon regeneration.


4) Finding new genes involved in CNS axon regeneration and repair. First, I initiated the study on the mammalian homologues of invertebrate DLK (dual leucine zipper-bearing kinase) and LZK in CNS axon growth and demonstrated that they both participate to axon growth control. Second, I was instrumental in the findings of the role of LZK as a master regulator of astrogliosis after spinal cord injury.

c)

5) Impact of age on axon growth

I made the surprising discovery that while Pten deletion in mid-aged to aging mice remains effective in preventing axotomy-induced decline in neuron-intrinsic growth state, axonal regeneration distal to injury is greatly diminished in older mice. This study was the first clear demonstration for an age-dependent decline in axon regeneration after injury in the mammalian CNS.


c) My NCBI link to all my publications in My Bibliography:
**D. Additional Information: Research Support and/or Scholastic Performance**

**Ongoing research support**

<table>
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<tr>
<th>Foundation</th>
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<tbody>
<tr>
<td>TIRR Foundation</td>
<td>Geoffroy (PI), Watkins (Co-PI)</td>
<td>03/01/2018-02/28/20</td>
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**Completed research support**

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<th>Foundation</th>
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<th>Start Date – End Date</th>
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<tbody>
<tr>
<td>Synthego Synthetic</td>
<td>Geoffroy (PI)</td>
<td>1/1/17– 03/30/2018</td>
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<tr>
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<td></td>
<td>“Non-viral CRISPR-mediated somatic gene targeting in vivo”</td>
</tr>
<tr>
<td>UCSD-HAI</td>
<td>Kisseleva (PI), Geoffroy (co-PI)</td>
<td>10/1/15 – 9/30/17</td>
</tr>
<tr>
<td></td>
<td></td>
<td>“IL-17 Signaling Regulates Age-Related Changes of Liver-Brain Axis”</td>
</tr>
<tr>
<td>Keck School of Medicine of USC</td>
<td>Xu (PI), Geoffroy (co-PI)</td>
<td>1/1/16 – 12/30/17</td>
</tr>
<tr>
<td></td>
<td></td>
<td>“IL-17 Signaling Regulates Alcohol-induced Damage of Liver-Brain Axis”</td>
</tr>
<tr>
<td>384971- C. Neilsen Foundation</td>
<td>Zheng (PI), Geoffroy (Co-I)</td>
<td>8/31/16 – 8/30/19</td>
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<tr>
<td></td>
<td></td>
<td>“The impact of aging on axon regeneration after spinal cord injury”</td>
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<tr>
<td>Thermo Fisher Collaborative Award</td>
<td>Geoffroy (PI)</td>
<td>12/1/15 – 9/30/16</td>
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<tr>
<td></td>
<td></td>
<td>“Developing non-viral based, CRISPR-mediated somatic gene targeting in vivo”</td>
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</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: 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>
<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>University of New England, Armidale, Australia</td>
<td>Ph.D.</td>
<td>1998</td>
<td>Physiology</td>
</tr>
<tr>
<td>University of Memphis, Memphis, TN</td>
<td>Post-doctoral</td>
<td>1997-1999</td>
<td>Psychology</td>
</tr>
<tr>
<td>UTMDACC, Bastrop, Texas</td>
<td>Post-doctoral</td>
<td>1999-2000</td>
<td>Psychology</td>
</tr>
<tr>
<td>Texas A&amp;M University</td>
<td>Post-doctoral</td>
<td>2002-2004</td>
<td>Psychology</td>
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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.

recovery following spinal cord injury. Front Neurol. 5:44. PMC3988397

B. POSITIONS AND HONORS

<table>
<thead>
<tr>
<th>Year</th>
<th>Position</th>
<th>Institution</th>
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<tbody>
<tr>
<td>1999</td>
<td>Part-time Assistant Professor.</td>
<td>Southwestern University.</td>
</tr>
<tr>
<td>2001</td>
<td>Part-time Assistant Professor.</td>
<td>Southwestern University.</td>
</tr>
<tr>
<td>2001-2002</td>
<td>Project Coordinator.</td>
<td>Georgia State University</td>
</tr>
<tr>
<td>2004-2008</td>
<td>Assistant Research Scientist</td>
<td>Texas A&amp;M University</td>
</tr>
<tr>
<td>2008-2011</td>
<td>Assistant Research Professor</td>
<td>Texas A&amp;M University</td>
</tr>
<tr>
<td>2011-2013</td>
<td>Associate Research Professor</td>
<td>Texas A&amp;M University</td>
</tr>
<tr>
<td>2014-present</td>
<td>Assistant Professor</td>
<td>Texas A&amp;M University</td>
</tr>
</tbody>
</table>

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:

- Society for Neuroscience
- International Behavioral Neuroscience Society
- National Neurotrauma Society
- International Association for the Study of Pain

C. CONTRIBUTION TO SCIENCE

1. **SCI increases symptoms of depression in a rodent model.** The incidence of depression is significantly increased in spinaly 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.


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

RO1 NS41548-06  Role: Col  Dates Funded: 2/1/07-1/1/12

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.

R01 HD058412  Role: Col (PI: Grau)  Dates Funded: 8/1/07-7/31/12
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.

Mission Connect  Role: Col (PI: Grau)  Dates funded: 10/1/12-9/30/14
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: Col (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. Follow this format for each person. DO NOT EXCEED FIVE PAGES.

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.)

<table>
<thead>
<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>Lamar University, Beaumont, TX</td>
<td>BS</td>
<td>05/1973</td>
<td>Biology</td>
</tr>
<tr>
<td>Lamar University, Beaumont, TX</td>
<td>MS</td>
<td>08/1975</td>
<td>Biology</td>
</tr>
<tr>
<td>University of Texas Medical Branch, Galveston, TX</td>
<td>Ph.D.</td>
<td>12/1980</td>
<td>Pharmacology/Neurosci</td>
</tr>
<tr>
<td>School of Pharmacy, University of London</td>
<td>Postdoctoral</td>
<td>12/80-10-82</td>
<td>Pharmacology/Neurosci</td>
</tr>
<tr>
<td>Baylor College of Medicine, Houston, TX</td>
<td>Postdoctoral</td>
<td>11/82-12-83</td>
<td>Neuroscience</td>
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</table>

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 learned 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+]), 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.

3. Murchison, D. and Griffith, W.H. Age-related alterations in caffeine-sensitive calcium stores and

Chico

B. Positions and Honors

<table>
<thead>
<tr>
<th>Date</th>
<th>Position</th>
<th>Institution</th>
</tr>
</thead>
<tbody>
<tr>
<td>12/1980-10/1982</td>
<td>Research Associate, Department of Pharmacology,</td>
<td>School of Pharmacy, University of London, Professor David A. Brown, Advisor</td>
</tr>
<tr>
<td>11/1982-12/1983</td>
<td>Postdoctoral Fellow, Department of Neurology, Section Neurophysiology, Baylor College of Medicine,</td>
<td>Dr. Daniel Johnston, Advisor</td>
</tr>
<tr>
<td>01/1984-08/1989</td>
<td>Assistant Professor, Department of Medical Pharmacology &amp; Toxicology</td>
<td>College of Medicine, Texas A&amp;M University</td>
</tr>
<tr>
<td>09/1089-08/1994</td>
<td>Associate Professor, Department of Medical Pharmacology &amp; Toxicology</td>
<td>College of Medicine, Texas A&amp;M University</td>
</tr>
<tr>
<td>09/94-12/20505</td>
<td>Professor, Department of Medical Pharmacology and Toxicology, College of Medicine, Texas A&amp;M</td>
<td>University Health Science Center</td>
</tr>
<tr>
<td>01/2006-present</td>
<td>Professor, Department of Neuroscience and Experimental Therapeutics</td>
<td>College of Medicine, Texas A&amp;M University Health Science Center</td>
</tr>
<tr>
<td>06/2006-present</td>
<td>Chair, Department of Neuroscience and Experimental Therapeutics</td>
<td>College of Medicine, Texas A&amp;M University Health Science Center</td>
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Honors and Professional Service

<table>
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<tr>
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<tbody>
<tr>
<td>1980</td>
<td>Pharmacology Research Award, National Student Research Forum</td>
</tr>
<tr>
<td>1980</td>
<td>Overall Excellence of Research Award, National Student Research Forum</td>
</tr>
<tr>
<td>1980</td>
<td>James E. Beall, II Memorial Award for Research in the Neurosciences</td>
</tr>
<tr>
<td>1981</td>
<td>Academic Excellence Award, Graduate School Biomedical Sciences at Galveston</td>
</tr>
<tr>
<td>1984-87</td>
<td>Councilor for the Texas A&amp;M Chapter of the Society for Neuroscience</td>
</tr>
<tr>
<td>1988</td>
<td>President, Texas A&amp;M Chapter of the Society for Neuroscience</td>
</tr>
<tr>
<td>1990-present</td>
<td>ad hoc reviewer, National Science Foundation</td>
</tr>
<tr>
<td>1991-2004</td>
<td>member, Oklahoma Center for the Advancement of Science and Technology (OCAST)</td>
</tr>
<tr>
<td>1993-present</td>
<td>ad hoc reviewer, National Institutes of Health</td>
</tr>
<tr>
<td>1994, 1998</td>
<td>NIH Intramural Review, Lab of Molecular and Cellular Neurobiology, NIAAA</td>
</tr>
<tr>
<td>1997</td>
<td>Editorial Board, American Journal of Physiology, Heart and Circulatory Physiology</td>
</tr>
<tr>
<td>1999</td>
<td>Chairman of Study Section Panels, OCAST</td>
</tr>
<tr>
<td>1999-2002</td>
<td>Editorial Board, British Journal of Pharmacology</td>
</tr>
<tr>
<td>2013-2014</td>
<td>Councilor (elected), Association of Medical School Neuroscience Department Chairpersons 2014</td>
</tr>
<tr>
<td>2016-2018</td>
<td>President (elected) of the Association of Medical School Neuroscience Department Chairpersons</td>
</tr>
</tbody>
</table>

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
BILOGRAPHICAL 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: John K Hubbard, PhD, PT

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

POSITION TITLE: Associate Professor (Instructional)

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>
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<tbody>
<tr>
<td>Brigham Young University</td>
<td>BS</td>
<td>04/1978</td>
<td>Physical Education (Pre-Physical Therapy)</td>
</tr>
<tr>
<td>University of Southern California</td>
<td>MS</td>
<td>01/1981</td>
<td>Physical Therapy</td>
</tr>
<tr>
<td>Texas A&amp;M University</td>
<td>PhD</td>
<td>08/1996</td>
<td>Allied Health Education (Medical Anatomy)</td>
</tr>
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</table>

A. Positions and Honors

Employment
1994-1998  Adjunct Assistant Professor, Department of Anatomy and Neurobiology, Texas A&M University Health Science Center – College of Medicine
1998 – 1999 Assistant Professor, Department of Physical Therapy, Eastern Washington University
1999 – 2000  Assistant Professor, School of Physical Therapy, Texas Woman’s University
2000 – 2003 Assistant Professor; Director, Human Anatomy Laboratory, Department of Physical Therapy, Angelo State University
2003 – 2007 Physical Therapist Assistant Program Director, Blinn College, Bryan, TX
2004 – 2014 Assistant Professor, Department of Neuroscience and Experimental Therapeutics, Texas A&M University Health Science Center – College of Medicine
2014 – Present Instructional Associate Professor, Department of Neuroscience and Experimental Therapeutics, Texas A&M University Health Science Center – College of Medicine

Awards
Medical Gross Anatomy “Best Course Award” by second year medical students (2006, 2007) M1
Association of Former Students Distinguished Teaching Award – 2015
TAMHSC College of Medicine class of 2018 – Outstanding Basic Science Faculty Award

B. Contributions to Science

Peer Reviewed Publications

4. Swanson, G & Hubbard J. A better understanding of the lymphatic drainage of the prostate with modern imaging and surgical techniques. Clinical Genitourinary Cancer. 11:4, 431-440, December 2013. (Published online July 1, 2013)


Peer Reviewed Abstracts


Selected Peer Reviewed Presentations


2. Differential Diagnosis of Renal Pathology vs. Low Back Pain; Anatomy and Histology of the Renal System, National Kidney Foundation Spring Clinical Meeting, Orlando, FL April, 2010

3. Functional Anatomy of the Foot and Ankle: A Cadaver Prosection Course, Scott & White Hospital and Clinics, Temple, TX April, 2010

4. Renal Anatomy and Histology Review for Medical Practitioners; Renal Anatomy Game Show, National Renal Foundation Spring Clinical Conference – April 2011, May 2012, April 2013


8. Peripheral Nerve Injuries, Scott & White Continuing Education Course – Temple, TX; April, 2014


15. “Combining Active Learning and Immediate Quiz Feedback: Engaging Students in Anatomy Medical Jeopardy!” Chico DE, Luna Arvizu LP, Allen GA, Brakora K, Hubbard JK, Carpenter RO, Chen WJ. American Association of Anatomists Meeting, 2018

17. An Anatomical Approach to Understanding and Performing Special Tests of the Shoulder Complex. Baylor, Scott and White Hospital and Clinic Continuing Education Course; April, 2018
C. Additional Information: Research Support and/or Scholastic Performance

Current Support:
None

Past Support:
San Angelo Health Foundation “Motion Analysis System”. Hubbard, JK (Project concept, initial research and authorship), Mason, C. This grant was developed to obtain funding for a gait analysis system and motion analysis system for the Human Performance Laboratory of the Angelo State University Department of Physical Therapy. 6/03-6/04 $288,000.00.

Angelo State University Carr Graduate Research Grant Foundation “The effect of stretching the iliotibial band on subjects with patellofemoral syndrome: Hubbard, JK (Primary author, Graduate student faculty advisor), Kiker, R. 5/03-5/04 $3,000.00.

Angelo State University Carr Graduate Research Grant Foundation “Hip internal/external rotation values in subjects with patellofemoral joint dysfunction” Hubbard, JK (Primary author, Graduate student faculty advisor), Alls, J. 5/02-5/03 $3,000.00.

Current College of Medicine Teaching Responsibilities
Medical Gross Anatomy – Course Director
Foundations of Medicine I – Histology Instructor
Clinical Neuroscience – Neuroanatomy Instructor
Baylor Scott and White Orthopedic Residency Program – Anatomy Rotation Director

Student Teaching Evaluations
Medical Gross Anatomy 3.82 / 4.0
Foundations of Medicine I – Histology 3.70 / 4.0
Clinical Neuroscience 3.75 / 4.0
NAME: Jeffery, Nick D

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

POSITION TITLE: Professor, Neurology and Neurosurgery

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>
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<tbody>
<tr>
<td>University of Bristol (UK)</td>
<td>BVSc</td>
<td>06/81</td>
<td>Veterinary Science / Medicine</td>
</tr>
<tr>
<td>University of Cambridge (UK)</td>
<td>PhD</td>
<td>10/97</td>
<td>Neuroscience</td>
</tr>
<tr>
<td>University College London (UK)</td>
<td>Postdoctoral</td>
<td>10/97-10/2000</td>
<td>Neuroscience</td>
</tr>
<tr>
<td>London School of Hygiene and Tropical Medicine, University of London (UK) [Distance learning]</td>
<td>MSc</td>
<td>10/2013</td>
<td>Clinical Trials (design and analysis)</td>
</tr>
<tr>
<td>European Colleges of Veterinary Neurology and Veterinary Surgery</td>
<td>Professional clinical diplomas</td>
<td>06/93 09/95</td>
<td>Veterinary Surgery Veterinary Neurology</td>
</tr>
</tbody>
</table>

A. Personal Statement

Since qualifying as a veterinarian, I have worked in many aspects of the discipline, including primary care practice, specialist private referral practice, bench-top research, clinical research and mid-level administration. My predominant clinical veterinary and research interest is spinal cord injury, especially comparative aspects with relevance to human spinal cord injury and incorporating formal clinical trials of novel therapies to ameliorate chronic loss of function after acute injuries. I have recently re-started a long-standing research question investigating the plastic changes in the nervous system induced by orthopedic lesions.

B. Positions and Honors

1981-1983: Veterinary Officer, People’s Dispensary for Sick Animals, London SE14, UK
1983-1990: Veterinary Assistant and later partner to Tom Yarrow MRCVS, Initially general practice, later solely referral practice in surgery / neurosurgery, London E7 and London E1, UK
1990-1993: Small Animal Surgeon, Animal Health Trust, Newmarket, UK
1993-1997: Wellcome Trust PhD Studentship, Dept Veterinary Medicine, University of Cambridge, UK
1997-2000: Wellcome Trust Research Fellowship, Dept Anatomy and Developmental Biology, University College London, UK
2000-2004: University Lecturer (~Associate professor) in Clinical Neurology, Dept Veterinary Medicine, University of Cambridge, UK
2004-2006: University Senior Lecturer in Clinical Neurology, Dept Veterinary Medicine, University of Cambridge, UK
2006-2010: Professor of Veterinary Clinical Studies, Dept Veterinary Medicine, University of Cambridge, UK
2010-2016: Professor, Neurology & Neurosurgery, College of Veterinary Medicine, Iowa State University, USA
Oct 2016: Professor, Neurology & Neurosurgery, College of Veterinary Medicine, Texas A&M University, USA

Other
Assistant editor The Veterinary Journal (from Jan 2013-May 2015)
Invited reviewer for American Kennel Club grants (neurology section) – from July 2013
Member of scientific committee of European College of Veterinary Neurology – Sep 2009 to date Member of credentials committee of European College of Veterinary Neurology – Sep 2010 to date
Editor-in-Chief of the Journal of Small Animal Practice (from May 2015)
Frank K Ramsey Endowed Chair in Veterinary Medicine, Iowa State University (2014)
Maureen E Mullins Professorship in Small Animal Clinical Sciences, Texas A&M University (2016)

c. Contribution to Science

1. Recognition of the important potential to use veterinary dog patients as a model for investigating the translational potential of interventions shown to be successful in laboratory animals, plus devising and executing relevant studies. There are a multitude of interventions that have been shown to ameliorate the functional deficits associated with spinal cord injury in experimental animals but none has so far been able to be translated into a successful therapy in human patients. Spinal cord injury is much more common in dogs and the mechanisms involved and patient heterogeneity are similar between the species. Therefore dogs present an excellent opportunity to test putative therapies in a ‘real-life’ clinical disease. This work has resulted in several publications (see below), focusing on development and testing of olfactory ensheathing cell transplantation (which we have shown to have some objectively quantifiable benefits and therefore has potential role to play in treatment of the human disease) but also currently continuing through a current clinical trial on chondroitinase.


2. Investigation of the functional effects of demyelination and remyelination in the CNS and disruption of normal myelin patterns in chronic spinal cord compression. Cell transplantation and myelin replacement are thought to be possible methods by which to reverse the functional deficits associated with demyelination in diseases such as multiple sclerosis. Although electrophysiological tests demonstrated that conduction could be restored through previously demyelinated axons through the process of remyelination it was not known whether this would also restore normal function. As a PhD student I devised a method to measure the functional deficits associated with demyelination in the dorsal funiculus of the rat spinal cord and used this method to show that demyelination was associated with detectable deficits that could be reversed by spontaneous or transplant-mediated remyelination. The observations I made through...
pathological analysis of naturally-occurring spinal cord injury in dogs have now become the basis for a PhD project (co-supervised by me) to investigate the structural disruption of the paranode associated with chronic spinal cord compression.


