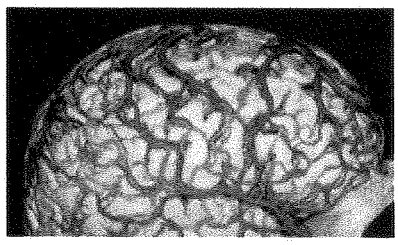

McKnight Brain Research Foundation
1st Inter-Institutional Meeting
University of Arizona
Tucson, AZ

April 16-18, 2008

Evelyn F. McKnight Brain Institute, University of Arizona
Evelyn F. McKnight Brain Institute, University of Alabama at Birmingham
Evelyn F. and William L. McKnight Brain Institute, University of Florida
Evelyn F. McKnight Center for Age-Related Memory Loss, University of Miami

Evelyn F. McKnight Brain Institute
The University of Arizona

UAB EVELYN F. MCKNIGHT BRAIN INSTITUTE
DEPARTMENT OF NEUROBIOLOGY

Evelyn F. and William L. McKnight Brain Institute of the University of Florida

MILLER SCHOOL OF MEDICINE
UNIVERSITY OF MIAMI

Special thanks to

The Evelyn F. McKnight Brain Research Foundation

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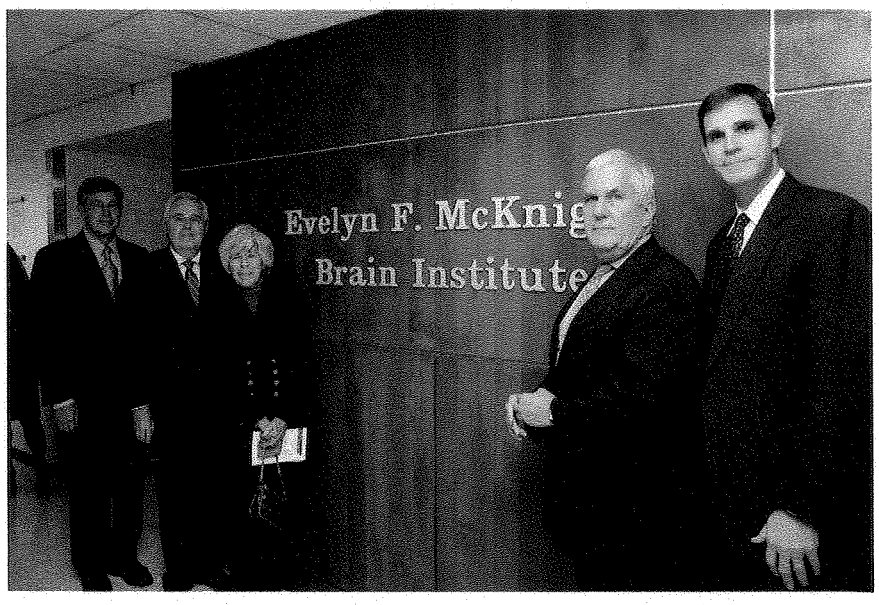

McKnight Brain Research Foundation
Inter-Institutional Meeting
April 16-18, 2008
Tucson, AZ

Wednesday, April 16

6:30pm – 8:00pm	Welcome Reception - Arizona Inn, 2200 East Elm Street African Room and Terrace

Thursday, April 17

7:00am – 8:00am	Buffet Breakfast for Participants Staying at the Arizona Inn Safari Room
7:30am – 8:30am	Shuttle service to Arizona Health Sciences Center (AHSC) 1501 N. Campbell Avenue (Meet shuttle in front of Arizona Inn)
8:00am – 8:45am	Registration (AHSC room 8403)
8:45am – 9:00am	Welcome by University of Arizona Administrators Dr. Leslie Tolbert, Vice President for Research Dr. Michael Cusanovich, Director, Arizona Research Laboratories Dr. Edward Donnerstein, Dean, College of Social and Behavioral Sciences
9:00am – 9:15am	Welcome and Introductions by Trustees The Evelyn F. McKnight Brain Research Foundation
9:15am – 10:00am	Carol A. Barnes, Ph.D. Summary of University of Arizona Institute
10:00am – 10:15am	Discussion
10:15am – 10:30am	Break
10:30am – 11:15am	Dennis A. Steindler, Ph.D. and Thomas C. Foster, Ph.D. Summary of University of Florida Institute
11:15am – 11:30am	Discussion
11:30am – 12:15pm	University of Arizona Evelyn F. McKnight Brain Institute Tour Life Sciences North Bldg – 3rd floor
12:15pm	Depart for Arizona Inn (Shuttles available in front of AHSC)
12:30pm – 1:45pm	Lunch at Arizona Inn (Tucson Room)