3. Development of methods to quantify the loss of function associated with spinal cord injury in large quadrupeds. To evaluate the effects of interventions designed to ameliorate the consequences of spinal cord injury it is necessary to have highly sensitive reproducible measures of loss of function. I used kinematic analysis of gait on a treadmill to achieve this, devising a measure of coordination between fore and hind limbs that was consistent across all gait patterns, sizes of animal and independent of non-neurologic deficits. This technique has formed the basis for objective quantification of the effect of interventions such as olfactory ensheathing cell transplantation and will be used in the current project.


b. Hamilton L, Franklin RJM, Jeffery ND (2008) Quantification of deficits in lateral paw positioning after spinal cord injury in dogs. BMC Veterinary Research 4:47


4. Much of my clinical work has focused on description of new methods of treatment for various, mostly neurosurgical, diseases and analysis of their effectiveness. The publications arising from this work have aided progress in veterinary medicine.


d. Research Support
Ongoing research Support

International Spinal Research Trust (UK) Granger & Jeffery (co-PIs) 03/01/2017-03/01/2019
Characterization of neurogenic urinary incontinence in the canine model of spinal cord injury using a rage of urodynamic measures

Keele University (UK) Chari, Granger, Jeffery (co-PIs) 9/1/2017-9/1/2018
RMCIF1: Protective matrices for delivery of genetically augmented canine olfactory cell transplants.
Funds given to support development of new methods of cell transplantation to the spinal cord to optimize cell survival

Recently completed research support

EPSRC (UK) Lacour (PI) 10/01/10-09/30/13
Nanoengineering to promote growth of damaged peripheral nerves
This project was carried out by a large consortium of UK engineers and neuroscientists aiming to develop implants through which severed peripheral nerves could grow and have their activity measured.
Role: Co-investigator

University of Iowa Howard (PI) 06/01/11-03/01/16
Development of the ‘I-patch’ spinal cord neuromodulation device
This project is led by Professor Howard at the University of Iowa and consists of a multidisciplinary team working to develop a novel spinal neuromodulatory device for control of pain and amelioration of post-traumatic spasticity. The funding provided to Iowa State University supports a post-doc who works with me to analyze locomotor effects of spinal cord stimulation.
Role: Co-PI

Grayson Jockey Fund Jeffery & Alcott (co-PIs) 01/01/13-01/01/16
The relationship between transcranial magnetic evoked motor potentials and lesions in the cervical spinal cord of ‘wobbler’ horses.
This project seeks to determine the potential use of transcranial magnetic motor evoked potential analysis as a means of measuring the severity of injury to the cervical spinal cord in an equine cervical spondylomyelopathic disease.
Role: PI

AKC Canine Health foundation Jeffery (PI) 01/01/13-06/30/15
Investigating the relationship between gut microbiota and development of meningoencephalomyelitis of unknown origin in dogs
This project seeks to compare the constituents of the gut microbiota between dogs affected by the immune-mediated disease ‘MUO’ and control dogs, as a means of identifying a trigger factor.
Role: PI

BioMarin Pharmaceutical Inc. Ellinwood (PI) 04/01/14-09/30/15
Twenty-six week or longer intracerebroventricular infusion study of BMN 250 administered biweekly in a canine model of mucopolysaccharidoses type IIIB (MPSIIIB).
This study is evaluating the ability of the agent to reverse clinical signs associated with MPS III in a colony of experimental dogs
Role: Co-PI

McGee-Wagner Foundation (ISU): Jeffery & Luecke (co-PIs) 06/01/14-05/31/17
Development of an implant to maintain locomotor equilibrium in dogs with chronic severe spinal cord injury. This project, funded by an internal foundation at Iowa State University, is a collaboration with engineering faculty to develop an electronic implant that will prevent falling in dogs with severe spinal cord injury.
Role: PI

Frank K Ramsey endowed chair (ISU) Jeffery (PI) 07/01/14-06/30/17
Funds to support research on any chosen topic
These funds are being used to develop the electronic implant for prevention of falls in severe spinal cord injured-dogs as described above.
Role: PI

International Spinal Research Trust (UK)  Jeffery & Bellamkonda (co-PIs)  06/01/13-12/31/16
Clinical trial to define effectiveness of intraspinal chondroitinase ABC injection as a treatment for severe spinal cord injury in dogs.
This clinical trial is designed to test the potential of ChABC to translate from lab to clinical spinal cord injury.
Role: PI

Wellcome Trust (UK)  Jeffery & Franklin (co-PIs)  07/01/08-12/31/16
Integrated Veterinary Training Fellowship
This funding provides support for a training program in clinical neurology and basic neuroscience. The neuroscience project is investigating paranodal demyelination in chronic spinal cord cord compression.
Role: Co-PI
NAME: Larry Johnson

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

POSITION TITLE: Professor, Faculties of Toxicology and Reproduction

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>
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<tr>
<td>North Carolina State University</td>
<td>B.Sc.</td>
<td>1971</td>
<td>Animal Science</td>
</tr>
<tr>
<td>University of Texas Health Science Center</td>
<td>Post Doc</td>
<td>1978-1980</td>
<td>Reprod. Biology</td>
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A. Personal Statement
To improve science education in rural middle schools and rural communities, I will help guide, as PI of this project, the training experiences of middle school science teachers, curriculum development, and contribute to app and online course development, and PEER website redesign. As a scientist first and more recently a science educator, I have been able to blend science and education in a series of projects including three R25 and two NSF GK12 grants. At every annual meeting of all these grant, my group produced and presented one or more posters describing our K12 activities that year. As a scientist, I have published over 110 original, peer-reviewed, scientific journal articles; given invited scientific talks on four continents; won a national research award; served on a research panel for the United States Congress; served on NIH, NSF, USDA, and NIOSH grant review panels; received NIH and/or NSF Funding for over 25 years; served on editorial boards of their scientific journals; and received both college-level and university teaching awards for my histology courses. As PI of three R25 grants over the last 12 years, I have directed teacher training throughout the state, and curricular development for middle school science, math, social studies and English language arts. While our group of faculty were “Visiting Scientists” in middle schools statewide (38,000 K-12 students and their 1,727 teachers), I visited 23,000 of these K-12 students and their teachers personally promoting health understanding and appreciation of the scientific process to K-12 students and teachers. In addition to having a scientist visit their classroom, 1,150 middle school teachers received 2-day workshop training on technology, integration or science curricular materials we developed and e-mentoring opportunities. Our group produced 150 days of integrative curriculum in four subject areas (all lessons tied to state educational standards). Our NSF GK12 project, on which I served as PI, trained 62 graduate students with science communication and mentoring through their K12 public school experiences. They interacted with 64 main teachers and 110 others and over 13,620 K-12 students (54% minority students and 59% eligible for reduced lunches). I have developed an elective for third year veterinary students in which they learn about pedagogy of public school interactions, develop a formal presentation, and give the presentation to middle school students (4-5 times @ ~ 100 students). Hence, I am experienced at effective teacher training and interactions, and with their interactions with veterinary students as well as graduate students. My background as a scientist has allowed me to recruit several scientists/veterinarians to partner with teachers on this project and share how their authentic research uses the scientific method with partner teachers. My histology teaching and online delivery (https://www.youtube.com/histology) will allow me to extend this course to high schools. I am currently producing an on line high school histology elective course for the Texas Education Agency. My presentations to students and teachers of rural areas and professional development of teachers in rural areas, equip me to fuse basic science concepts through animal and human subject use in research to a One-Health world view whereby research benefits all species and environments. My focus as project leader is to promote One-Health information and STEM
understanding in rural schools by combining teacher professional development, curricular development, and delivery of information via an updated PEER website with online courses and educational app.

B. Positions and Honors

Positions and Employment
1980-1987: Asst. Professor in Cell Biology, Univ, of Texas Health Science Center - Dallas, TX
1988-1993: Assistant Professor, Human Nutrition, Center for Environmental and Rural Health, Texas 1987-1992
    Assoc. Professor, Division of Cell Biology & Ultrastructure, Department of Veterinary
    Anatomy, College of Veterinary Medicine, Texas A&M University, College Station, TX.
1992-present Professor, Faculty of Toxicology, and Faculty of Reproductive Biology, Department of Veterinary Integrative Biosciences, College of Veterinary Medicine and Biosciences, Texas A&M University, College Station, Texas.

Other Experience and Professional Memberships
1989-1995 Member of the Editorial Board of Biology of Reproduction
1989-1995 Member of the Editorial Board of Journal of the American Aging Association
1997-2008 Member of the Editorial Board of Journal of Andrology

Honors
1983-1986 NIH New Investigator Research Award.
1988 Young Andrologist Award, from the American Society of Andrology.
1988 Invited speaker (Spermatogenesis, Animal Species and Humans) at 1988 International Symposium on Gamete Physiology, Serono Symposia, USA;
1990 Invited speaker (Spermatogenesis in humans and animal species), Japan Soc of Androl
1995 Invited speaker (Spermatogenesis in domestic animals and approaches to its enhancement) at the XI Brazilian Congress of Animal Reproduction.
1996 Invited speaker (Efficiency of Spermatogenesis in Humans), College of Medicine, University of Utrecht, The Netherlands.
1996 University-level teaching award from TAMU Honors Undergraduate Program.
1997 Invited speaker (Transplantation on Spermatogenesis: Sperm Decline in Humans), La Federation Francaise Pour L’etude de la Reproduction.
1999 College-level Teaching Award in Biomedical Science Undergraduate Program.
2001 The KINDER Award (Kids in Need of Drug Evaluation and Rx Treatment Clinic in Houston, TX) for contributions to the wellbeing of children at risk through our Partnership for Environmental Education and Rural Health Program (http://peer.tamu.edu)
2001 Local Texas A&M University Chapter of Sigma Xi Science Communication Award.
2007 Association of Former Students Distinguished Achievement Award for Extension and Outreach from Texas A&M University.
2008 Invited speaker (Introduction to Toxicology) at the University of Science and Technology—Beijing and at Beijing Normal University.
2009 Texas A&M University Bush Excellence Award for Faculty in Public Service/Outreach
2012 Outstanding Educator Award – Dean’s Roundtable for College of Education and Human Development, Texas A&M University.
2014 Invited Speaker (scientific ethics) at the University of Beijing Science and Technology and “Love your lungs” given at a local high school in Beijing
2015 National Sigma Xi – The Evan Ferguson Award for Service
C. Contributions to Science
110 Original Scientific Publications and Promotion of Science Education

1. REGULATION OF SPERMATOGENESIS
Our overall research objective is to understand the process of spermatogenesis: what is the role of germ cell degeneration and how Sertoli cell number and function regulated spermatogenesis. We have systematically studied spermatogenesis through its process and identified key steps of development where degeneration reduces sperm production ratios in humans and horses. We have shown age-related decline in Sertoli cell numbers in humans that corresponds to the decline in sperm production rates. We found seasonal variation in Sertoli cell numbers and function in horses. To help explain seasonal differences in Sertoli cell numbers, we experimentally altered the Sertoli cell division pattern in rats.


2. PROMOTION OF SCIENCE EDUCATION HAS TAKEN THESE TWO FORMS: PEER EXPERIENTIAL LEARNING AND HISTOLOGY TECHNOLOGY
Partnership for Environmental Education and Rural Health (PEER) was developed as a K-12 program to provide professional development of teachers, develop materials to aid in their teaching, and to place scientists in the classroom. A unique feature of PEER is its searchable Teacher Requested Resources (see http://peer.tamu.edu/DLC/NSF_Resources.asp). The feature allows teachers in remote rural locations to request lesson plans, activities, and websites as they inform us of their specific needs and direct our development. Once the initial response has been produced by the undergraduate/graduate Fellows, it is evaluated for content by STEM faculty and for public school and age appropriateness by middle school teachers both in PEER staff and in the field. Currently we have 678 such lessons. Each year PEER is the host of a statewide group of high school FFA students attended the Texas State FFA training and veterinary assistance contest sponsored by PEER. PEER is also collaborating with 4-H summer programs.

As stated in my personal statement, we have trained 1,150 middle school teachers and visited 38,000 K-12 students in their schools. I personally presented to 23,000 students and their teachers. I reached over 7,000 students through two teleconferences last year (http://peer.tamu.edu/Videoconferences.asp). Over the 15 years of our science promotion efforts, we have developed the complete science curricula for 6-8 grade through integrated curricula, life science curricula, and veterinary-related curricula. We have also provided teacher requested resources where by teachers submit a request for a lesson. The typical response yielded a PowerPoint introduction to the topic, a lesson guide, activity description, and corresponding worksheets. This way teachers help direct us to what teachers need and are using in schools. (http://peer.tamu.edu/DLC/TeacherRequest.asp)

Experiential learning was provided to undergraduate students and graduate students who receive training on learning and lesson creation. The undergraduates responded to the teacher requests by making a fun PowerPoint presentation related to the content and finding neat activities that illustrated the content material. Our PEER teachers on payroll would refine it into a formal lesson to be used by the requesting teacher and other teachers via the PEER website. Veterinary students take a formal course, whereby they receive training on pedagogy, how kids think, and are motivated as well as how to deliver and interesting lesson. These vet students present their presentation and hands-on activity for the middle school students. Hence by interacting with PEER teacher...
staff and faculty, both undergraduate students and veterinary students learn how to communicate effectively with different levels of students and develop a lifelong interest in the promotion of science.

Through technological advancements and access, I have made courses’ laboratory material (presentation and images) available from three histology courses (freshman, and senior undergraduate, level and medical school) at

https://www.youtube.com/histology. I have provided free science education for a year and a half and the freshman course (medical school histology basics) has matching PowerPoints that give access to the original images as if one was looking at the histology slide. This STEM YouTube channel enjoys 8,157 subscribers and 629,267 views of which only 28% are from the USA. Hence, it has an international reach. These videos will contribute to the online course and app delivery of the cell biology and histology content.

In addition to abstracts in annual proceedings for K12 NIH and NSF grants PEER have enjoyed that accompanied poster presentations of our K12 activities that year we produced three magazines on our K12 activities.

a. Virginia Traweek and Larry Johnson. 2006. PEER Perspectives: Broadening the Reach of University Resources by Advancing STEM (http://peer.tamu.edu/mags/PEER_perspectives.pdf)

D. Research Support

National Science Foundation 2014-2017
Research on Education and Learning (DIR). Data-Driven Methods to Improve Student Learning from Online Courses. The goal is to produce online STEM courses for middle schools to alter delivery in schools and to compare the online version of the content delivery to conventional text book/lecture formats used in schools
Role: Co-I

Barnsly Foundation 2011-2017
Veterinary School Promotes Animal Welfare in Midland/Odessa/Crane area High School. The objective is for veterinary students to share as near-peers their interest in veterinary medicine, provide their academic path, and career opportunities in STEM in schools near the Foundation (Midland/Odessa/Crane area).
Role: PI

TAMU Allocation Funds 2011- Current
Professional Student Service Learning Experiences. Veterinary School Promotes STEM in middle schools and in informal settings. The objective is for veterinary students to share as near-peers their interest in veterinary medicine, provide their academic path, tips on success in college, and career opportunities in STEM.
Role: PI

TAMU Allocation Funds 2011- Current
Larry Johnson, Pt.
Undergraduate Service Learning Experiences.
Undergraduate students STEM in rural middle/high school through teacher-requested resources. The objective is for undergraduate students to work closely with middle school teachers and PEER scientists to provide lessons requested by teachers (http://peer.tamu.edu/DLC/NSF_Resources.asp?type=search&num=5&hl=no)
Role: PI

Texas Agricultural Experiment Station 2014- Current
Larry Johnson, Pt.
4-H/FFA project interfacing with PEER. Goals are to provide 4H and FFA students with STEM content related to their rural farm interest and provide the FFA state contest for veterinary assistance competition based on knowledge in several categories
Role: PI
John Deere

Undergraduate Service Learning Experiences

Undergraduate students STEM in rural middle/high school through teacher-requested resources. The objective is for undergraduate students to work closely with middle school teachers and PEER scientists to provide lessons requested by teachers (http://peer.tamu.edu/DLC/NSF_Resources.asp?type=search&num=5&hl=no)

Role: PI
NAME: Sharon C. Kerwin

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.)

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<tr>
<td>Texas A&amp;M University</td>
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<td>Veterinary Neurology</td>
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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:

Chico


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:

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
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 GMRD 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 (Pelizaeous Merzbacher) and cerebellar hypoplasia, as well as those discussed under “imaging” and “comparative oncology” below.


3. Cross-Sectional Imaging as a Diagnostics 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 Hoffman’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 09/01/2015-08/31/2018
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 04/01/2016-06/30/2018
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) 07/01/14-08/14/17
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 01/01/14-12/31/17
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) 04/1/2017-03/31/2018 (Year 4 of parent grant)
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.
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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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

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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) 10/11/10-1/15
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) 3/11/13-3/1/13
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) 1/10/13-1/1/13
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
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: 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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<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>
</tr>
<tr>
<td>University of Rochester, Rochester, N.Y.</td>
<td>Ph.D.</td>
<td>1989</td>
<td>Biopsychology/Neurobiology</td>
</tr>
</tbody>
</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 (PMCC5102408) 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  Psychaid, Bombay, India, Clinical assistant, psychological testing
1984-1989  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 mediating 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, GABA 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
nerve growth factor receptor mRNAs in adult sensory neurons. Journal of Neuroscience, 14, 459-471.


Complete List of published works in MyBibliography:

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 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.

OVERLAP: None.

R21 NS091723 (Grau, PI) 4/1/2016-3/31/2018
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.
OVERLAP: None.

Completed Research Support
RO1AA13440 (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.
CURRICULUM VITAE

Name: Hongmin Qin
Title: Associate Professor
Address: 3258 TAMU, College Station, TX 77843
Telephone: (979)-458-0512  Fax: (979)
E-mail: hqin@bio.tamu.edu

A. Education/Training

<table>
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<th>INSTITUTION AND LOCATION</th>
<th>DEGREE</th>
<th>YEAR(s)</th>
<th>FIELD OF STUDY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Shandong University, China</td>
<td>B.S.</td>
<td>1989-1993</td>
<td>Microbiology</td>
</tr>
<tr>
<td>Shandong University, China</td>
<td>M.S.</td>
<td>1993-1996</td>
<td>Microbiology</td>
</tr>
<tr>
<td>The Institute of Microbiology, Chinese Academy of Sciences, China</td>
<td>Ph. D.</td>
<td>1996-1999</td>
<td>Plant Molecular Biology</td>
</tr>
<tr>
<td>Yale University</td>
<td>Postdoctoral Training</td>
<td>1999-2005</td>
<td>Cell Biology</td>
</tr>
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</table>

B. Positions and Employment

2013 - Present      Associate Professor, Department of Biology, Texas A&M University
2006 - 2013         Assistant Professor, Department of Biology, Texas A&M University
1999 - 2005         Postdoctoral Research Fellow, MCDB Dept. Yale University

C. Awards and Honors

2006               TAMU nominee for Searle Scholar Program (two from each invited university)
2002 - 2004        Polycystic Kidney Disease Foundation Postdoctoral Fellow
2001               Best-Paper Award, Beijing Society for Plant Pathology (Highlighted in the publication list)
1999               “DiAo” Predoctoral Fellowship, Chinese Academy of Sciences, China
1993 - 1996        “SunShine” undergraduate excellence Fellowship, China

D. Professional Experience

- Dept. of Biology Student and Postdoctoral Research Committee, Chair, 2017-
- Genetics Graduate Advisory Committee member, 2017-
- Dept. of Biology Student and Postdoctoral Research Committee, member, 2016
- Faculty mentor for TAMU Posse Atlanta 5, 2017-
- Dept. of Biology Graduate Program committee, member, 2012 – 2016; 2017-
- Dept. of Biology Graduate Admission and Recruitment committee, member, 2007-2009
- Student Research Week judge, 2006, Texas A&M University.
- Ad Hoc reviewer, Israel Science Foundation, 2009, total 1 proposal.
- Book reviewer, reviewed 4 chapters for a Cell Biology text book (Author and educator, Dr. George Plopper) from the Jones & Bartlett Publishers, LLC, 2010.
Biographical Sketch

- 2010 and 2011, Student/ Post-doc Research Conference (SPRC) judge, Department of Biology, TAMU.
- The Texas A&M System Louis Stokes Alliance for Minority Participation (LSAMP TAMU) 7th Annual Symposium judge, 2010, supported by NSF.
- Reviewer for scientific publications for *Molecular Biology of Cell, Current Biology, Genetics, the Journal of Cell Biology, Nature of Cell Biology, Sensors, PLOS One, Trends in Cell Biology*
- Speaker at the annual meetings of American for Cell Biology, Gordon Conference and other professional meetings
- Coordinator for a teaching in China program “The GREAT Program” offered by the Capital Normal University in China, 2008-2016.
- Advisor for the exchange student program sponsored by the Dean’s exchange program from the Capital Normal University in China, Nov. 2009-Mar, 2010.

LIST OF PUBLICATIONS

*(This section does not count towards the 2-page limit set by USDA.)*


Biographical Sketch


**Invited Review or Book Chapters**
1. Qin H. Regulation of intraflagellar transport and ciliogenesis by small g proteins. *Int Rev Cell Mol Biol.* 2012;293:149-68.


3. Richey E, Qin H. Isolation of Intraflagellar Transport Particle Proteins from *Chlamydomonas reinhardtii*. In press at *Methods in Enzymology (Cilia)*.

**Patents (In China)**


**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 (if applicable)</th>
<th>Completion Date MM/YY</th>
<th>FIELD OF STUDY</th>
</tr>
</thead>
<tbody>
<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>
</tr>
</tbody>
</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**

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<tr>
<th>Year</th>
<th>Position / Employment</th>
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<tr>
<td>1992-1993</td>
<td>Junior Research Fellow in Pharmacology, Panjab University, Chandigarh (India)</td>
</tr>
<tr>
<td>1994-1996</td>
<td>Senior Research Fellow in Pharmacology, Panjab University, Chandigarh (India)</td>
</tr>
<tr>
<td>1997-1998</td>
<td>Assistant Professor (Lecturer) of Pharmacology, Panjab University, Chandigarh</td>
</tr>
<tr>
<td>1998-2001</td>
<td>Postdoctoral Fellow, Epilepsy Research Branch, NINDS, NIH, Bethesda, MD</td>
</tr>
<tr>
<td>2002-2007</td>
<td>Assistant Professor of Pharmacology, North Carolina State University, Raleigh, NC</td>
</tr>
<tr>
<td>2008-2013</td>
<td>Associate Professor, Department of Neuroscience and Experimental Therapeutics, Texas A&amp;M University Health Science Center, College of Medicine, Bryan, Texas.</td>
</tr>
<tr>
<td>2008-present</td>
<td>Faculty Member, Texas A&amp;M Institute of Neuroscience (TAMIN), College Station, Texas.</td>
</tr>
<tr>
<td>2013-present</td>
<td>Professor, Department of Neuroscience and Experimental Therapeutics, Texas A&amp;M University Health Science Center, College of Medicine, Bryan, Texas.</td>
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**NIH Study Sections and Editorial Activity**

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<tbody>
<tr>
<td>2010-present</td>
<td>Scientific Reviewer, US Department of Defense Medical Research Program grant review panel.</td>
</tr>
<tr>
<td>2010-present</td>
<td>Member NIH Special emphasis panel – ZRG1-MDCN-50(J)– CounterACT program.</td>
</tr>
<tr>
<td>2010-2012</td>
<td>Chartered Member, NIH Study Section – CNNT– Clinical Neuroplasticity and Neurotransmitters.</td>
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<tr>
<td>2011-2011</td>
<td>Member, NIH Special emphasis panel – ZNS1 SRB-B-32 – EUREKA epilepsy grants.</td>
</tr>
<tr>
<td>2008-2010</td>
<td>Chartered Member, NIH Study Section – ANIE – Acute Neuronal Injury and Epilepsy.</td>
</tr>
<tr>
<td>2007-2008</td>
<td>Ad hoc Member NIH Study Section – CND– Clinical Neuroscience and Disease.</td>
</tr>
<tr>
<td>2007-2009</td>
<td>Ad hoc Member NIH Study Section – CNNT– Clinical Neuroplasticity and Neurotransmitters.</td>
</tr>
<tr>
<td>2009-present</td>
<td>Ad hoc Member NIH Study Section – ICP1– International and Cooperative Projects.</td>
</tr>
<tr>
<td>2009-present</td>
<td>Member Grant Review Panel– New Zealand Neuroscience Foundation.</td>
</tr>
<tr>
<td>2012-present</td>
<td>Grant Reviewer, Texas A&amp;M—Weizmann Institute Israel Collaborative Program.</td>
</tr>
<tr>
<td>2008-2010</td>
<td>Executive Editor, International Journal of Pharmaceutical Sciences and Nanotechnology</td>
</tr>
<tr>
<td>2011-present</td>
<td>Editor-in-Chief, International Journal of Pharmaceutical Sciences and Nanotechnology</td>
</tr>
<tr>
<td>2010-present</td>
<td>Review Editor, Frontiers in Aging Neuroscience; Frontiers in Pharmacology</td>
</tr>
<tr>
<td>2003-present</td>
<td>Editorial boards/reviewer: over 20 pharmacology/neuroscience journals.</td>
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**Professional Activity and Membership**

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<tr>
<td>1999-present</td>
<td>Member: Society for Neuroscience; International Brain Research Organization</td>
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<tr>
<td>1999-present</td>
<td>Member: American Epilepsy Society &amp; Coordinator – AES’s SIG on Neuroendocrinology</td>
</tr>
<tr>
<td>1999-present</td>
<td>Member: American Society for Pharmacology and Experimental Therapeutics</td>
</tr>
<tr>
<td>1999-present</td>
<td>Member: American Association of Pharmaceutical Scientists; AAAA</td>
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<tr>
<td>2001-2002</td>
<td>Member, NIH Scientific Committee, Fellows Award for Research Excellence</td>
</tr>
<tr>
<td>2008-present</td>
<td>Scientific program/ abstract committee, American Association of Pharma Scientists</td>
</tr>
<tr>
<td>2010-present</td>
<td>Coordinator, American Epilepsy Society Neuroendocrinology SIG session.</td>
</tr>
<tr>
<td>2012-present</td>
<td>Member, The United States Pharmacopoeia (USP).</td>
</tr>
<tr>
<td>2016-present</td>
<td>Member, The United States Environmental Protection Agency (EPA) Science Advisory Board.</td>
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**Honors and Awards**

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<tr>
<td>1992</td>
<td>Gold Medalist (six medals for academic excellence), Kakatiya University, Warangal, India</td>
</tr>
<tr>
<td>1992</td>
<td>Master’s Fellowship, University Grants Commission, New Delhi, India</td>
</tr>
<tr>
<td>1993</td>
<td>GP Nair Award, Indian Drug Manufacturers' Association, Bombay, India</td>
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</table>
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
1998  **BK Anand Prize** in Physiology & ND Dutta Prize in Pharmacology Research
2000  **NIH Fellows Award for Research Excellence**, National Institutes of Health, USA
2004  **Young Scientist Travel Award**, American Society for Pharmacol Exp Therap (ASPET)
2006  **Faculty Research Award**, NC State Sigma Xi award for research excellence, USA
2007  **Young Scientist Award**, ASIOA – Neuroscience meeting, San Diego, USA
2012  **NATA Research Excellence Award** – GBR Convention meeting, Houston, TX, USA
2013  **TANA Award of Excellence** – TANA meeting at Dallas Convention Center, Dallas.
2014  **Hind Rattan (Jewel of India) Award**, New Delhi, India.
2014  ATA Award for Excellence in Science Research (2014), a non-profit association in USA.
2014  Research Award of the American Association of Physicians of Indian Origin (AAPI), the largest ethnic medical organization in the USA with ~ 75,000 physicians/ medical residents.
2014  **FAAPS - Fellow of the American Association of Pharmaceutical Scientists** (2014), the highest professional honor of achievement in pharmaceutical field worldwide.
2015  **FAAAS - Fellow of the American Association for the Advancement of Science** (2015), one of the most prestigious scientific organizations in the world & the publisher of the journal *Science*.
2016  **FAES -- Fellow of the American Epilepsy Society**, a lifetime professional honor in recognition of sustained accomplishments and dedication to excellence in epilepsy.

### 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.

   - Gangisetty O and Reddy DS (2010). Neurosteroid withdrawal regulates GABA-A receptor α4-subunit expression and seizure susceptibility by activation of PR-independent Egr3 pathway. *Neuroscience* 170: 865-
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: http://www.ncbi.nlm.nih.gov/pubmed/?term=Reddy%2BDS
Biographical Sketch

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
BIOGRAPHICAL 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

University of Utah  
postdoc  
1992-1995  
Zebrafish Devel.

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.

Honors

B.A. with distinction, University of Colorado, 1982.
Postdoctoral Fellowship, ACS, University of Utah, 1994-1996.
NIH awardee, 1998-present.
Biographical Sketch
C. Selected Peer-Reviewed Publications


Biographical Sketch


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
Biographical Sketch

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
**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:** 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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<td>University of Maryland at Baltimore, MD</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**
Biographical Sketch

**Positions and Employment**
1998  Instructor, Department of Biology, New Mexico State University.
Biographical Sketch

2000-07  Assistant Professor, Section of Neurobiology, University of Texas, Austin, TX.
2007-12  Assistant Professor, Department of Neuroscience & Experimental Therapeutics, College of Medicine, Texas A&M Health Science Center, College Station, TX.
2007-08  Adjunct Assistant Professor, Section of Neurobiology, University of Texas, Austin, TX.
2012-    Associate Professor (Tenured), Department of Neuroscience & Experimental Therapeutics, College of Medicine, Texas A&M Health Science Center, Bryan, TX.

Other Experience and Professional Memberships
1995-    Member, Society for Neuroscience
2008-    Member, International Society for Developmental Neuroscience
2004     Invited Lecturer, International Brain Research Organization (IBRO) Course in Neuroscience, University of Bucharest, Romania. May 3-12
2005     Member, Training Faculty, Texas Consortium in Behavioral Neuroscience
2004-06  Ad hoc reviewer, Association Française contre les Myopathies (French Association against Myopathies),
2007     Ad hoc reviewer, National Science Foundation
2010     Ad hoc reviewer, US-Israel Binational Science Foundation
2013     Ad hoc reviewer, Swiss Foundation for Research on Muscle Diseases
2013     Ad hoc reviewer, NIH Study Sections Neurodifferentiation, Plasticity and Regeneration (NDPR) and Molecular Neurogenetics (MNG)
2016     Ad hoc reviewer for Early Career Awards at the Uniformed Services University for Health Sciences, Bethesda, MD

Honors
1986     Summa cum laude, Universidad de Los Andes, Mérida, Venezuela
1995-96  Postdoctoral Fellowship Myasthenia Gravis Foundation of America
1996-97  NIH Training Grant 2T32NS07158-16 021
1999-00  NIH Underrepresented Minority Research Supplement, R01 NS27963, Steven J. Burden, Ph.D., Principal Investigator
2003     Summer Research Travel Award, Minority Access to Research Careers (MARC) Program, Federation of American Societies for Experimental Biology (FASEB)
2006     UT-Austin, College of Natural Sciences Teaching Excellence Award

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 AChRβ 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 AChRβ (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 *ChRNA* 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 prepattern. 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
Biographical Sketch

R03NS071141  M. Rimer (PI)  02/01/11- 01/31/14 NIH
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
          Faculty of Reproductive Biology
          Faculty of Marine Biology
2005-date  Director, Centro de Investigaciones Científicas de las Huastecas “Aguazarca”
2010-2014  Chair, Faculty of Ecology and Evolutionary Biology
2009-2013  Associate Professor, Department of Biology
2006-2009  Assistant Professor, Department of Biology
2002-2006  Assistant Professor, Boston University Marine Program, Department of Biology, Boston University
2003  Visiting professor, Interuniversity Institute, Eilat, Israel
2000-2002  National Eye Institute postdoctoral fellow

(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;
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.)

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<td>B.A.</td>
<td>12/1995</td>
<td>Psychology</td>
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<td>State University of New York, Stony Brook</td>
<td>M.S.</td>
<td>06/2000</td>
<td>Biopsychology</td>
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<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

01/2001-05/2003 Lecturer, Department of Biology, SUNY Stony Brook, Stony Brook, NY
10/2005-12/2007 Assistant 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).
12/2007- 08/2015 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).
Effective 09/2015

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.


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 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.)

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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.

4. L. N. Smith et al., Fragile X mental retardation protein regulates synaptic and behavioral plasticity to
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.
   d. Polesskaya, O.O., Fryxell, K.J., Merchant, A.D., Locklear, L.L., Ker, K., McDonald, C.G.,
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 MyBiography:
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: 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>
<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>St. Xaviers 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, NY</td>
<td>M.S.</td>
<td>1989</td>
<td>Neurobiology</td>
</tr>
<tr>
<td>University of Rochester, Rochester, NY</td>
<td>Ph.D.</td>
<td>1991</td>
<td>Biopsychology/Neurobiology</td>
</tr>
<tr>
<td>Columbia University Medical Center, NY, NY</td>
<td>Postdoctoral</td>
<td>1991-1994</td>
<td>Neurobiology/MolecularBiology/Tissue Culture</td>
</tr>
</tbody>
</table>

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 neuroprotector.

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 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 antagomirs in a stroke model. Although this
approach proved successful in young females it 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 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 neuroinflammatary 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
Completed (in the last 3 years):

1R01AG041360 (PI: Griffith)  Role: Co-I  4/1/11-3/31/16
NIH/NIA
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.

#14GRNT18370013 (PI: Earnest)  Role: Co-I  1/1/14-12/31/15  AHA
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.
Brandon J Schmeichel

Professor of Psychology

POSITIONS:
Professor, Texas A&M University, 2015-present
Associate Professor, Texas A&M University, 2010-2015
Assistant Professor, Texas A&M University, 2005-2010

EDUCATION:
Ph. D. in Social Psychology, Florida State University, 2005
M. S. in Experimental Psychology, Georgia Southern University, 2000
B. A. in Psychology, University of Nebraska, 1996

SELECT HONORS/AWARDS:
Fellow of the Society for Personality and Social Psychology (2015)

GRANTS AS PI, co-PI, or co-I:


SELECT PUBLICATIONS (of 60 journal articles, 13 book chapters, 3 other):


SELECT PRESENTATIONS:

Schmeichel, B. J. (2017, June). Cognitive ability and human emotional life. Keynote address at the Swiss Summer School on Cognitive Control and Consciousness hosted by the University of Bern and the Jacobs Foundation, Weggis, Switzerland.


SIGNIFICANT TEACHING ACTIVITIES:

I teach PSYC 301 (Statistics) to undergraduates at least once a year. This course is required of all Psychology majors and is considered a large service course. I also routinely enroll 10 to 20 undergraduate research assistants in my laboratory each semester, and I am currently supervising 3 Ph.D. students in my laboratory.
MAJOR SERVICE ACTIVITIES:
Editorial Board, *Psychological Science*, 2012-present
Editorial Board, *Self and Identity*, 2012-present
Editorial Board, *Journal of Experimental Psychology: General*, 2011-present
Editorial Board, *Emotion*, 2010-present
Editorial Board, *Social Psychological and Personality Science*, 2010-present
Head, Psychology Department Human Participant Pool Committee (2011-present)
Head, Psychology Department Affective Science Job Search Committee (2016-2017)
Member, Psychology Department Faculty Evaluation Committee (2013-2015)
Co-Organizer, SPSP Self and Identity Preconference, 2015 (with Michelle vanDellen)
Organizer, SPSP Self and Identity Preconference, 2014
Co-Organizer, SPSP Self and Identity Preconference, 2013 (with Roy Baumeister)
Associate Editor, *Journal of Experimental Social Psychology*, 2011-2013
Program Committee, Society for Personality and Social Psychology, 2011-2012
Member, Texas A&M University Institutional Review Board (2009-2012)
NAME: Rahul Srinivasan

BIOGRAPHICAL SKETCH

POSITION TITLE: Assistant Professor

EDUCATION/TRAINING

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<tr>
<td>University of Mumbai, India</td>
<td>MBBS</td>
<td>01/2000</td>
<td>Medicine</td>
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<tr>
<td>University of Pittsburgh, Pittsburgh, PA</td>
<td>PhD</td>
<td>06/2006</td>
<td>Human Genetics</td>
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<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>
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</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:
1999-2000    Internship; Residencies – University of Mumbai, India
2000-2001   General Physician, Mumbai, India
2001-2006   Doctorate, Human Genetics, University of Pittsburgh
2006-2007   Postdoctoral Associate, Molecular Genetics & Biochemistry, University of Pittsburgh
2007-2013   Postdoctoral Research Scholar, Division of Biology, California Institute of Technology
2013- Dec. 2016   Assistant Research Physiologist, Dept. of Physiology, UCLA
Jan. 2017-present   Assistant Professor, Dept. of Neuroscience and Experimental Therapeutics
                          Texas A&M Health Science Center

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
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:
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  Andrew Tag**

**Professional Preparation**

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<th>Years</th>
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<td>Texas A&amp;M University</td>
<td>Postdoc</td>
<td>Biology</td>
<td>2002-2012</td>
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<tr>
<td>Texas A&amp;M University</td>
<td>Ph.D.</td>
<td>Plant Pathology &amp; Microbiology</td>
<td>2003</td>
</tr>
<tr>
<td>Southeast Missouri State University</td>
<td>M.S.</td>
<td>Biology</td>
<td>1994</td>
</tr>
<tr>
<td>Southeast Missouri State University</td>
<td>B.S.</td>
<td>Biology</td>
<td>1990</td>
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**Professional Appointments**

**Texas A&M University**

- **2016-Present**
  - Senior Lecturer
  - Department of Biology

- **2003-2016**
  - Lecturer/Laboratory Instructor (Fall 2011)
  - Department of Biology

- **2002-2012**
  - Post-Doctoral Research Associate
  - Department of Biology
  - Texas A&M University
  - Laboratory of Dr. Terry L. Thomas
  - Functional genomics of *Magnaporthe grisea*
  - Gene regulatory networks of embryonic development in zebrafish

- **1991-1992**
  - Lab Technician
  - Ralston Purina Laboratories, St. Louis, Missouri
  - Analytical Microbiology Department

**Publications**


**Synergistic Activities**

2018. Faculty Fellow. Yale Center for Teaching and Learning Summer Institute on Scientific Teaching.

2018. *Laboratory Course Redesign to Enhance Learning and Improve Student Outcomes in Introductory Biology I & II*. Enhancing the Design of Gateway Experiences. PI. $100,000.


2017. Member, Undergraduate Programs Committee. Department of Biology. Texas A&M University
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>FIELD OF STUDY</th>
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<tr>
<td>Vassar College</td>
<td>B.A.</td>
<td>1976</td>
<td>Biopsychology</td>
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<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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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*
Selected 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
- 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
- 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

- 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
Biographical Sketch

- Texas A&M Student Research Week, Rebecca Rhodes, First Prize in Psychology Research, 2010
• Melbern G. Glasscock Center Second Prize in Humanities for Undergraduate Research, Student Research Week: Elena Kulikova Pritchett, 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.

Biographical Sketch


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.*
Jyotsna Vaid Publications from 2012-2017

BOOK


**GUEST EDITOR FOR SPECIAL ISSUE OF JOURNAL**


**Refereed Journal Articles (student co-authors are indicated with an asterisk)**


Biographical Sketch


**Book Chapters**


Symbol/meaning paired-associate recall: An “archetypal memory” advantage? In Huskinson, Lucy
Biographical Sketch


**BIOGRAPHICAL SKETCH**

**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.)*

<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>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.


**Biographical Sketch**

**Positions and Honors**

**Positions and Employment**

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<tr>
<td>2004-2007</td>
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<td>2007-2010</td>
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<tr>
<td>2010-2011</td>
<td>Senior Research Scientist, Ernest Gallo Clinic and Research Center</td>
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<tr>
<td>2011-2013</td>
<td>Staff Research Investigator, Ernest Gallo Clinic and Research Center</td>
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<tr>
<td>2011-2013</td>
<td>Adjunct Assistant Professor, Department of Neurology, University of California, San Francisco</td>
</tr>
<tr>
<td>2013-2018</td>
<td>Assistant Professor, Department of Neuroscience and Experimental Therapeutics, College of Medicine, Texas A&amp;M University Health Science Center</td>
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**Other Experience and Professional Memberships**

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<td>Membership, Society for Neuroscience</td>
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<td>2002-2006</td>
<td>Membership, Biophysics Society</td>
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<td>2005-2006, 2009-2010</td>
<td>Membership, Research Society on Alcoholism</td>
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**Honors**

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<tr>
<td>1997</td>
<td>First Award of Science and Technology Achievement, Department of Health, Hubei, China</td>
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<tr>
<td>1998</td>
<td>Di-Ao Award, Chinese Academy of Sciences, China</td>
</tr>
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</table>

**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 2012).
2010a), 2) this long-term facilitation is mediated by GluN2B-, but not GluN2A-containing (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 GABAAR-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 GABAARs. How does alcohol alter AMPAR and GABAAR 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 amygdalostrital 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
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 dorsostriatal 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
NAME: Wellman, Paul

eERA 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.)