2:00pm – 2:45pm	J. David Sweatt, Ph.D Summary of University of Alabama at Birmingham Institute
2:45pm – 3:00pm	Discussion
3:00pm – 3:15pm	Break
3:15pm – 4:00pm	Ralph L. Sacco, M.D. Summary of University of Miami Center
4:00pm – 4:15pm	Discussion
4:15pm – 5:15pm	Tour of Evelyn F. McKnight Memory and Cognitive Assessment Clinic Psychology Building (Shuttle available in front of AHSC)
5:15pm	Depart for Arizona Inn (Shuttle available from Psychology Building)
5:30pm – 6:30pm	Cocktail Reception at Arizona Inn (Tucson Patio)
6:30pm	Dinner at Arizona Inn (Tucson Room)

Friday, April 18 – By invitation (Arizona Inn)

8:00am – 8:30am	Meeting of the Trustees, McKnight Directors, Endowed Professors and Endowed Chairs to discuss breakout group structure (Arizona Inn, Flandrau House)
8:00am – 9:00am	Buffet Breakfast at the Arizona Inn Safari Room and African Terrace
9:00am	Comments from Trustees and Break-out Group Assignment (African Room)
9:15am – 10:30am	Break-out Group Discussions of Potential Collaborations Group #1: Flandrau House East Parlor Group #2: Flandrau House East Patio Courtyard Group #3: Flandrau House Backyard Group #4: Flandrau House West Parlor Group #5: Flandrau House Living Room Group #6: Flandrau House Flagstone Courtyard
10:30am – 10:45am	Break (Tucson Room)
10:45am – 11:30am	Summary of Collaborative Ideas (Tucson Room)
11:30am	Box lunch provided (departure for airport or remain in the meeting)

Substantial overlap of science internal & external -

Evelyn F. McKnight Brain Institute Meeting Participants
University of Arizona

Carol A. Barnes, Ph.D.
Director, Evelyn F. McKnight Brain Institute
Evelyn F. McKnight Endowed Chair for Learning and Memory in Aging
Regents' Professor, Psychology and Neurology

Geoffrey L. Ahern, M.D., Ph.D.
Bruce and Lorraine Cuming Endowed Chair in Alzheimer's Research
Professor, Neurology, Psychology and Psychiatry

Gene E. Alexander, Ph.D.
Professor and Director, Brain Imaging Behavior and Aging Lab
Department of Psychology

E. Fiona Bailey, Ph.D.
Assistant Professor
Physiology

Sara N. Burke, M.S.
Graduate Research Associate
Neuroscience Program

Monica K. Chawla, Ph.D.
Assistant Research Scientist
ARL Neural Systems, Memory and Aging

Haiyen Chen, Ph.D.
Postdoctoral Fellow
ARL Neural Systems, Memory and Aging

Jean-Marc Fellous, Ph.D.
Associate Professor
Psychology, Applied Mathematics, and
ARL Neural Systems, Memory and Aging

Ralph Fregosi, Ph.D.
Professor
Physiology and Neurology

Andrew J. Fuglevand, Ph.D.
Associate Professor
Physiology and Neurobiology

Elizabeth L. Glisky, Ph.D.
Professor, Associate Head, Director of Graduate Studies
Department of Psychology

Katalin M. Gothard, M.D., Ph.D.
Assistant Professor
Physiology and Neurology

G. Alex Hishaw, M.D.
Evelyn F. McKnight Fellow in Behavioral Neurology and Neuropsychiatry

Lan T. Hoang, B.S.
Graduate Research Assistant
Neuroscience Program

Nathan Insel, M.S.
Graduate Research Associate
Cognition and Neural Systems Program

Alfred W. Kaszniak, Ph.D.
Professor of Psychology, Neurology and Psychiatry
Head, Department of Psychology

Thabelo Khoboko, M.Sc. (Med)
Graduate Research Assistant
Neuroscience Program

James P. Lister, Ph.D.
Postdoctoral Research Associate
ARL Neural Systems, Memory and Aging

Bruce L. McNaughton, Ph.D.
Professor, Psychology and Physiology
Director, ARL Division of Neural Systems, Memory and Aging
Chair, Graduate Interdisciplinary Program in Neuroscience

University of Arizona (continued)

Lynn Nadel, Ph.D.
Regents' Professor, Psychology and Cognitive Science
Director, Cognition and Neural Systems Program

Marsha R. Penner, M.S.
Graduate Research Associate
Neuroscience Program

Mary A. Peterson, Ph.D.
Professor, Department of Psychology
Research Social Scientist, Cognitive Science Program