<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>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>
</tr>
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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.
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.)

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<th>INSTITUTION AND LOCATION</th>
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<th>FIELD OF STUDY</th>
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<td>University of Florida, Gainesville, USA</td>
<td>M.S.</td>
<td>5/1986</td>
<td>Plant Physiology</td>
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<tr>
<td>University of Bremen, Germany</td>
<td>M.S.</td>
<td>10/1986</td>
<td>Biology</td>
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<tr>
<td>University of Bremen, Germany</td>
<td>Ph.D.</td>
<td>12/1999</td>
<td>Cell biology</td>
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<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>
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</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 behavioral 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.


4) Winzer-Serhan UH. Long-term consequences of maternal smoking and developmental chronic nicotine
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
**BIOPGRAPHICAL 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**
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.)

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<td>M.A.</td>
<td>12/2007</td>
<td>Psychology</td>
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<td>University of Texas at Austin</td>
<td>Ph.D.</td>
<td>08/2010</td>
<td>Psychology</td>
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**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*
C. Selected Peer-reviewed Publications


repeat carriers show enhanced attention to high-priority items in the environment.
Nourisoncence, 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
Biographical Sketch


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).
Takashi Yamauchi

**Associate Professor**

**POSITIONS:**
- 2008-Present: Associate professor of psychology at Texas A&M University
- 2008: Assistant professor of psychology at Texas A&M University
- 2000-2001: Visiting assistant professor of psychology at Texas A&M University
- 1998-2000: Research associate at the Center for Interdisciplinary Research on Constructive Learning Environments (supported by the University of Pittsburgh and Carnegie Mellon University).

**EDUCATION:**

**SELECT HONORS/AWARDS:**
- Texas A&M University Faculty Development Leave Award 2013 Faculty Fellow, Race and Ethnic Studies Institute, 2009
- Digital Humanities/Glasscock Center Stipendiary Faculty Fellow 2009 College Faculty Research Enhancement Award 2002
- Fellow, Society for Psychonomic Society, 2005

**GRANTS AS PI, co-PI, or co-I:**
- “Connecting Across Distances: Emotional Support for At-Risk Individuals through Remote Touch” National Science Foundation (co-PI), 2016-2019. Total award of $499,992
- “Foreign Accent Conversion Through Articulatory Inversion of the Vocal-Tract Frontal Cavity” National Science Foundation (senior personnel), 2010-2011. Total award of $210,587

**SELECT PUBLICATIONS (of 59 journal articles, 2 book chapters): Journal articles (psychology)**
Biographical Sketch


**Peer-reviewed conference proceedings (computer science)**


selected as the finalists for the “Best of ACII” (approximately 10 out of 175 submissions).


**SELECT PRESENTATIONS:**
Biographical Sketch


SIGNIFICANT TEACHING ACTIVITIES:

Faculty Advisor
Undergraduate University Scholar

Student Research week awards
1st award (Frankie Lara, 2008, 2009), 1st place Session winner (Frankie Lara, 2009), 1st place Glasscock award (Frankie Lara, 2009).

TAMU Psychology Club and Psi Chi faculty advisor

MAJOR SERVICE ACTIVITIES:

National
National Science Foundation: Innovation Corps Learning Program team mentor (2013-2014)
National Science Foundation / PAC College of Reviewers Board (2013~)

Associate Editor

Guest Editor

Editorial Board
Psychologia
Insights in Psychology
Madridge Journal of Behavioral and Social Sciences Journal
Journal of Brain and Neuroscience Research

University
Glasscock Center for Humanities Research, Advisory Committee member (2010 ~2012)
APPENDIX I

List of Specific Clinical Or In-Service Sites to Support the Proposed Program
Not Applicable
APPENDIX J

Letter of Support from Peer Institutions and/or Area Employers
Date: September 24, 2018

Re: Texas A&M interdisciplinary BS program in Neuroscience

To Whom It May Concern:

The University of Texas at Dallas offers a PhD program in Cognition and Neuroscience, which offers advanced study and research training for students seeking to become leading scientists and scholars in the field. This is a competitive program, seeking to train the best and brightest in the field.

I understand that Texas A&M University is developing an interdisciplinary BS program in Neuroscience, which will provide students with foundational knowledge in the concepts and methodologies employed in the field. Students from this type of program, particularly who have excelled during research experiences with nationally recognized neuroscience scholars, would be highly recruited for positions within our graduate program.

Please feel free to contact me if I can provide any additional information.

Sincerely yours,

Francesca Filbey, PhD
Bert Moore Chair
Associate Provost
Professor and Area Head, Cognition and Neuroscience
School of Behavioral and Brain Sciences
Director, Cognitive Neuroscience of Addictive Behaviors Laboratory
3 October 2018

To Whom It May Concern:

I understand that Texas A&M University is developing an interdisciplinary BS program in Neuroscience, which will provide students with foundational knowledge in the concepts and methodologies employed in the field. Neuroscience is one of the fastest growing and most exciting areas of science and students with undergraduate majors in neuroscience, particularly those who have excelled during relevant research experiences with nationally recognized neuroscience scholars, would be highly recruited for positions within our imaging facility and in other laboratories at Baylor College of Medicine.

Sincerely,

Michael S. Beauchamp, Ph.D.
Academic Director, Core for Advanced MRI
Vice Chair for Basic Research,
Department of Neurosurgery
Professor, Departments of
Neurosurgery and Neuroscience
AGENDA ITEM BRIEFING

Submitted by:  Michael K. Young, President
Texas A&M University

Subject: Approval of a New Bachelor of Science with a Major in Neuroscience Degree Program, and Authorization to Request Approval from the Texas Higher Education Coordinating Board

Proposed Board Action:

Approve the establishment of a new degree program at Texas A&M University (Texas A&M) leading to a Bachelor of Science (B.S.) in Neuroscience, authorize the submission of this degree program to the Texas Higher Education Coordinating Board (THECB) for approval and certify that all applicable THECB criteria have been met.

Background Information:

Neuroscience is the study of the nervous system and its impact on behavior and cognitive functions. This interdisciplinary field integrates several disciplines, including psychology, psychiatry, biology, chemistry, and physics. It is the interdisciplinary nature of neuroscience that requires the participation of multiple units in offering this degree, including the Department of Biology, the Department of Psychological & Brain Sciences, and the College of Veterinary Medicine and Biomedical Sciences, in collaboration with the Department of Neuroscience and Experimental Therapeutics (NExT) in the College of Medicine, as well as the Texas A&M Institute for Neuroscience (TAMIN). The proposed B.S. in Neuroscience will have three concentrations: Molecular & Cellular Neuroscience, Behavioral & Cognitive Neuroscience, and Translational & Preclinical Neuroscience.

Students will develop competency in foundational coursework in the life and physical sciences, including biology, chemistry, and physics. Based on their individual career aspirations and interests, students will complete coursework in neuroscience that involves psychological processes, biological processes, translational issues relevant to medical science and/or pharmacology, neural engineering, and biochemistry. The proposed program will achieve national prominence through the success of its graduates. This will be achieved through structured advising opportunities, opportunities to engage with faculty and students involved in TAMIN, and high impact experiences that involve students in laboratory or field research with faculty and students focused on neuroscience techniques and concepts. Nationwide, there is increasing interest in neuroscience programs and training. In part, this interest is driven by changes in the employment market that focus on technical and medical support jobs. Students completing a B.S. in Neuroscience will be well prepared for graduate study, as well as entry-level healthcare and technical occupations. Unlike neuroscience undergraduate programs housed in a single department or college, the proposed B.S. in Neuroscience is truly interdisciplinary. Students will have well-rounded and in-depth learning experiences regardless of which concentration the students take.
A&M System Funding or Other Financial Implications:

The cost for initially developing and implementing the B.S. in Neuroscience will be minimal because the participating units currently offer courses in neuroscience and additional courses can be accomplished with existing facilities and faculty. As enrollments grow, additional advising and instructional support will be needed. Total new costs for the first five years of the program are estimated at $1,460,000, with estimated funding of $10,714,419.
Members, Board of Regents
The Texas A&M University System

Subject: Approval of a New Bachelor of Science with a Major in Neuroscience Degree Program and Authorization to Request Approval from the Texas Higher Education Coordinating Board

I recommend adoption of the following minute order:

“The Board of Regents of The Texas A&M University System approves the establishment of a new degree program at Texas A&M University leading to a Bachelor of Science in Neuroscience.

The Board also authorizes submission of Texas A&M University’s new degree program request to the Texas Higher Education Coordinating Board for approval and hereby certifies that all applicable criteria of the Coordinating Board have been met.”

Respectfully submitted,

Michael K. Young
President

Approval Recommended: Approved for Legal Sufficiency:

______________________________  ______________________________
John Sharp                   Ray Bonilla
Chancellor                  General Counsel

______________________________  ______________________________
Billy Hamilton               James R. Hallmark, Ph.D.
Deputy Chancellor and        Vice Chancellor for Academic Affairs
Chief Financial Officer
Texas A&M University

Bachelor of Science
with a major in Neuroscience
(CIP 26.1501.00)

Program Review Outline

BACKGROUND & PROGRAM DESCRIPTION

Administrative Unit: Texas A&M University, with concentrations in the College of Science, the College of Liberal Arts, and the College of Veterinary Medicine and Biomedical Sciences

Neuroscience is the study of the nervous system and its impact on behavior and cognitive functions. This interdisciplinary field integrates several disciplines, including psychology, medicine, psychiatry, biology, chemistry, and physics. It is the interdisciplinary nature of neuroscience that requires the participation of multiple units across Texas A&M University (Texas A&M), specifically the Department of Biology, Department of Psychological & Brain Sciences, and the College of Veterinary Medicine and Biomedical Sciences, in collaboration with the College of Medicine and Texas A&M Institute of Neuroscience (TAMIN). Students completing a Bachelor of Science (B.S.) in Neuroscience will be well prepared for graduate study, as well as entry-level healthcare and technical occupations.

The proposed B.S. in Neuroscience will be a cross-college degree and administratively housed at the University level. Initially there will be three available concentrations:

1. Molecular & Cellular Neuroscience (NRSC-MCB), within the Department of Biology in the College of Science;
2. Behavioral & Cognitive Neuroscience (NRSC-BCN), within the Department of Psychological & Brain Science in the College of Liberal Arts; and
3. Translational & Preclinical Neuroscience (NRSC-TLPC), within the (Department of Veterinary Integrative Biosciences) in the College of Veterinary Medicine and Biomedical Sciences, in collaboration with the Department of Neuroscience and Experimental Therapeutics in the College of Medicine.

All students will complete a core set of courses that will provide a foundation of life and physical science course work and a foundational sequence in neuroscience that will prepare students for more advanced courses. Students will also complete a first-year seminar in neuroscience to orient them to the major and their course of study. The recommended core curriculum for this degree was developed by a TAMIN-designated committee with the expertise required in foundational knowledge in neuroscience across disciplines. Each concentration was then developed by committees designated within each respective participating unit. For the NRSC-MCB concentration, students will complete courses focused on biological processes as well as specialized courses focused on molecular and cellular neuroscience. For the NRSC-BCN concentration, students will complete courses focused on behavioral and cognitive neuroscience. For the NRSC-TLPC concentration, students will complete courses focused on translational and preclinical neuroscience. The courses associated with each concentration already exist in the respective departments, and all participating departments include faculty members with expertise
in neuroscience. The development of this interdisciplinary degree will permit students to specialize in programs related to neuroscience and collaborative instruction and research between participating departments is anticipated.

The program will have four learning objectives:

1. Provide students with a broad understanding of basic concepts in the field of neuroscience, with specific advanced knowledge in a subfield (molecular & cellular; behavioral & cognitive; translational & preclinical);
2. Provide students with the ability to explain neuroscience concepts to the lay public;
3. Enhance student understanding of diversity in all forms, including neuro-diversity, neurodevelopment, and individual differences; and
4. Provide students with strong writing and technical skills necessary to communicate and work in fields associated with neuroscience.

The proposed implementation date is fall 2020.

Texas A&M certifies that the proposed new degree program meets the criteria under 19 Texas Administrative Code, Section 5.45 in regards to need, quality, financial and faculty resources, standards and costs. New costs during the first five years will not exceed $2 million.

I. NEED

A. Employment Opportunities

Nationwide, there is increasing interest in neuroscience programs and training. In part, this interest is driven by changes in the employment market. The Bureau of Labor Statistics estimates, for the period of 2014-2024, an increase of 7.4% in demand for life, physical, and social science occupations, and a 16.4% increase in demand for healthcare practitioners and technical occupations. Together, this represents an increase of about 1.5M jobs. Students completing a B.S. in Neuroscience will be well prepared for graduate study, as well as entry-level healthcare and technical occupations.

B. Projected Enrollment

With an expected 75 new students each year, enrollment in the program is projected to reach about 235 to 250 students by year 5. The anticipation is that the neuroscience majors will be largely comprised of students that otherwise would have enrolled in existing programs in the respective departments. As a result, the total number of students served by each department is likely to increase only marginally; the major benefit of this program is that it permits training in a highly employable field.

<table>
<thead>
<tr>
<th></th>
<th>Year 1</th>
<th>Year 2</th>
<th>Year 3</th>
<th>Year 4</th>
<th>Year 5</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Total New Students</strong></td>
<td>75</td>
<td>75</td>
<td>75</td>
<td>75</td>
<td>75</td>
</tr>
<tr>
<td><strong>Attrition</strong></td>
<td>5</td>
<td>5</td>
<td>5</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td><strong>Cumulative Headcount</strong></td>
<td><strong>75</strong></td>
<td><strong>145</strong></td>
<td><strong>190</strong></td>
<td><strong>235</strong></td>
<td><strong>235</strong></td>
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<tr>
<td><strong>FTSE</strong></td>
<td>76</td>
<td>149</td>
<td>188</td>
<td>227</td>
<td>227</td>
</tr>
<tr>
<td><strong>Graduates</strong></td>
<td>0</td>
<td>25</td>
<td>25</td>
<td>70</td>
<td>70</td>
</tr>
</tbody>
</table>
C. Existing State Programs

There are two existing Bachelor of Science in Neuroscience degree programs in the state of Texas, one at The University of Texas at Austin and one at The University of Texas at Dallas. These two programs do not have sufficient capacity to meet the increasing demand for employees in health-related fields. Given the unique strengths of Texas A&M, the proposed neuroscience program offers the additional advantage of building collaborations in research and instructional opportunities with existing strong programs in Engineering and Veterinary Medicine. Students will also have the opportunity to complete courses or research experiences with faculty in the College of Medicine.

II. QUALITY & RESOURCES

A. Faculty

The courses will be taught by the current faculty members in the Department of Veterinary Integrative Biosciences, the Department of Psychological and Brain Sciences, the Department of Biology, and the Department of Neuroscience and Experimental Therapeutics. Given the projected marginal net increase in enrollment, new faculty are not anticipated. However, if total enrollment increases are larger than expected, one additional faculty member may be needed in year 4 to meet demand (estimated salary $150,000 x 2 years). The home department for this additional faculty member will be determined by which neuroscience concentration has the greatest need at the time.

B. Program Administration

The program will be administered as an interdisciplin ary program, with three concentrations (NRSC-MCB, NRSC-BCN, and NRSC-TPC), in collaboration with TAMIN. Participating units will be responsible for the administration of the concentration associated with their unit, including advising, scheduling courses, providing learning and research opportunities, assessing learning outcomes, and encouraging timely graduation. Administration costs include oversight of advertising and developing the program.

C. Other Personnel

Graduate Assistants (TAs) are employed to support writing courses and lab sections, at a ratio of approximately one TA for every 50 majors ($8,400,000 over 5 years). If enrollment rates hit projected levels in years 4 and 5, an academic advisor will be required to support the students, either centralized or assigned within colleges, at salary of approximately $45,000 per advisor.

D. Supplies, Materials

Teaching laboratories will require consumable supplies and materials for program establishment. The cost estimate for these items is $30,000 per year for the first five years of the program.
E. Library & IT Resources

The existing library resources at Texas A&M are sufficient to support the proposed program, and the Library is committed to supporting a new B.S. in Neuroscience degree.

The proposed degree program will require additional computing resources, for neuroimaging work and bioinformatics, at an estimated cost of approximately $30,000 in year 2.

F. Equipment and Facilities

Existing equipment and facilities are sufficient to support the proposed program.

G. Accreditation

The discipline of neuroscience does not have a specific accreditation process or accrediting agency or organization. All degree programs are reviewed every seven years through Texas A&M’s academic program review process.

III. NEW 5 YEAR COSTS & FUNDING SOURCES

<table>
<thead>
<tr>
<th>NEW FIVE-YEAR COSTS</th>
<th>SOURCES OF FUNDING</th>
</tr>
</thead>
<tbody>
<tr>
<td>Faculty (1)</td>
<td>$300,000</td>
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<tr>
<td>Program Administration</td>
<td>$140,000</td>
</tr>
<tr>
<td>Graduate Assistants</td>
<td>$840,000</td>
</tr>
<tr>
<td>Supplies &amp; Materials</td>
<td>$150,000</td>
</tr>
<tr>
<td>Library &amp; IT Resources</td>
<td>$30,000</td>
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<tr>
<td>Equipment, Facilities</td>
<td>$0</td>
</tr>
<tr>
<td><strong>Estimated 5-Year Costs</strong></td>
<td><strong>$ 1,460,000</strong></td>
</tr>
<tr>
<td>Estimated 5-Year Revenues</td>
<td><strong>$10,714,419</strong></td>
</tr>
</tbody>
</table>

The discipline of neuroscience does not have a specific accreditation process or accrediting agency or organization. All degree programs are reviewed every seven years through Texas A&M’s academic program review process.
June 15, 2018

In our Memorandum of Understanding to develop an undergraduate major in Neuroscience (dated 10/25/17), and the Addendum to that MOU (dated 4/10/18), all participating units agreed that any major changes to the degree would be routed to each participating unit and TAIMIN for review and comment. These agreements further stated that any major changes must have the consensus of the heads of all participating units and the chair of TAIMIN. All agreements regarding this degree are available in the CARS system.

This is an interdisciplinary degree, housed at the university level, with concentrations currently proposed in three units. We provide this document as evidence that each participating unit has reviewed the changes detailed below, and has reached consensus with respect to those changes. In this instance, the changes consist of the proposed concentrations.

- Concentration in Translational and Preclinical Neuroscience (BS-NRSC-TPC), administered by the College of Veterinary Medicine and Biomedical Sciences in collaboration with the College of Medicine
- Concentration in Molecular and Cellular Neuroscience (BS-NRSC-MCB), administered by the Department of Biology
- Concentration in Behavioral and Cognitive Neuroscience (BS-NRSC-BCN), administered by the Department of Psychological and Brain Sciences

Elizabeth Crouch  
Associate Dean for Undergraduate Education  
College of Veterinary Medicine & Biomedical Sciences

Thomas D. McKnight  
Head, Department of Biology

Heather Lenc  
Head, Department of Psychological & Brain Sciences

Michael Smothersman  
Chair, Texas A&M Institute of Neuroscience
Degree Evaluation
BS-NRSC-MCB
Molecular & Cellular Neuroscience

Undergraduate Required Areas: 120 hours

Major Coursework: 7.00 credits (Need to add ‘Must make a grade of “C” or better.’ to every rule in this section)
   A. VIBS101 – Neuroscience overview
   B. NRSC/VIBS 277 – Introduction to Neuroscience
   C. NRSC/PSYC 235 – Introduction to Behavioral and Cognitive Neuroscience

Concentration Coursework: (54.00 credits)
   A. CHEM 227
   B. CHEM 237
   C. CHEM 228
   D. CHEM 238
   E. BIOL 213
   F. BIOL 413
   G. BICH 410
   H. BIOL 388
   I. NRSC/Biol 434
   J. NRSC/Biol 435
   K. NRSC/VIBS 450
   L. BICH 411
   M. STAT 302 or STAT 303
   N. Concentration electives (12.00 credits): Select any course NRSC 300-499, or BIOL 430

Communication: 6.00 credits
   A. ENGL 104
   Must have a grade of “C” or better.
   B. Communication 3 credits
   Select from any course with the Communication attribute [KCOM].

Mathematics: 8.00 credits
   A. Select either MATH 147 or 151 and MATH 148 OR MATH 152

Life and Physical Sciences: 24.000 credits
   A. BIOL 111
   Must have a grade of “C” or better
   B. BIOL 112
   Must have a grade of “C” or better
   C. CHEM 119
   D. CHEM 120
   E. PHYS 201
   F. PHYS 202
Language, Philosophy and Culture: 3.00 credits
   A. Language, Philosophy, and Culture 3 hours
      Select any course with the Language, Philosophy and Culture [KLPC] attribute.

Creative Arts: 3.00 credits
   A. Creative Arts 3 hours
      Select any course with the Creative Arts [KCRA] attribute.

Social and Behavioral Science: 3.00 credits
   A. Social and Behavioral
      PSYC 107

Citizenship: 12.00 credits; This is a university area and will be added automatically

General Electives: 7.00 credits; Select any 100-499 course not used elsewhere except AGLS 101;
BIMS 101; BIOL 107, BIOL 113, BIOL 206; BUSN 100; CAEN100-499; CAEX 100-499; CHEM 106,
CHEM 116; HORT 101; MATH 102; STLC 100-499; WFSC 101. Only one KINE 199 can be used as a Free
Elective.

Work Not Applied: This is a university area and will be added automatically

University Writing Req.: 2 courses min. BIOL 351, BIOL 388, BIOL 401, BIOL 423

Int’l & Cultural Discourse: This is a university area and will be added automatically

Foreign Language: For programs that do not require a foreign language area this is the university
approved foreign language area

Residence Requirement – 36hrs of 300-400 level coursework must be completed at TAMU. 12 hrs must
be in major field.: BIOL388, BIOL413, BIOL430; NRSC/BIL434, NRSC/BIL435, NRSC/VIB5450; any
NRSC300-400

GPR – Major: Major GPR 31+ hours
   Includes major and concentration coursework.: VIBS101, NRSC111, NRSC/VIBS277,
NRSC/PSY235, BIOL111, BIOL112, BIOL213, BIOL388, BIOL413, BIOL430, NRSC/BIL434, NRSC/BIL435,
NRSC/VIBS450, any NRSC300-499
Proposal for a New Bachelor’s or Master’s Degree Program

Directions: Texas public institutions of higher education must complete this form to propose: (1) Bachelor’s or Master’s Degree programs in engineering; (2) Bachelor’s or Master’s degree programs that have an estimated cost of more than $2 million in the first five years of operation; and/or (3) Bachelor’s or Master’s degree programs that do not meet the certification requirements set forth in Texas Administrative Code (TAC), Title 19, Chapter 5, Subchapter C, Section 5.44 (a)(3).

Institutions should notify the Division of Academic Quality and Workforce of its intent to plan a new engineering program with a letter submitted through the Document Submission Portal prior to submission of the Proposal for a New Bachelor’s or Master’s Degree Program. The letter should include the title, degree designation, CIP code of the program, the anticipated submission date of the proposal, and a brief description of the program. Address the letter to the Assistant Commissioner of the Academic Division of Academic Quality and Workforce.

In completing the proposal, the institution should refer to the document Standards for Bachelor’s and Master’s Degree Programs, which prescribes specific requirements for new degree programs.

This form requires the signatures of: (1) the Chief Executive Officer, certifying adequacy of funding for the new program, the notification of other Texas public institutions of higher education, and adherence to Texas Education Code (TEC) Sections 61.822 through 61.823; (2) the Chief Financial Officer, certifying the accuracy of funding estimates for the new program; and (3) a member of the Board of Regents (or designee) certifying Board approval.

Contact: Division of Academic Quality and Workforce, 512-427-6200.

Administrative Information

1. Institution Name and Coordinating Board Accountability Group: Texas A&M University

2. Proposed Program:

Bachelor of Science in Neuroscience
With concentrations in
Behavioral & Cognitive Neuroscience
Molecular & Cellular Neuroscience
Translational & Preclinical Neuroscience


4. Semester Credit Hours Required: 120 SCH

5. Location and Delivery of the Proposed Program:
   Face-to-face delivery on the main campus in College Station
6. Administrative Unit:

This is an interdisciplinary degree, and will therefore be housed at the University level with concentrations administered by units.

Bachelor of Science in Neuroscience – Texas A&M University

With concentrations in:

Behavioral & Cognitive Neuroscience – Department of Psychological & Brain Sciences, College of Liberal Arts

Molecular & Cellular Neuroscience – Department of Biology, College of Science

Translational & Preclinical Neuroscience – College of Veterinary Medicine and Biomedical Sciences

7. Program Description:

Neuroscience is the study of the nervous system and its impact on behavior and cognitive functions. This interdisciplinary field integrates several disciplines, including psychology, veterinary and human medicine, psychiatry, biology, chemistry, and physics. It is the interdisciplinary nature of neuroscience that requires the participation of multiple units, specifically the Department of Biology, the Department of Psychological & Brain Sciences, and the College of Veterinary Medicine and Biomedical Sciences. The BS in Neuroscience degree is intended as a cross-college degree, and therefore will be administratively housed at the University level. The core courses for this degree will include a foundation in the life sciences, and a foundational sequence in neuroscience that will prepare students for more advanced courses. They will also complete a first-year seminar in neuroscience to orient them to the major and their course of study. For the concentration of the degree administered by the Department of Biology, students will complete courses focused on biological processes as well as specialized courses focused on molecular and cellular neuroscience. For the concentration of the degree administered by the Department of Psychological & Brain Sciences, students will complete courses focused on behavioral and cognitive neuroscience. For the concentration of the degree administered by the College of Veterinary Medicine and Biomedical Sciences, in collaboration with the Department of Neuroscience and Experimental Therapeutics in the College of Medicine, students will complete courses focused on biomedical, translational, and preclinical neuroscience. The majority of the courses associated with these concentrations already exist in the respective units, as all participating units include faculty members with expertise in neuroscience. The development of this interdisciplinary degree will permit students to specialize in programs related to neuroscience, and we anticipate will foster collaborative instruction and research among participating units.

8. Proposed Implementation Date:

Provide the date that students would enter the proposed program

August 1, 2020.
9. Institutional and Departmental Contacts:

Behavioral & Cognitive Neuroscience:
Name: Heather Lench  
Title: Professor and Head of Psychological & Brain Sciences  
E-mail: hlench@tamu.edu  
Phone: (979) 845-0377

Molecular & Cellular Neuroscience:
Name: Thomas McKnight  
Title: Professor and Head of Biology  
E-mail: mcknight@bio.tamu.edu  
Phone: (979) 845-3896

Translational & Preclinical Neuroscience:
Name: Elizabeth Crouch  
Title: Associate Dean for Undergraduate Education, College of Veterinary Medicine & Biomedical Sciences  
E-mail: ecrouch@cvm.tamu.edu  
Phone: (979) 845-4941

10. Notification to Area Institutions:
Provide a copy of the notification sent to area institutions.
Proposed Bachelor’s or Master’s Degree Program Information

I. Need

A. Job Market Need

Nationwide, there is increasing interest in neuroscience programs and training. In part, this interest is driven by changes in the employment market. The Bureau of Labor Statistics estimates, for the period of 2014-2024, an increase of 7.4% in demand for life, physical, and social science occupations, and a 16.4% increase in demand for healthcare practitioners and technical occupations. Together, this represents an increase of about 1.5M jobs. Given increasing emphasis on the biological bases of mental health disorders and dementia, as well as larger cohorts of older adults, many of these jobs are likely to use the knowledge and skills developed in a neuroscience program. Students completing a BS in Neuroscience will be well prepared for graduate study, as well as entry-level healthcare and technical occupations.

B. Existing Programs

Peer and aspirant peer universities have developed successful undergraduate programs in neuroscience. University of Texas, Austin enrolled 244 students in their neuroscience program in FY2015; University of Texas in Dallas had over 400 neuroscience majors in FY2015. As of Winter 2017, according to their program director, the University of Michigan had about 600 students enrolled in their Neuroscience program that is jointly administered by Psychology and Biology (as well as over 500 students enrolled in the neuroscience program administered solely within Psychology).

There are two existing Bachelor of Science with a major in Neuroscience degree programs in the state of Texas: The University of Texas at Austin and The University of Texas at Dallas. According to the THECB database on graduation rates, the number of graduates from these programs has been increasing over time, at UT Austin from 52 in 2013 to 244 in 2015. These two programs are not sufficient to meet the increasing demand for employment in health-related fields (see increasing job market demands above). Given the unique development of the campus, a neuroscience program at Texas A&M further offers the additional advantage of building collaborations in research and instructional opportunities with existing strong programs in Engineering, Medicine, and Veterinary Medicine.

The majority of the courses associated with the major already exist in the respective units, as all participating units include faculty members with expertise in neuroscience. The development of this interdisciplinary degree will permit students to specialize in programs related to neuroscience, and we anticipate will better serve the career aspirations of students. Establishing a neuroscience major at Texas A&M will help to meet the current and future local and national demand for neuroscience training among students and employers.

C. Student Demand

The data above suggest high, and rapidly growing, enrollments in Neuroscience programs at other institutions. At Texas A&M, the Department of Biology currently serves about 1,500 undergraduate students; the Department of Psychological & Brain Sciences also serves about
1,500 undergraduate students; the College of Veterinary Medicine and Biomedical Sciences serves about 2,400 undergraduate students. An online poll was taken among graduating seniors from the Texas A&M Psychology program in 2016 and nearly a third of those that responded indicated they would have preferred to graduate from a Neuroscience major with a focus on psychological processes if it had been available. This indicates strong interest in the program and a significant group of students that would have been better served by a neuroscience degree. Specifically, students were asked how interested they would have been in a BS in Neuroscience program specializing in psychological processes, if such a degree had been available when they applied to the university, and students rated their interest on a 5-point scale from not at all interested (1) to very interested (5). The proportion above reflects the 39.6% of students who indicated they would have been “very interested” in a BS Neuroscience degree. Similar opinions have been expressed by some of the students majoring in Biomedical Sciences and Biology. Documentation has not been collected to indicate that qualified applicants are leaving Texas for similar programs in other states; however, both the Texas A&M Institute for Neuroscience and the Department of Psychological and Brain Sciences receive multiple email and phone inquiries about these programs every admission cycle. Current undergraduate students have organized and participate in BRAINS (Building Researchers and Innovators in Neuroscience and Society), a student organization focused on neuroscience research, professional development, and outreach.

D. Enrollment Projections

The institution has calculated enrollment projections that reflect student demand estimates to ensure financial self-sufficiency of the program by the end of the program’s fifth year. The estimates below are based on assumptions that about 50 freshmen and 25 new transfers will enroll every year, with a loss of 5 freshmen in year 2; freshmen graduate in four years and transfer students in two years.

<table>
<thead>
<tr>
<th></th>
<th>Year 1</th>
<th>Year 2</th>
<th>Year 3</th>
<th>Year 4</th>
<th>Year 5</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Total New Students</strong></td>
<td>75</td>
<td>75</td>
<td>75</td>
<td>75</td>
<td>75</td>
</tr>
<tr>
<td>Attrition</td>
<td>5</td>
<td>5</td>
<td>5</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td><strong>Cumulative Headcount</strong></td>
<td>75</td>
<td>145</td>
<td>190</td>
<td>235</td>
<td>235</td>
</tr>
<tr>
<td>FTSE</td>
<td>76</td>
<td>149</td>
<td>188</td>
<td>227</td>
<td>227</td>
</tr>
<tr>
<td>Graduates</td>
<td>0</td>
<td>25</td>
<td>25</td>
<td>70</td>
<td>70</td>
</tr>
</tbody>
</table>

E. Student Recruitment

The institution has developed a plan to recruit, retain, and graduate students from underrepresented groups for the program. This will be accomplished through broad advertising of the program to high schools, including events hosted at the schools and regular outreach through advising units associated with each unit. Advertising to current students in associated programs at Texas A&M University will also be comprehensive (e.g., upon approval of the degree program, recently admitted students in the Psychology degree program will be notified through announcements that a Neuroscience program is available that specializes in psychological processes). A first-year seminar will also be implemented as part of this degree to enhance retention and timely progress toward the degree, by incorporating career and degree planning into the seminar. All units use similar strategies for recruitment of students into their current degree programs, including through contact with high schools and community colleges.
that serve students from underrepresented groups, and have been successful in recruiting, retaining, and graduating students, as well as recruiting and graduating students from diverse and underrepresented backgrounds.

II. Quality

A. Degree Requirements

Students will complete 120 hours for the BS in Neuroscience degree program. The courses required in the major include a two-course lower-division sequence (6 credit hours) focused on foundational neuroscience concepts, as well as a freshman orientation course (1 credit hour) that focuses on degree and career options. Students will also complete foundational coursework in life and physical sciences (biology, chemistry, physics). At the upper-division level, students will complete coursework in neuroscience that varies by concentration and permits depth and breadth of study in neuroscience.

This degree structure is similar to neuroscience programs developed at peer and aspirant peer universities, in that there is one interdisciplinary degree that offers specialized areas of study within neuroscience. The specific concentrations vary across universities, depending on the strengths of the participating departments. At University of Michigan, for example, there is an interdisciplinary program focused on molecular and cellular neuroscience (biology & psychology), and a program focused on cognitive and behavioral neuroscience (psychology). At UCLA, as another example, students can choose to concentrate in one of three programs, focused on behavioral and cognitive neuroscience, systems and integrative neuroscience, or molecular cellular and developmental neuroscience. These programs typically include similar foundational neuroscience coursework and life and physical science courses (in biology, physics, chemistry) as included in the proposed Texas A&M program, and then similar advanced coursework in an area of specialization.

Table 2. Semester Credit Hour Requirements by Category

<table>
<thead>
<tr>
<th>Category</th>
<th>Semester Credit Hours</th>
<th>Clock Hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>General Education Core Curriculum (Bachelor’s degree program only)</td>
<td>42</td>
<td></td>
</tr>
<tr>
<td>Required Courses</td>
<td>23 (plus 8 hrs prescribed in core curriculum)</td>
<td></td>
</tr>
<tr>
<td>Prescribed Electives</td>
<td>47-49</td>
<td></td>
</tr>
<tr>
<td>Electives</td>
<td>7-9</td>
<td></td>
</tr>
<tr>
<td>Other (Specify, e.g., internships, clinical work)</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td><strong>120</strong></td>
<td></td>
</tr>
</tbody>
</table>

*Note: Bachelor’s degree programs should not exceed 120 SCHs. Bachelor’s degree programs that exceed 120 SCH must provide detailed documentation describing the compelling academic reason for the number of required hours, such as program accreditation requirements, statutory requirements, and/or licensure/certification requirements that cannot be met without exceeding 120 SCH.*

B. Curriculum

Neuroscience is the study of the nervous system and its impact on behavior and cognitive functions. This interdisciplinary field integrates several disciplines, including psychology, medicine, psychiatry, biology, chemistry, and physics. It is the interdisciplinary nature of
neuroscience that requires the participation of multiple units, specifically the Department of Biology, Department of Psychological & Brain Sciences, and the College of Veterinary Medicine and Biomedical Sciences, in collaboration with the College of Medicine and Texas A&M Institute of Neuroscience. Students completing a BS in Neuroscience will be well prepared for graduate study, as well as entry-level healthcare and technical occupations.

The program will have four learning objectives:

1. To provide students with a broad understanding of basic concepts in the field of neuroscience, with specific advanced knowledge in a subfield (molecular & cellular; behavioral & cognitive; translational & preclinical).
2. To provide students with the ability to explain neuroscience concepts to the lay public.
3. To enhance student understanding of diversity in all forms, including neuro-diversity, neurodevelopment, and individual differences.
4. To provide students with strong writing and technical skills necessary to communicate and work in fields associated with neuroscience.

The core content of the major degree program is similar to neuroscience degree programs developed at peer and aspirant peer institutions. However, students in the proposed program will uniquely benefit, compared to those institutions, from the existing strong programs in Engineering, Medicine, and Veterinary Medicine available at Texas A&M University and represented in elective coursework available for the degree program. These courses offer students the opportunity to develop knowledge and skills relevant to advanced study (e.g., in BioEngineering) or in careers that utilize neuroscience skill sets where students will benefit from cross-discipline knowledge (e.g., bioengineering firms, MRI technician). There is increasing interest locally and nationwide in neuroscience programs and training. The proposed program will achieve national prominence through the success of its graduates. This will be achieved through structured advising opportunities when students are admitted to the program (in a one-hour seminar focused on career and training opportunities), opportunities to engage with faculty and students involved in the Texas A&M Institute for Neuroscience, and high impact experiences that involve students in laboratory or field research with faculty and students focused on neuroscience techniques and concepts.

Students will complete foundational coursework in the life and physical sciences, including biology, chemistry, and physics, which are required for upper-level study in neuroscience and to understand neuroscience techniques. The courses required in the major include a two-course lower-division sequence (6 credit hours) focused on foundational neuroscience concepts, required in all concentrations, as well as a freshman orientation course (1 credit hour) that focuses on degree and career options. At the upper-division level, students will complete coursework in neuroscience that varies by concentration and permits depth and breadth of study in neuroscience. Based on their individual career aspirations and interests, students can complete coursework in neuroscience that involves psychological processes, biological processes, translational issues relevant to medical science and/or pharmacology, neural engineering, and biochemistry. All students will complete a minimum of two courses focused on writing and communicating neuroscience concepts as part of the major.

Courses that are considered equivalent for US colleges and universities will transfer (see full listings at https://compass-ssb.tamu.edu/pls/PROD/bwxkwtes.P_TransEquivMain). Within the major, students can transfer courses that offer similar content and learning objectives as the TAMU courses. Students who complete relevant work experience as part of the degree program
can register for 484 (internship) experiences to include this work in their program of study.

Alternative learning strategies are implemented to accomplish a number of goals that can be summarized as improving learning approaches, decrease time in the classroom and time to degree and increasing college affordability. The basis of this focus on education is to provide increased and improved learning strategies and to measure learning and not necessarily measure time to degree. The critical aspect of using alternative learning strategies in higher education is to measure competence or mastery of knowledge and skills (competencies). The assumption is that most (but perhaps not all) students may be able to gain necessary competencies in less time than the time devoted to traditional, didactic, semester-long courses. The goal of such educational practices is to reduce the number of time students spend sitting in a classroom listening to lectures and more time in situations that create flexibility in learning modalities and allows students to demonstrate mastery of academic content at a pace that suits their learning needs and style. Alternative learning strategies currently used in the units proposing this major include: 1) Some courses are designed to be modular – students can take one, two or three modules for 3 or 4 weeks during a semester for one or two credits each. 2) Online courses, 3) Some courses have significant individualized instruction, field trips, observations, and experiments, 4) Study abroad courses, 5) Peer teaching takes place in some classes, 6) Writing intensive assignments in courses not designated as writing courses, 7) Group projects and group learning exercises, 8) Interactive and instant learning outcome assessment in the classroom through use of audience response systems. We will continue to develop competency-based learning opportunities, including further use of case studies, simulations, and games. A critical aspect of increasing alternative learning strategies is increased use of state-of-the-art educational technologies. Use of computer-based instruction is instrumental in individualized learning and allowing students to learn outside of a classroom setting.

Table 3. Required/Core Courses

<table>
<thead>
<tr>
<th>Prefix and Number</th>
<th>Required/Core Course Title</th>
<th>SCH</th>
</tr>
</thead>
<tbody>
<tr>
<td>NRSC/VIBS 101*</td>
<td>Neuroscience 101</td>
<td>1</td>
</tr>
<tr>
<td>NRSC/VIBS 277</td>
<td>Introduction to Neuroscience</td>
<td>3</td>
</tr>
<tr>
<td>NRSC/PSYC 235</td>
<td>Introduction to Behavioral &amp; Cognitive Neuroscience</td>
<td>3</td>
</tr>
</tbody>
</table>

Prescribed Life and Physical Sciences Core Courses

<table>
<thead>
<tr>
<th>Course Code</th>
<th>Course Title</th>
<th>Credits</th>
</tr>
</thead>
<tbody>
<tr>
<td>BIOL 111</td>
<td>Introductory Biology I</td>
<td>4</td>
</tr>
<tr>
<td>BIOL 112</td>
<td>Introductory Biology II</td>
<td>4</td>
</tr>
<tr>
<td>CHEM 119</td>
<td>Fundamentals of Chemistry I</td>
<td>4</td>
</tr>
<tr>
<td>CHEM 120</td>
<td>Fundamentals of Chemistry II</td>
<td>4</td>
</tr>
<tr>
<td>PHYS 201 or 208</td>
<td>College Physics</td>
<td>4</td>
</tr>
<tr>
<td>PHYS 202 or 218</td>
<td>College Physics II</td>
<td>4</td>
</tr>
</tbody>
</table>
## CONCENTRATION IN BEHAVIORAL AND COGNITIVE NEUROSCIENCE (NRSC-BCN)

### Table 4. Prescribed Elective Courses

<table>
<thead>
<tr>
<th>Prefix and Number</th>
<th>Prescribed Elective Course Title</th>
<th>SCH</th>
</tr>
</thead>
<tbody>
<tr>
<td>STAT 302</td>
<td>Statistical Methods</td>
<td>3</td>
</tr>
</tbody>
</table>

**Prescribed elective – Concentration coursework (15 SCH)**

<table>
<thead>
<tr>
<th>Prefix and Number</th>
<th>Prescribed Elective Course Title</th>
<th>SCH</th>
</tr>
</thead>
<tbody>
<tr>
<td>NRSC/PSYC 320</td>
<td>Sensation - Perception</td>
<td>3</td>
</tr>
<tr>
<td>NRSC/PSYC 332</td>
<td>Neuroscience of Learning and Memory</td>
<td>3</td>
</tr>
<tr>
<td>NRSC/PSYC 333</td>
<td>Biology of Psychological Disorders</td>
<td>3</td>
</tr>
<tr>
<td>NRSC/PSYC 350</td>
<td>Cognitive Neuroscience</td>
<td>3</td>
</tr>
<tr>
<td>NRSC/PSYC 340</td>
<td>Psychology of Learning</td>
<td>3</td>
</tr>
<tr>
<td>NRSC/PSYC 360</td>
<td>Health Psychology &amp; Behavioral Medicine</td>
<td>3</td>
</tr>
<tr>
<td>NRSC/PSYC 311</td>
<td>Psychology of Animal Behavior</td>
<td>3</td>
</tr>
<tr>
<td>NRSC/PSYC 336</td>
<td>Drugs and Behavior</td>
<td>3</td>
</tr>
</tbody>
</table>

**Prescribed elective – concentration elective coursework (29 SCH)**

<table>
<thead>
<tr>
<th>Prefix and Number</th>
<th>Required/Core Course Title</th>
<th>SCH</th>
</tr>
</thead>
<tbody>
<tr>
<td>NRSC 300-499</td>
<td>Any courses in Neuroscience (including courses not used to fulfill requirements above).</td>
<td></td>
</tr>
<tr>
<td>PSYC 484</td>
<td>Field Experiences</td>
<td>Varies</td>
</tr>
<tr>
<td>PSYC 485</td>
<td>Directed Studies</td>
<td>Varies</td>
</tr>
<tr>
<td>PSYC 491</td>
<td>Research</td>
<td>Varies</td>
</tr>
<tr>
<td>PSYC 471*</td>
<td>Research Writing in Neuroscience</td>
<td>1</td>
</tr>
<tr>
<td>PSYC 475*</td>
<td>Communicating Neuroscience Concepts</td>
<td>1</td>
</tr>
<tr>
<td>CHEM 227</td>
<td>Organic Chemistry I</td>
<td>3</td>
</tr>
<tr>
<td>CHEM 237</td>
<td>Organic Chemistry I Lab</td>
<td>1</td>
</tr>
<tr>
<td>CHEM 228</td>
<td>Organic Chemistry II</td>
<td>3</td>
</tr>
<tr>
<td>CHEM 238</td>
<td>Organic Chemistry II Lab</td>
<td>1</td>
</tr>
<tr>
<td>BIOL 213</td>
<td>Molecular and Cellular Biology</td>
<td>3</td>
</tr>
<tr>
<td>BIOL 413</td>
<td>Cell Biology</td>
<td>3</td>
</tr>
<tr>
<td>BICH 410</td>
<td>Comprehensive Biochemistry I</td>
<td>3</td>
</tr>
</tbody>
</table>

### Table 5. Elective Courses

<table>
<thead>
<tr>
<th>Prefix and Number</th>
<th>Required/Core Course Title</th>
<th>SCH</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Select any 100-499 course not used elsewhere.</td>
<td>9</td>
</tr>
</tbody>
</table>

## CONCENTRATION IN MOLECULAR AND CELLULAR NEUROSCIENCE (NRSC-MCB)

### Table 4. Prescribed Elective Courses

<table>
<thead>
<tr>
<th>Prefix and Number</th>
<th>Prescribed Elective Course Title</th>
<th>SCH</th>
</tr>
</thead>
<tbody>
<tr>
<td>STAT 302</td>
<td>Statistical Methods</td>
<td>3</td>
</tr>
<tr>
<td>CHEM 227</td>
<td>Organic Chemistry I</td>
<td>3</td>
</tr>
<tr>
<td>CHEM 237</td>
<td>Organic Chemistry I Lab</td>
<td>1</td>
</tr>
<tr>
<td>CHEM 228</td>
<td>Organic Chemistry II</td>
<td>3</td>
</tr>
<tr>
<td>CHEM 238</td>
<td>Organic Chemistry II Lab</td>
<td>1</td>
</tr>
</tbody>
</table>
Proposal for a New Bachelor’s or Master’s Degree Program

<table>
<thead>
<tr>
<th>Prefix and Number</th>
<th>Required/Core Course Title</th>
<th>SCH</th>
</tr>
</thead>
<tbody>
<tr>
<td>BIOL 213</td>
<td>Molecular and Cellular Biology</td>
<td>3</td>
</tr>
<tr>
<td>BIOL 413</td>
<td>Cell Biology</td>
<td>3</td>
</tr>
<tr>
<td>BICH 410</td>
<td>Comprehensive Biochemistry I</td>
<td>3</td>
</tr>
</tbody>
</table>

**Prescribed elective – Concentration coursework (15 SCH)**

<table>
<thead>
<tr>
<th>Prefix and Number</th>
<th>Required/Core Course Title</th>
<th>SCH</th>
</tr>
</thead>
<tbody>
<tr>
<td>BIOL 388</td>
<td>Principles of Animal Physiology</td>
<td>4</td>
</tr>
<tr>
<td>NRSC/BIOL 434</td>
<td>Regulatory and Behavioral Neuroscience</td>
<td>3</td>
</tr>
<tr>
<td>BIOL 435</td>
<td>Laboratory for Regulatory and Behavioral Neuroscience</td>
<td>1</td>
</tr>
<tr>
<td>NRSC/VIBS 450</td>
<td>Functional Neuroanatomy</td>
<td>4</td>
</tr>
<tr>
<td>BICH 411</td>
<td>Comprehensive Biochemistry II</td>
<td>3</td>
</tr>
</tbody>
</table>

**Prescribed elective – concentration elective coursework (12 SCH)**

<table>
<thead>
<tr>
<th>Prefix and Number</th>
<th>Required/Core Course Title</th>
<th>SCH</th>
</tr>
</thead>
<tbody>
<tr>
<td>NRSC 300-499</td>
<td>Any courses in Neuroscience (including courses not used to fulfill requirements above).</td>
<td>0-12</td>
</tr>
<tr>
<td>BIOL 430</td>
<td>Any courses in Biology not used to fulfill requirements above</td>
<td>0-12</td>
</tr>
<tr>
<td>GENE/BICH 302</td>
<td>Principles of Genetics</td>
<td>3</td>
</tr>
<tr>
<td>GENE/BICH 431</td>
<td>Molecular Genetics</td>
<td>3</td>
</tr>
</tbody>
</table>

**Table 5. Elective Courses**

<table>
<thead>
<tr>
<th>Prefix and Number</th>
<th>Required/Core Course Title</th>
<th>SCH</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Select any 100-499 course not used elsewhere.</td>
<td>7</td>
</tr>
</tbody>
</table>

**CONCENTRATION IN TRANSLATIONAL & PRECLINICAL NEUROSCIENCE (NRSC-TPC)**

**Table 4. Prescribed Elective Courses**

<table>
<thead>
<tr>
<th>Prefix and Number</th>
<th>Prescribed Elective Course Title</th>
<th>SCH</th>
</tr>
</thead>
<tbody>
<tr>
<td>STAT 302</td>
<td>Statistical Methods</td>
<td>3</td>
</tr>
<tr>
<td>CHEM 227</td>
<td>Organic Chemistry I</td>
<td>3</td>
</tr>
<tr>
<td>CHEM 237</td>
<td>Organic Chemistry I Lab</td>
<td>1</td>
</tr>
<tr>
<td>CHEM 228</td>
<td>Organic Chemistry II</td>
<td>3</td>
</tr>
<tr>
<td>CHEM 238</td>
<td>Organic Chemistry II Lab</td>
<td>1</td>
</tr>
<tr>
<td>BIOL 213</td>
<td>Molecular and Cellular Biology</td>
<td>3</td>
</tr>
<tr>
<td>BICH 410</td>
<td>Comprehensive Biochemistry I</td>
<td>3</td>
</tr>
<tr>
<td>BICH 411</td>
<td>Comprehensive Biochemistry II</td>
<td>3</td>
</tr>
<tr>
<td>GENE 302 or GENE/BIMS 320</td>
<td>Principles of Genetics or Biomedical Genetics</td>
<td>3</td>
</tr>
</tbody>
</table>

**Prescribed elective – Concentration coursework (14 SCH)**

<table>
<thead>
<tr>
<th>Prefix and Number</th>
<th>Required/Core Course Title</th>
<th>SCH</th>
</tr>
</thead>
<tbody>
<tr>
<td>PSYC 107</td>
<td>Introduction to Psychology</td>
<td>3</td>
</tr>
<tr>
<td>NRSC/BIOL 434</td>
<td>Regulatory and Behavioral Neuroscience</td>
<td>3</td>
</tr>
<tr>
<td>BIOL 435</td>
<td>Laboratory for Regulatory and Behavioral Neuroscience</td>
<td>1</td>
</tr>
<tr>
<td>NRSC/VIBS 450</td>
<td>Functional Neuroanatomy</td>
<td>4</td>
</tr>
<tr>
<td>PSYC 300-499</td>
<td>Any course in Psychology or</td>
<td>3</td>
</tr>
<tr>
<td>Or NRSC 300-499</td>
<td>Any course in neuroscience (NRSC)</td>
<td></td>
</tr>
</tbody>
</table>
Proposal for a New Bachelor’s or Master’s Degree Program

Table 5. Elective Courses

<table>
<thead>
<tr>
<th>Prefix and Number</th>
<th>Required/Core Course Title</th>
<th>SCH</th>
</tr>
</thead>
<tbody>
<tr>
<td>NRSC/VIBS 401</td>
<td>Developmental Neurotoxicology</td>
<td>2</td>
</tr>
<tr>
<td>NRSC/VIBS 407</td>
<td>Core Ideas in Neuroscience</td>
<td>2</td>
</tr>
<tr>
<td>VIBS 408</td>
<td>Neuroscience and Religion</td>
<td>3</td>
</tr>
<tr>
<td>VIBS 447</td>
<td>Neurophysiology of Music</td>
<td>2</td>
</tr>
<tr>
<td>VIBS 606/NRSC 605</td>
<td>Neuroanatomical Systems (VIBS 4xx stacked)</td>
<td>3</td>
</tr>
<tr>
<td>VIBS 424/VTTP 424</td>
<td>Biomedical Neuroendocrinology and Endocrine Disorders</td>
<td>3</td>
</tr>
<tr>
<td>VIBS 422</td>
<td>Endocrine Toxicology</td>
<td>4</td>
</tr>
<tr>
<td>VIBS 343</td>
<td>Histology</td>
<td>4</td>
</tr>
<tr>
<td>VIBS 443</td>
<td>Biology of Mammalian Cells and Tissues</td>
<td>4</td>
</tr>
<tr>
<td>KINE 406</td>
<td>Motor Learning and Skill Performance</td>
<td>3</td>
</tr>
<tr>
<td>VTPP 323</td>
<td>Physiology of Domestic Animals</td>
<td>3</td>
</tr>
<tr>
<td>VTPP 425</td>
<td>Pharmacology</td>
<td>3</td>
</tr>
<tr>
<td>VIBS/NRSC 640</td>
<td>Neurobiology (VIBS 4xx stacked)</td>
<td>3</td>
</tr>
<tr>
<td>VTMI 662</td>
<td>Advanced Immunologic Concepts (VTMI 4xx stacked)</td>
<td>3</td>
</tr>
<tr>
<td>NRSC 485</td>
<td>Directed Studies</td>
<td>Varies</td>
</tr>
<tr>
<td>NRSC 491</td>
<td>Research</td>
<td>Varies</td>
</tr>
<tr>
<td></td>
<td>Select any 100-499 course not used elsewhere.</td>
<td>8</td>
</tr>
</tbody>
</table>

C. Strategic Plan and Marketable Skills

The updated Texas A&M University strategic plan, An Ideal 21st Century University, is available at: [http://provost.tamu.edu/Provost/media/Assets/pdfs-strategicplan/FINALSTRATPLAN.pdf](http://provost.tamu.edu/Provost/media/Assets/pdfs-strategicplan/FINALSTRATPLAN.pdf)

The proposed program is an interdisciplinary degree that involves three primary units, as well as courses from additional departments and colleges. This is consistent with the University’s strategy of strengthening multidisciplinary programs and initiatives, while aligning them with existing disciplinary structures. The program is also consistent with the overall goal of offering exceptional undergraduate education that reflects the changing demographics of the state and nation. As noted above, there is increasing demand in the job market for students who have neuroscience backgrounds, at both the undergraduate and graduate level. Therefore graduates of the proposed program are likely to be highly sought after and to fulfill critically needed job positions within the state and nation. Graduates of this program will also be well prepared to continue their education in graduate and professional schools.

Consistent with the University strategy of career preparation and appropriate learning outcomes, the proposed program involves targeted learning outcomes that will be regularly assessed and improved upon, career development opportunities and advising, and high impact learning opportunities that involve neuroscience concepts and techniques.

The proposed program aligns with the state’s 60x30TX plan. As noted above, there is increasing
demand locally and nationally for graduates with knowledge and skills related to neuroscience training. Providing this educational opportunity to students within the state increases the likelihood that students will pursue employment within the state after graduation. Further, the proposed program aligns with current and projected workforce demands, as well as evidence of existing student demand within Texas A&M University, as survey results indicate nearly 500 students per year in the Psychology program would be arguably better served by a Neuroscience degree. Texas A&M University, and the participating units, have all committed to recruiting and retaining students from diverse backgrounds, timely graduation, and low student debt. The proposed program will also align with these university and state goals by assessing and eliminating roadblocks to student success and progression, engaging students in career and course planning and advising early in their academic careers, active outreach to high schools and community college programs that serve students from underrepresented backgrounds, and striving to keep tuition rates and other educational expenses low for students. Students will attain marketable skills as part of the proposed program, and these skills will be communicated to students in program materials, in the foundational courses associated with the major, and as students complete advising appointments shortly before graduation. These skills include: 1) **scientific skills**: develop solutions for problems using scientific concepts from anatomy, biology, chemistry, and physics, 2) **scientific communication**: express ideas, including oral and written communication, to convey scientific information and concepts to experts and non-specialists, 3) **critical thinking and research skills**: use quantitative reasoning and data analytic skills to identify problems, develop a research-oriented approach to address the problem, and incorporate numerical information to identify solutions, 4) **appreciation for diversity**: evaluate and understand individual differences and diversity, including neurodiversity, to analyze organizational and training problems and policies, 5) **initiative and decision making ability**: seek out information and innovative solutions and use work to identify viable solutions.

Texas A&M University has multiple units and faculty who are nationally and internationally renowned for their scientific contributions to neuroscience. The proposed program will provide an opportunity for undergraduate students to benefit from this expertise and knowledge, while developing marketable skills that are in demand locally and nationally. The participating units (Biology, Psychological & Brain Sciences; College of Veterinary Medicine) have existing strengths in undergraduate education, and the proposed program will leverage these existing strengths to offer an innovative interdisciplinary program to our students.

**D. Faculty**

The percent time in the table below is based on expected enrollments associated with the BS Neuroscience program. The percentage of time for faculty is on the lower side because 1) these are courses that our faculty members already teach, meaning that Neuroscience majors will constitute a portion of the students enrolled in the course, and 2) there are multiple professors that can teach each course, meaning that the faculty members will not teach every course every semester. The courses assigned are also courses that support the existing BA/BS programs and a Neuroscience minor program. All of the faculty have earned their PhDs in related disciplines and have experience in teaching these courses and mentoring undergraduate and graduate students in research experiences in neuroscience. Supporting faculty are those who will primarily contribute through offering research experiences to Neuroscience majors.

The new program includes core curriculum courses in biology, chemistry, math, political science, and English, which are regularly offered at high enrollment numbers to support existing programs. We anticipate that some new students will be attracted to the Neuroscience program
that otherwise would have studied at another university; but the majority of the students will be students who otherwise would have enrolled in another major. Therefore the impact on other departments should be minimal as the overall number of students will increase only marginally. The benefit to students is participation in a degree that better aligns with their interests and career aspirations. The courses in this degree are already offered as part of the curriculum for existing degrees in the participating units. Therefore, the impact of the new degree program will be to change the frequency with which courses are taught and a likely increased enrollment in existing neuroscience courses.

Across concentrations, we project the need for an additional faculty member in Year 4 and Year 5 of the program, with the specific area to be determined by student enrollment across the concentrations, or targeting an interdisciplinary neuroscience hire.

**CONCENTRATION IN BEHAVIORAL AND COGNITIVE NEUROSCIENCE (NRSC-BCN)**

**Table 5. Core Faculty**

<table>
<thead>
<tr>
<th>Name and Rank of Core Faculty</th>
<th>Highest Degree and Awarding Institution</th>
<th>Courses Assigned in Program</th>
<th>% Time Assigned to Program</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brian Anderson, Assistant Professor</td>
<td>PhD in Psychological &amp; Brain Sciences, Johns Hopkins University</td>
<td>PSYC/NRSC 235, PSYC/NRSC 320</td>
<td>8%</td>
</tr>
<tr>
<td>Joseph Orr, Assistant Professor</td>
<td>PhD in Psychology, University of Michigan</td>
<td>PSYC/NRSC 350, PSYC/NRSC 235</td>
<td>8%</td>
</tr>
<tr>
<td>Annmarie MacNamara, Assistant Professor</td>
<td>PhD in Clinical Psychology, Stony Brook University</td>
<td>PSYC/NRSC 350, PSYC/NRSC 235</td>
<td>8%</td>
</tr>
<tr>
<td>Jessica Bernard, Assistant Professor</td>
<td>PhD in Psychology, University of Michigan</td>
<td>PSYC/NRSC 235</td>
<td>8%</td>
</tr>
<tr>
<td>Vani Mathur, Assistant Professor</td>
<td>PhD in Psychology, Northwestern University</td>
<td>PSYC/NRSC 360</td>
<td>8%</td>
</tr>
<tr>
<td>James Grau, Professor</td>
<td>PhD in Psychology, University of Pennsylvania</td>
<td>PSYC/NRSC 340</td>
<td>8%</td>
</tr>
<tr>
<td>Shoshana Eitan, Associate Professor</td>
<td>PhD in Neurobiology, Weizmann Institute of Science</td>
<td>PSYC/NRSC 333, PSYC/NRSC 335</td>
<td>8%</td>
</tr>
<tr>
<td>Mark Packard, Professor</td>
<td>PhD in Experimental Psychology, McGill University</td>
<td>PSYC/NRSC 335</td>
<td>8%</td>
</tr>
<tr>
<td>Justin Moscarello, Assistant Professor</td>
<td>PhD in Psychology, University of California, Santa Barbara</td>
<td>PSYC/NRSC 332</td>
<td>8%</td>
</tr>
<tr>
<td>Steve Maren, Distinguished Professor</td>
<td>PhD in Biological Sciences, University of Southern California</td>
<td>PSYC/NRSC 235, PSYC/NRSC 332</td>
<td>8%</td>
</tr>
<tr>
<td>Carlos Bolanos, Associate Professor</td>
<td>PhD in Experimental Psychology, Northeastern University</td>
<td>PSYC/NRSC 235</td>
<td>8%</td>
</tr>
<tr>
<td>Name and Rank of Core Faculty</td>
<td>Highest Degree and Awarding Institution</td>
<td>Courses Assigned in Program</td>
<td>% Time Assigned to Program</td>
</tr>
<tr>
<td>------------------------------</td>
<td>----------------------------------------</td>
<td>-----------------------------</td>
<td>---------------------------</td>
</tr>
<tr>
<td>Mary Meagher, Professor</td>
<td>PhD in Experimental and Biological Psychology, University of North Carolina at Chapel Hill</td>
<td>PSYC/NRSC 360</td>
<td>8%</td>
</tr>
<tr>
<td>Naomi Nagaya, Research Assistant Professor</td>
<td>PhD in Biological Sciences, University of Southern California</td>
<td>PSYC/NRSC 311</td>
<td>8%</td>
</tr>
<tr>
<td>Rachel Smith, Assistant Professor</td>
<td>PhD in Neuroscience, University of Pennsylvania</td>
<td>PSYC/NRSC 332</td>
<td>8%</td>
</tr>
</tbody>
</table>

**CONCENTRATION IN MOLECULAR & CELLULAR NEUROSCIENCE (NRSC-MCB)**

Table 5. Core Faculty

<table>
<thead>
<tr>
<th>Name and Rank of Core Faculty</th>
<th>Highest Degree and Awarding Institution</th>
<th>Courses Assigned in Program</th>
<th>% Time Assigned to Program</th>
</tr>
</thead>
<tbody>
<tr>
<td>U.J. McMahan Professor</td>
<td>PhD in Anatomy, University of Tennessee Medical School</td>
<td>BIOL/NRSC 434</td>
<td>8%</td>
</tr>
<tr>
<td>Wesley Thompson Professor</td>
<td>PhD in Molecular Biology, UC Berkeley</td>
<td>BIOL/NRSC 401</td>
<td>8%</td>
</tr>
<tr>
<td>Paul Hardin Distinguished Professor</td>
<td>PhD in Genetics, Indiana University</td>
<td>BIOL 213</td>
<td>8%</td>
</tr>
<tr>
<td>Rene Garcia Professor</td>
<td>PhD in Microbiology, University of Texas at Austin</td>
<td>BIOL 413</td>
<td>8%</td>
</tr>
<tr>
<td>Steve Lockless Associate Professor</td>
<td>PhD in Molecular Biophysics, UT Southwestern Medical School</td>
<td>BIOL 213</td>
<td>8%</td>
</tr>
<tr>
<td>Michael Smotherman Associate Professor</td>
<td>PhD in Physiological Science, UCLA</td>
<td>BIOL/NRSC 434 &amp; 435</td>
<td>8%</td>
</tr>
<tr>
<td>Mark Zoran Professor</td>
<td>PhD in Zoology, Iowa State University</td>
<td>BIOL 388</td>
<td>8%</td>
</tr>
<tr>
<td>Larry Griffing Associate Professor</td>
<td>PhD in Biology, Stanford</td>
<td>BIOL 430</td>
<td>8%</td>
</tr>
<tr>
<td>Jennifer Dulin Assistant Professor</td>
<td>PhD in Neuroscience, UT Health Science Center, Houston</td>
<td>BIOL/NRSC 489</td>
<td>8%</td>
</tr>
</tbody>
</table>
CONCENTRATION IN TRANSLATIONAL & PRECLINICAL NEUROSCIENCE (NRSC-TLPC)

Table 5. Core Faculty

<table>
<thead>
<tr>
<th>Name and Rank of Core Faculty</th>
<th>Highest Degree and Awarding Institution</th>
<th>Courses Assigned in Program</th>
<th>% Time Assigned to Program</th>
</tr>
</thead>
<tbody>
<tr>
<td>C. Jane Welsh, Professor</td>
<td>PhD in Biochemistry, London University</td>
<td>VTMI 662 VIBS 606</td>
<td>8%</td>
</tr>
<tr>
<td>Gladys Ko, Associate Professor</td>
<td>PhD in Neuroscience in Biomedical Sciences, Kent State University</td>
<td>NRSC/VIBS 450 NRSC/VIBS 277 VIBS 289</td>
<td>12%</td>
</tr>
<tr>
<td>Jianrong Li, Associate Professor</td>
<td>PhD in Biochemistry, University of Hawaii</td>
<td>VIBS 640</td>
<td>4%</td>
</tr>
<tr>
<td>Joe Arosh, Professor</td>
<td>PhD in Reproductive Endocrinology, Laval University</td>
<td>VIBS 424</td>
<td>4%</td>
</tr>
<tr>
<td>Sakhila Banu, Associate Professor</td>
<td>PhD in Endocrinology, University of Madras</td>
<td>VIBS 422</td>
<td>4%</td>
</tr>
<tr>
<td>Peter Nghiem, Assistant Professor</td>
<td>PhD in Molecular Medicine, The George Washington University and DVM, Texas A&amp;M University</td>
<td>VIBS 289</td>
<td>4%</td>
</tr>
<tr>
<td>Micah Waltz, Lecturer</td>
<td>MS in Integrative Physiology, West Virginia University</td>
<td>VIBS 447</td>
<td>4%</td>
</tr>
<tr>
<td>William Klemm, Senior Professor</td>
<td>PhD in Biology, Notre Dame and DVM, Auburn</td>
<td>VIBS 407 VIBS 408 VIBS 489</td>
<td>12%</td>
</tr>
</tbody>
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CONCENTRATION IN BEHAVIORAL AND COGNITIVE NEUROSCIENCE (NRSC-BCN)

Table 6. Support Faculty

<table>
<thead>
<tr>
<th>Name and Rank of Support Faculty</th>
<th>Highest Degree and Awarding Institution</th>
<th>Courses Assigned in Program or Other Support Activity</th>
<th>% Time Assigned to Program</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paul Wellman, Professor</td>
<td>PhD in Psychology, Iowa State University</td>
<td>Research experiences</td>
<td>2%</td>
</tr>
<tr>
<td>Darrell Worthy, Associate Professor</td>
<td>PhD in Psychology, University of Texas at Austin</td>
<td>Research experiences</td>
<td>2%</td>
</tr>
<tr>
<td>Jyotsna Vaid, Professor</td>
<td>PhD in Experimental Psychology, McGill University</td>
<td>Research experiences</td>
<td>2%</td>
</tr>
</tbody>
</table>
Gerianne Alexander, Professor  
PhD in Clinical Psychology, McGill University  
Research experiences 2%

Rebecca Brooker, Assistant Professor  
PhD in Developmental Psychology, The Pennsylvania State University  
Research experiences 2%

Shereece Fields, Associate Professor  
PhD in Clinical Psychology, University of South Florida  
Research experiences 2%

Brandon Schmeichel, Professor  
PhD in Social Psychology, Florida State University  
Research experiences 2%

Takashi Yamauchi, Associate Professor  
PhD in Psychology, Columbia University  
Research experiences 2%

**CONCENTRATION IN MOLECULAR AND CELLULAR NEUROSCIENCE (NRSC-MCB)**

**Table 6. Support Faculty**

<table>
<thead>
<tr>
<th>Name and Rank of Support Faculty</th>
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<th>Courses Assigned in Program or Other Support Activity</th>
<th>% Time Assigned to Program</th>
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</thead>
<tbody>
<tr>
<td>Bruce Riley, Professor</td>
<td>PhD in Molecular Biology, University of Wisconsin</td>
<td>Research Experiences</td>
<td>2%</td>
</tr>
<tr>
<td>W. Michael Kemp, Professor</td>
<td>PhD in Biology, Tulane</td>
<td>BIOL 111</td>
<td>2%</td>
</tr>
<tr>
<td>James Erickson, Associate Professor</td>
<td>PhD in Bacteriology, University of Wisconsin</td>
<td>BIOL 111</td>
<td>2%</td>
</tr>
<tr>
<td>Hongmin Qin, Associate Professor</td>
<td>PhD in Genetics, Chinese Academy of Sciences</td>
<td>BIOL 413</td>
<td>2%</td>
</tr>
<tr>
<td>David Earnest, Professor</td>
<td>PhD in Neurobiology, Northwestern University</td>
<td>Research Experiences</td>
<td>2%</td>
</tr>
<tr>
<td>Gil Rosenthal, Professor</td>
<td>PhD in Zoology, University of Texas at Austin</td>
<td>Research Experiences</td>
<td>2%</td>
</tr>
<tr>
<td>Wayne Versaw, Associate Professor</td>
<td>PhD in Biomolecular Chemistry, University of Wisconsin</td>
<td>BIOL 413</td>
<td>2%</td>
</tr>
<tr>
<td>Andrew Tag, Senior Lecturer</td>
<td>PhD in Plant Pathology and Microbiology, Texas A&amp;M University</td>
<td>BIOL 111 &amp; 112</td>
<td>2%</td>
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</tbody>
</table>
CONCENTRATION IN TRANSLATIONAL & PRECLINICAL NEUROSCIENCE (NRSC-TLPC)

Table 6. Support Faculty

<table>
<thead>
<tr>
<th>Name and Rank of Support Faculty</th>
<th>Highest Degree and Awarding Institution</th>
<th>Courses Assigned in Program or Other Support Activity</th>
<th>% Time Assigned to Program</th>
</tr>
</thead>
<tbody>
<tr>
<td>John Buchanan, Professor</td>
<td>PhD in Brain Science, Florida Atlantic University</td>
<td>KINE 406</td>
<td>4%</td>
</tr>
<tr>
<td>Jonathan Levine, Professor</td>
<td>DVM, Cornell University Diploma ACVIM (Neurology)</td>
<td>Research experiences</td>
<td>2%</td>
</tr>
<tr>
<td>Joe Kornegay, Professor</td>
<td>PhD in Veterinary Pathology, University of Georgia and DVM, Texas A&amp;M University</td>
<td>Research experiences</td>
<td>2%</td>
</tr>
<tr>
<td>C. Elizabeth Boudreau, Assistant Professor</td>
<td>PhD in Neuroscience, Baylor College of Medicine and DVM, Texas A&amp;M University Diploma ACVIM (Neurology)</td>
<td>Research experiences</td>
<td>2%</td>
</tr>
<tr>
<td>Larry Johnson, Professor</td>
<td>PhD in Reproductive Physiology, Colorado State University</td>
<td>VIBS 243 VIBS 443</td>
<td>8%</td>
</tr>
<tr>
<td>Tamy Frank-Cannon, Clinical Assistant Professor</td>
<td>PhD in Veterinary Anatomy and DVM, Texas A&amp;M University</td>
<td>Research experiences</td>
<td>2%</td>
</tr>
<tr>
<td>Candice Brinkmeyer-Langford, Research Assistant Professor</td>
<td>PhD in Genetics, Texas A&amp;M University</td>
<td>Research experiences</td>
<td>2%</td>
</tr>
<tr>
<td>Nick Jeffrey, Professor</td>
<td>PhD in Neuroscience and BVSc, University of Cambridge (University of Bristol) Diplomate in Veterinary Surgery and Veterinary Neurology (European Colleges of Veterinary Neurology and Veterinary Surgery)</td>
<td>Research experiences</td>
<td>2%</td>
</tr>
<tr>
<td>Sharon Kerwin, Professor</td>
<td>DVM, Texas A&amp;M University Diploma ACVS (Veterinary Surgery) and ACVIM (Neurology)</td>
<td>Research experiences</td>
<td>2%</td>
</tr>
<tr>
<td>William H. Griffith, Professor and Head</td>
<td>Ph.D. in Pharmacology/Neuroscience University of Texas Medical Branch – Galveston</td>
<td>Research experiences</td>
<td>2%</td>
</tr>
<tr>
<td>Name</td>
<td>Degree Information</td>
<td>Research experiences</td>
<td></td>
</tr>
<tr>
<td>-----------------------------</td>
<td>-----------------------------------------------------------------------------------------------------------------------</td>
<td>-----------------------</td>
<td></td>
</tr>
<tr>
<td>Gregg Allen, Instr. Assistant Prof.</td>
<td>Ph.D. in Medical Science, Texas A&amp;M University</td>
<td>2%</td>
<td></td>
</tr>
<tr>
<td>Katherine Brakora, Instr. Assistant Prof.</td>
<td>Ph.D. in Integrative Biology, University of California</td>
<td>2%</td>
<td></td>
</tr>
<tr>
<td>Diane Chico, Assoc. Prof.</td>
<td>Ph.D. in Cell Biology, University of Texas Medical Branch – Galveston</td>
<td>2%</td>
<td></td>
</tr>
<tr>
<td>Dustin DuBois, Assoc. Prof.</td>
<td>Ph.D. in Biomedical Science and Pharmacology, Texas A&amp;M University</td>
<td>2%</td>
<td></td>
</tr>
<tr>
<td>Rajesh Miranda, Prof.</td>
<td>Ph.D. in Biopsychology/Neurobiology, University of Rochester</td>
<td>2%</td>
<td></td>
</tr>
<tr>
<td>Samba Reddy, Prof.</td>
<td>Ph.D. in Pharmacology, Panjab University, Chandigarh, India</td>
<td>2%</td>
<td></td>
</tr>
<tr>
<td>Mendell Rimer, Assoc. Prof.</td>
<td>Ph.D. in Molecular and Cell Biology, University of Maryland at Baltimore</td>
<td>2%</td>
<td></td>
</tr>
<tr>
<td>Lee Shapiro, Assoc. Prof.</td>
<td>Ph.D. in Neuroscience, State University of New York</td>
<td>2%</td>
<td></td>
</tr>
<tr>
<td>Rahul Srinivasan, Assoc. Prof.</td>
<td>PhD in Human Genetics, University of Pittsburgh</td>
<td>2%</td>
<td></td>
</tr>
<tr>
<td>Jun Wang, Assoc. Prof.</td>
<td>M.D., Ph.D. in Neurobiology, Tongji Medical University, Shanghai Brain Research Institute</td>
<td>2%</td>
<td></td>
</tr>
<tr>
<td>Michelle Hook, Assoc. Prof.</td>
<td>Ph.D. in Physiology, University of New England, Australia</td>
<td>2%</td>
<td></td>
</tr>
<tr>
<td>David Earnest, Prof.</td>
<td>Ph.D. in Neurobiology, Northwestern University</td>
<td>2%</td>
<td></td>
</tr>
<tr>
<td>Laura Smith, Assoc. Prof.</td>
<td>Ph.D. in, George Mason University</td>
<td>2%</td>
<td></td>
</tr>
<tr>
<td>Cedric Geoffroy, Assoc. Prof.</td>
<td>Ph.D. in Neuroscience, University of Cambridge, UK</td>
<td>2%</td>
<td></td>
</tr>
<tr>
<td>Ursula Winzer-Serhan, Assoc. Prof.</td>
<td>Ph.D. in Cell Biology, University of Bremen, Germany</td>
<td>2%</td>
<td></td>
</tr>
<tr>
<td>Farida Sohrabji, Prof.</td>
<td>Ph.D. in Biopsychology/Neurobiology, University of Rochester</td>
<td>2%</td>
<td></td>
</tr>
<tr>
<td>John K Hubbard, PhD, PT; Instr. Assoc. Prof.</td>
<td>Ph.D. in Allied Health Education, Medical Anatomy, Texas A&amp;M University</td>
<td>2%</td>
<td></td>
</tr>
</tbody>
</table>
E. Library Resources

Attached please find a letter of support from the Dean of University Libraries, Dr. David Carlson. As noted in that letter, the Libraries can currently support the proposed BS in Neuroscience program and is committed to supporting the program. Students access library resources through an online searchable platform, with many media available electronically and some print holdings available at libraries on campus (or by request for an electronic copy or mailed copy). Faculty and administrators communicate with librarians as needed to provide support to students or request additional materials.

F. Facilities and Equipment

The program will utilize space allocated to the Department of Psychological and Brain Sciences, the Department of Biology, the College of Veterinary Medicine and Biomedical Sciences, and the Department of Neuroscience and Experimental Therapeutics in the College of Medicine in multiple buildings that span the campus. The Department of Psychological and Brain Sciences (PBSI) maintains three large classrooms and several smaller rooms, which accommodate the majority of department courses offered every semester. The College of Veterinary Medicine and Biomedical Sciences has three new teaching buildings as well as one large classroom and multiple smaller classrooms located in the older, the older Veterinary Medicine Administration building. Faculty laboratories that will support hands-on research experiences for the program are located in the Psychology Building, the Peterson building, the Milner Building, the Interdisciplinary Life Sciences Building (ILSB), Texas A&M Institute for Preclinical Studies (TIPS), multiple biology buildings, the Veterinary Medicine Administration Building (VMA), the Veterinary Research Building (VRB), the addition to the VRB Building, the Veterinary Teaching Hospital, COM buildings include the Health Professions Education Building, the Medical Research & Education Building and the School of Public Health (building number 1518). PBSI is scheduled to lose lab space currently designated in the Peterson building, but planning is underway to provide equivalent space and associated vivarium facilities in Nagle Hall. Students in these laboratories will gain experience with neuroscience methods using animal and human models to address psychological and physical health and disorder related to neuroscience.

G. Accreditation

The discipline of Neuroscience does not have a specific accreditation process or accrediting agency or organization, nor do any of the associated disciplines (e.g., Psychology, Biology, Veterinary Medicine and Human Medicine) at the undergraduate level. Each program undergoes external review every seven years, consistent with University policy. Each degree program offered is also reviewed annually through university assessment procedures, to meet requirements for the Southern Association of Colleges and Schools Commission on Colleges accreditation process.

H. Evaluation

Degree programs undergo external review every seven years, consistent with University policy. Each degree program offered is also reviewed annually through university assessment procedures, to meet requirements for the Southern Association of Colleges and Schools Commission on Colleges accreditation process.
Students will complete several assessments during the proposed degree program to assess development and mastery of core competencies. These assessments will assess each of the four learning objectives:

1. To provide students with a broad understanding of basic concepts in the field of neuroscience, with specific advanced knowledge in a subfield (molecular & cellular; behavioral & cognitive; translational & preclinical).
2. To provide students with the ability to explain neuroscience concepts to the lay public.
3. To enhance student understanding of diversity in all forms, including neuro-diversity, neurodevelopment, and individual differences.
4. To provide students with strong writing and technical skills necessary to communicate and work in fields associated with neuroscience.

Assessment includes graduation rates, time to degree, employment upon graduation, self-reported evaluations of the program, as well as direct assessment of mastery of the learning objectives above. These assessments are collected and reviewed annually, with plans to improve outcomes developed and accomplished in conjunction with the assessment.

III. Costs and Funding

A. Five-Year Costs and Funding Sources Summary

Estimated costs and funding are provided below. Identifiable revenues cover the incremental costs of operating the program. Costs are estimated to be minimal, given that faculty, facilities, and resources are currently available to contribute to this program. Costs for faculty are estimated based on anticipated enrollment in the program; if the students are new students rather than those that would complete an existing major, at least one additional faculty member is likely to be needed in year 4 to meet demand (estimated salary $150,000 x 2 years). Administration includes oversight of advertising and developing the program ($50,000). Graduate assistants is estimated based on 1 graduate student TAs for every 50 majors to provide writing and grading support and to supervise teaching labs ($840,000 in new costs over 5 years; depending on enrollment patterns this could be reallocated or new student support). Clerical/staff support is estimated as an additional advisor when enrollments hit approximately 200 students ($45,000 per year in year 4 and 5). IT resources include high throughput computing support for imaging data ($30,000). Supplies and materials include estimates for teaching labs and demonstrations ($150,000).

B. Signature Page

The signature page must be signed by the required institutional officials and board of regents.

V. Additional Distance Education Delivery Consideration

This is not a distance education program.
VI. Required Appendices (see individual attachments)

A. Course Descriptions and Prescribed Sequence of Courses
B. Five-Year Faculty Recruitment Plan/Hiring Schedule
C. Institution’s Policy on Faculty Teaching Load
D. Itemized List of Capital Equipment Purchases During the Past Five Years¹
   *Equipment* means an article of nonexpendable, tangible personal property having a useful life of more than one year and an acquisition cost, which equals or exceeds the lesser of the capitalization level established by the governmental unit for financial statement purposes, or $5,000.

E. Librarian’s Statement of Adequate Resources

See attached letter from the Library Dean, confirming the availability of adequate resources and support for the program.

F. Articulation Agreements with Partner Institutions

Include copies of any agreements or Memoranda of Understanding related to the proposed program. These include formal and sustained arrangements with other universities, private businesses, or governmental agencies that contribute directly to the proposed program and student research/residency opportunities.

   No such partners exist at this time.

G. Curricula Vitae for Core Faculty

H. Curricula Vitae for Support Faculty

I. List of Specific Clinical or In-Service Sites to Support the Proposed Program

J. Letters of Support from Peer Institutions and/or Area Employers

Letters from regional and national companies who have made commitments to hire graduates from the proposed new program are particularly helpful. Also, include statements of support or commitments to shared research projects from other institutions in the state with similar programs.

¹”Equipment” has the meaning established in the Texas Administrative Code §252.7(3) as items and components whose cost are over $5,000 and have a useful life of at least one year.
## Costs to the Institution of the Proposed Program

Complete the table to show the costs to the institution that are anticipated from the proposed program.

<table>
<thead>
<tr>
<th>Cost Category</th>
<th>Cost Sub-Category</th>
<th>1st Year</th>
<th>2nd Year</th>
<th>3rd Year</th>
<th>4th Year</th>
<th>5th Year</th>
<th>TOTALS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Faculty Salaries(^1)</td>
<td>New</td>
<td></td>
<td></td>
<td>$150,000</td>
<td>$150,000</td>
<td>$300,000</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Reallocated</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Program Administration</td>
<td>New</td>
<td>$10,000</td>
<td>$10,000</td>
<td>$10,000</td>
<td>$10,000</td>
<td>$10,000</td>
<td>$50,000</td>
</tr>
<tr>
<td></td>
<td>Reallocated</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Graduate Assistants</td>
<td>New</td>
<td>$120,000</td>
<td>$120,000</td>
<td>$200,000</td>
<td>$200,000</td>
<td>$200,000</td>
<td>$840,000</td>
</tr>
<tr>
<td></td>
<td>Reallocated</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clerical/Staff</td>
<td>New</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>$90,000</td>
</tr>
<tr>
<td></td>
<td>Reallocated</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Student Support (Scholarships)</td>
<td>Supplies and Materials</td>
<td>$30,000</td>
<td>$30,000</td>
<td>$30,000</td>
<td>$30,000</td>
<td>$30,000</td>
<td>$150,000</td>
</tr>
<tr>
<td></td>
<td>Library &amp; Instructional Technology Resources(^2)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>$30,000</td>
</tr>
<tr>
<td></td>
<td>Equipment(^2)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Facilities</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Other (Identify)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>TOTALS</td>
<td>$160,000</td>
<td>$190,000</td>
<td>$240,000</td>
<td>$435,000</td>
<td>$435,000</td>
<td>$1,460,000</td>
</tr>
</tbody>
</table>

\(^1\) Report costs for new faculty hires, graduate assistants, and technical support personnel. For new faculty, prorate individual salaries as a percentage of the time assigned to the program. If existing faculty will contribute to program, include costs necessary to maintain existing programs (e.g., cost of adjunct to cover courses previously taught by faculty who would teach in new program).

\(^2\)Equipment has the meaning established in the Texas Administrative Code §252.7(3) as items and components whose cost are over $5,000 and have a useful life of at least one year.
### Anticipated Sources of Funding

Complete the table to show the amounts anticipated from various sources to cover new costs to the institution as a result of the proposed program. Use the Non-Formula Sources of Funding form to specify as completely as possible each non-general revenue source.

<table>
<thead>
<tr>
<th>Funding Category</th>
<th>1st Year</th>
<th>2nd Year</th>
<th>3rd Year</th>
<th>4th Year</th>
<th>5th Year</th>
<th>TOTALS</th>
</tr>
</thead>
<tbody>
<tr>
<td>I. Formula Funding¹</td>
<td></td>
<td></td>
<td>$220,564</td>
<td>$220,564</td>
<td>$433,378</td>
<td>$874,506</td>
</tr>
<tr>
<td>II. Other State Funding</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>III. Reallocation of Existing Resources</td>
<td>$10,000</td>
<td>$10,000</td>
<td>$10,000</td>
<td>$10,000</td>
<td>$10,000</td>
<td>$50,000</td>
</tr>
<tr>
<td>IV. Federal Funding (In-hand only)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>V. Tuition and Fees (includes statutory,</td>
<td>$858,433</td>
<td>$1,681,020</td>
<td>$2,122,500</td>
<td>$2,563,980</td>
<td>$2,563,980</td>
<td>$9,789,913</td>
</tr>
<tr>
<td>designated, and fees)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VI. Other Funding²</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TOTALS</td>
<td>$868,433</td>
<td>$1,691,020</td>
<td>$2,353,064</td>
<td>$2,794,544</td>
<td>$3,007,358</td>
<td>$10,714,419</td>
</tr>
</tbody>
</table>

¹ Indicate formula funding for students new to the institution because of the program; formula funding should be included only for years three through five of the program and should reflect enrollment projections for years three through five.

² Report other sources of funding here. In-hand grants, "likely" future grants, and special item funding can be included.
Non-Formula Sources of Funding

Complete the table to specify each of the non-formula funding sources for the amounts listed on the Anticipated Sources of Funding form.

<table>
<thead>
<tr>
<th>Funding Category</th>
<th>Non-Formula Funding Sources</th>
</tr>
</thead>
<tbody>
<tr>
<td>II. Other State Funding</td>
<td>#1</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>#2</td>
</tr>
<tr>
<td>III. Reallocation of Existing Resources</td>
<td>#1</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>#2</td>
</tr>
<tr>
<td>IV. Federal Funding (In-hand only)</td>
<td>#1</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>#2</td>
</tr>
<tr>
<td>V. Tuition and Fees</td>
<td>#1</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>#2</td>
</tr>
<tr>
<td>VI. Other Funding</td>
<td>#1</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>#2</td>
</tr>
</tbody>
</table>
1. **Adequacy of Funding and Notification of Other Institutions** – The Chief Executive Officer shall sign the following statements:

   I certify that the institution has adequate funds to cover the costs of the proposed program. Furthermore, the proposed program will not reduce the effectiveness or quality of existing programs at the institution.

   I certify that my institution has notified all public institutions within 50 miles of the teaching site of our intention to offer the proposed program at least 30 days prior to submitting this request. I also certify that if any objections were received, those objections were resolved prior to the submission of this proposal.

   I certify that my institution will adhere to Texas Education Code (TEC), Sections 61.822 through 61.823, requiring my institution to accept and apply to the proposed program Core Curriculum and Field of Study courses in transfer.

   
   ________________  ________________
   Chief Executive Officer          Date

2. **Accuracy of Financial Estimates** – The Chief Financial Officer shall sign the following statement:

   I certify that the estimated costs and sources of funding presented in the proposal are complete and accurate.

   
   ________________  ________________
   Chief Financial Officer          Date

3. **Board of Regents or Designee Approval** – A member of the Board of Regents or designee shall sign the following statement:

   On behalf of the Board of Regents, I hereby certify that the proposed program is appropriate for the mission of this institution and the Board of Regents has approved the proposed program.

   
   ________________  ________________
   Board of Regents (Designee)          Date
Directions: Texas public universities and health-related institutions complete this form to add a new bachelor’s or master’s degree program, if the following criteria for approval are met, per Texas Administrative Code (TAC), Title 19, Chapter 5, Subchapter C, Section 5.44 (a) (3): (A) the proposed program has institutional and board of regents approval; (B) the institution certifies compliance with the Standards for New Bachelor’s and Master’s Programs; (C) the institution certifies that adequate funds are available to cover the costs of the new program; (D) new costs to the program during the first five years of the program would not exceed $2 million; (E) the proposed program is a non-engineering program; and (F) the proposed program would be offered by a public university or health-related institution.

If the proposed program does not meet the criteria listed above, the institution must submit a request using the Full Request Form.

This form requires the signatures of: (1) the Chief Executive Officer, certifying adherence to the Texas Administrative Code (TAC), Title 19, Chapter 5, Subchapter C, Section 5.44 (a) (3) criteria, adequacy of funding for the new program, the notification of other Texas public institutions of higher education, and adherence to Texas Education Code (TEC) Sections 61.822 through 61.823; and (2) a member of the Board of Regents (or designee) certifying Board approval.

Contact: Division of Academic Quality and Workforce, 512-427-6200.

Administrative Information

1. Institution Name and Coordinating Board Accountability Group:
   Texas A&M University

2. Proposed Program:
   Bachelor of Science in Neuroscience


4. Semester Credit Hours Required: 120 SCH

5. Location and Delivery of the Proposed Program:
   Face-to-face delivery on the main campus in College Station
6. Administrative Unit:

This is an interdisciplinary degree, and will therefore be housed at the University level with concentrations administered by units.

The degree will have concentrations (not requested to be listed individually on the Program Inventory) in the following areas:

1. Behavioral & Cognitive Neuroscience – Department of Psychological & Brain Sciences, College of Liberal Arts
2. Molecular & Cellular Neuroscience – Department of Biology, College of Science
3. Translational & Preclinical Neuroscience – College of Veterinary Medicine and Biomedical Sciences

7. Proposed Implementation Date:
Provide the date that students would enter the proposed program

August 1, 2020

8. Institutional and Department Contacts:
Provide contact information for the person(s) responsible for addressing any questions related to the proposed program.

1. Behavioral & Cognitive Neuroscience:
   Name: Heather Lench
   Title: Professor and Head of Psychological & Brain Sciences
   E-mail: hlench@tamu.edu
   Phone: (979)845-0377

2. Molecular & Cellular Neuroscience:
   Name: Thomas McKnight
   Title: Professor and Head of Biology
   E-mail: mcknight@bio.tamu.edu
   Phone: (979) 845-3896

3. Translational & Preclinical Neuroscience:
   Name: Elizabeth Crouch
   Title: Associate Dean for Undergraduate Education, College of Veterinary Medicine & Biomedical Sciences
   E-mail: ecrouch@cvm.tamu.edu
   Phone: (979) 845-4941
Signature Page

1. **Chief Executive Officer Certification** – The Chief Executive Officer shall sign the following statements:

   *I hereby certify that all of the following criteria have been met in accordance with the procedures outlined in Texas Administrative Code (TAC), Title 19, Chapter 5, Subchapter C, Section 5.44 (a) (3):*

   (A) The proposed program has institutional and governing board approval.

   (B) The institution certifies compliance with the *Standards for New Bachelor’s and Master’s Programs.*

   (C) The institution certifies that adequate funds are available to cover the costs of the new program.

   (D) New costs during the first five years of the program would not exceed $2 million.

   (E) The proposed program is a non-engineering program.

   (F) The proposed program would be offered by a public university or health-related institution.

   *I certify that my institution has notified all public institutions within 50 miles of the teaching site of our intention to offer the proposed program at least 30 days prior to submitting this request. I also certify that if any objections were received, those objections were resolved prior to the submission of this request.*

   *I certify that my institution will adhere to Texas Education Code (TEC), Sections 61.822 through 61.823, requiring my institution to accept and apply to the degree program Core Curriculum and Field of Study courses in transfer.*

   _______________________________    _______________________
   Chief Executive Officer                  Date

2. **Board of Regents or Designee Approval** – A member of the Board of Regents or designee shall sign the following statement:

   *On behalf of the Board of Regents, I hereby certify that the proposed program is appropriate for the mission of this institution, and the Board of Regents has approved the proposed program.*

   Date of Board of Regents approval: ____________________

   _______________________________    _______________________
   Board of Regents (Designee)                  Date
MEMORANDUM

7 June, 2018

TO: Dr. Carol A. Fierke, Provost and Executive Vice President

THROUGH: Dr. Eleanor M. Green, Carl B. King Dean of Veterinary Medicine
Dr. Pamela R. Matthews, Dean of Liberal Arts
Dr. Meigan C. Aronson, Dean of Science

FROM: Dr. Elizabeth Crouch, Associate Dean for Undergraduate Education,
College of Veterinary Medicine and Biomedical Sciences
Dr. C. Jane Welsh, Interim Head of Veterinary Integrative Biosciences
Dr. Heather C. Lench, Head of Psychological & Brain Sciences
Dr. Thomas D. McKnight, Head of Biology
Dr. Michael S. Smotherman, Chair, Texas A&M Institute for Neuroscience

SUBJECT: Proposed Bachelor of Science in Neuroscience

The heads of the Departments of Psychological & Brain Sciences (PBSI) in the College of Liberal Arts, Biology (BIOL) in the College of Science, and Veterinary Integrative Biosciences (VIBS) in the College of Veterinary Medicine and Biomedical Sciences, as well as the chair of Texas A&M Institute for Neuroscience (TAMIN) and the TAMIN Undergraduate Program Committee have discussed and all agree that it would be highly advantageous to the development of the Proposed Bachelor of Science in Neuroscience if the College of Veterinary Medicine and Biomedical Sciences in collaboration with the Neuroscience and Experimental Therapeutics (NExT) department in the College of Medicine were to simultaneously participate in the development of the Bachelor of Science Degree in Neuroscience. As stated previously, the degree will be placed at the University level but with three concentrations, one in Biology, one in Psychology and one jointly in the College of Veterinary Medicine and Biomedical Sciences and NExT. We believe that adding the third concentration, which will be Translational and Preclinical Neuroscience, will add significant depth and rigor to the overall undergraduate neuroscience degree program as well as enhance the interdisciplinary impact of the major.

Additional points to consider:

- TAMIN initiated the idea to develop the undergraduate neuroscience degree program and TAMIN faculty of continue to be highly supportive of the proposed Bachelor of Science in Neuroscience. In fact, TAMIN has already developed and currently administers the Neuroscience Minor at TAMU. Undergraduate student enrollment in the Neuroscience Minor has grown significantly since its inception: 2010 – 80
  2011 – 160
  2017 – 291
  2018 – 340

- The formation of three concentrations at this time for the Bachelor of Science in Neuroscience is ideal for the major because we will be able to offer courses in three major areas of neuroscience: cognitive, molecular / biochemical and biomedical/translational.

- The department head of NExT in the College of Medicine has met with the department heads of the other participating departments and agrees that neuroscience faculty in NExT will collaborate with the College of
Veterinary Medicine and Biomedical Sciences to administer the Translational and Preclinical Neuroscience concentration. Neuroscience faculty in NExT will hold adjunct appointments in the one of the participating departments from the College of Veterinary Medicine and Biomedical Sciences or Biology or Psychological & Brain Sciences.

We are submitting a degree planning memo that is in conjunction with and adds to the degree planning memo that was submitted to the Office of the Provost by the Biology and Psychology Departments on October 25, 2017. We have provided information that was requested in the degree proposal memo from your office dated December 6, 2016. We also are attaching a copy of the original Memorandum of Understanding to this memo. We will be happy to discuss any additional details or answer any questions that may arise.

The following information is the same as can be found in the Memo from October 25, 2017 that came from the Psychology and Biology Departments.

**Title of the degree** – Neuroscience

**Level of the degree** – Undergraduate, Bachelor of Science

**CIP code** – 26.1501

The **Description, Justification and Implementation date** can be found on the Memo from October 25, 2017, a copy of which can be found at the end of this memo.

**5-year costs** – In addition to the costs outlined in the Memo from October 25, 2017 from the Psychology and Biology departments, costs for the third track are estimated as follows. The cost for initial development and implementation of the BS in Neuroscience will be minimal. We currently have courses totaling 22 credits of upper division neuroscience related courses that are being offered. We anticipate developing 4 or 5 additional upper division courses that will include capstone and writing intensive courses in the major. If student enrollments reach projected levels by the 4th or 5th year of the program we will need to add additional support for undergraduate advising and assessment. We anticipate the cost would be $45,000 per year per advisor FTE. Due to the large number of neuroscience faculty currently residing in the College of Veterinary Medicine and the NExT department in the College of Medicine, we do not anticipate needing additional faculty FTEs to support this concentration. We expect that any neuroscience faculty who leave the university will be replaced by their respective departments.

**B.S. in Neuroscience – Draft 5/21/18**

**Information for third concentration**

<table>
<thead>
<tr>
<th>Degree Code</th>
<th>Translational &amp; Preclinical Neuroscience</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initiating Units</td>
<td>College of Veterinary Medicine &amp; Biomedical Sciences and NExT-COM</td>
</tr>
<tr>
<td>Foundations in Neuroscience (7 credits) - REQUIRED</td>
<td></td>
</tr>
<tr>
<td>These are courses that are common to all three concentrations in the major</td>
<td></td>
</tr>
<tr>
<td>Supporting Foundational Coursework (44 credit hours) - REQUIRED</td>
<td></td>
</tr>
<tr>
<td>• NRSC 101 - overview seminar and orientation (1 credit)</td>
<td></td>
</tr>
<tr>
<td>• NRSC/PSYC 335 – Physiological Psychology (3 credits)</td>
<td></td>
</tr>
<tr>
<td>• NRSC/VIBS 277 – Introduction to Neuroscience (3 credits)</td>
<td></td>
</tr>
<tr>
<td>• BIOL 111 - Introductory Biology I (4 credits, lecture and lab)</td>
<td></td>
</tr>
<tr>
<td>• BIOL 112 - Introductory Biology II (4 credits, lecture and lab)</td>
<td></td>
</tr>
<tr>
<td>• CHEM 101 – Fundamentals of Chemistry I (3 credits)</td>
<td></td>
</tr>
</tbody>
</table>
| Concentration Coursework: Breadth in Concentration (11 credit hours) - REQUIRED | • NRSC/BIOL 434 Neurobiology (3 credits)  
• NRSC/BIOL 435 Neurobiology lab (1 credit)  
• NRSC/VIBS 450 Functional Neuroanatomy – Lecture and lab (4 credits)  
• Select one upper division Psychology course (3 credits) |
|---|---|
| Concentration Coursework: Electives (15 credit hours) | Select from:  
• NRSC/VIBS 401 – Developmental Neurotoxicology (2 credits)  
• NRSC/VIBS 407 – Core Ideas of Neurosciences (2 credits)  
• VIBS 408 – Neuroscience and Religion (3 credits; W course)  
• VIBS 447 – Neurophysiology of Music (2 credits; C course)  
• VIBS 606 (stacked course) – Neuroanatomical Systems (3 credits)  
• VIBS 424 – Biomedical Neuroendocrinology and Endocrine Disorders (3 credits)  
• VIBS 640 (stacked course) – Neurobiology (3 credits)  
• VIBS 422 – Endocrine Toxicology (4 credits)  
• VTMI 662 (stacked course) – Neuroimmunology (2 credits)  
• VIBS 343 – Histology – lecture and lab (4 credits; W course)  
• VIBS 443 – Biology of Cells – lecture and lab (4 credits)  
• VTPP 323 – Physiology of Domestic Animals (3 credits)  
• VTPP 425 – Pharmacology (3 credits)  
• Any NRSC 300-489 course |
| Free Electives (8-10 credit hours) | Select from Courses list |
| Core Curriculum – General Coursework (33-35 credit hours) - REQUIRED | Communications (6 credits)  
• ENG 104 (3 credits)  
• Communication Elective (3 credits)  
• MATH 147 & MATH 148 (8 credits)  
OR  
• Math 151 & MATH 152 (8 credits) – (required for minor in biochemistry or biomedical engineering) |
<table>
<thead>
<tr>
<th>Citizenship (12 credits)</th>
</tr>
</thead>
<tbody>
<tr>
<td>• American History (6 credits)</td>
</tr>
<tr>
<td>• POLS 206 (3 credits)</td>
</tr>
<tr>
<td>• POLS 207 (3 credits)</td>
</tr>
<tr>
<td>Creative Arts (3 credits)</td>
</tr>
<tr>
<td>International / cultural diversity (also counts as Language, Philosophy and Culture) (6 credits)</td>
</tr>
<tr>
<td>Social Science / Behavioral Science (3 credits)</td>
</tr>
<tr>
<td>• PSYCH 107</td>
</tr>
</tbody>
</table>

Total hours = 120; Must complete 2 W courses