Naomi E. Rance, Ph.D.
Professor of Pathology, Neurology, Cell Biology and Anatomy
Associate Head or Pathology

Eric M. Reiman, M.D.
Executive Director,
Banner Alzheimer's Institute
Clinical Director, Neurogenomics Division,
Translational Genomics Research Institute
Professor and Associate Head, Psychiatry
University of Arizona
Director, Arizona Alzheimer's Consortium

Linda L. Restifo, M.D., Ph.D.
Professor, ARL Division of Neurobiology and Neurobiology

Lee Ryan, Ph.D.
Associate Professor
Psychology and Neurology

Rachel Samson, Ph.D.
Postdoctoral Research Associate
ARL Neural Systems, Memory and Aging

Lesley A. Schimanski, Ph.D.
Postdoctoral Research Associate
ARL Neural Systems, Memory and Aging

Robert S. Sloviter, Ph.D.
Professor
Pharmacology and Neurology

Alexander Thome, B.A.
Graduate Research Associate
Neuroscience Program

Michelle Valfre, B.A.
Graduate Student, Psychology
Cognition and Neuroimaging Laboratory

Katrin Walther, Ph.D.
Postdoctoral Fellow
Department of Psychology

Carol A. Barnes, Ph.D.
Regents' Professor, Psychology and Neurology
Director, Evelyn F. McKnight Brain Institute
Research Scientist, ARL Division of Neural Systems,
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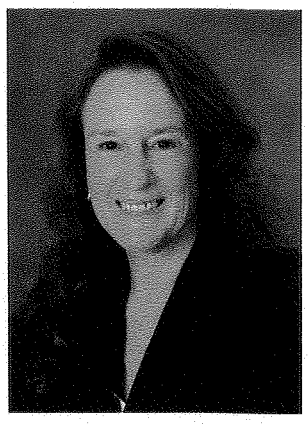

The central goal of Dr. Barnes' research and teaching program is the question of how the brain changes during the aging process and the functional consequences of these changes on information processing and memory in the elderly. Her research program involves studies of behavior and neurophysiology in young and old laboratory animals. This work provides a basis for understanding the basic mechanisms of normal aging in the brain and sets a background against which it is possible to assess the effects of pathological changes such as Alzheimer's disease. Some current work also includes an assessment of therapeutic agents that may be promising in the alleviation or delay of neural and cognitive changes that occur with age. Dr. Barnes is a Regents' Professor at the University of Arizona, Director of the Evelyn F. McKnight Brain Institute at the University of Arizona and recipient of the Evelyn F. McKnight Endowed Chair for Learning and Memory in Aging. The objective of the Evelyn F. McKnight Brain Institute is to uncover the neurobiological changes in the brain that cause memory changes as we age, and to unravel which changes are due to normal aging and which are due to disease states.

Geoffrey L. Ahern, M.D., Ph.D.
Bruce and Lorraine Cumming Endowed Chair in
Alzheimer's Research
Professor of Neurology, Psychology, Psychiatry
Affiliate, Evelyn F. McKnight Brain Institute

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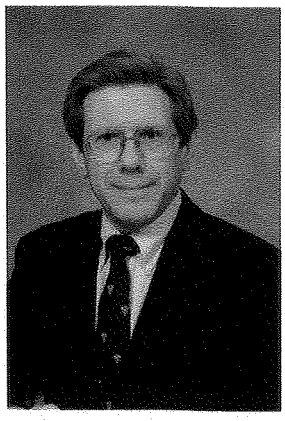

Dr. Ahern is Medical Director of the Behavioral Neuroscience and Alzheimer's Clinic at the University of Arizona. He is board-certified in Neurology and Behavioral Neurology & Neuropsychiatry. He is a member of the American Academy of Neurology and the American Neurological Association.

His clinical & research interests include: Behavioral Neurology; Dementia; Alzheimer's Disease; Intracarotid Amobarbital (Wada) Test; Paraneoplastic Syndromes; Cerebral Lateralization for Emotional Processes; Quantitative EEG; and Psychophysiology. He directs the University of Arizona site of the Arizona Alzheimer's Disease Core Center. He has participated in a number of clinical research studies, including those involved in the development of *Aricept* and *Reminyl/Razadyne*, which are now considered standard of care in the clinical treatment of Alzheimer's Disease.

Dr. Ahern's participation in the Evelyn F. McKnight Brain Institute centers on the interface between normal age-related changes in cognition and alterations in cognition that signify the onset of more ominous entities, e.g., Mild Cognitive Impairment (MCI), Alzheimer's Disease, and other dementing illnesses.

Gene E. Alexander, Ph.D.
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Director, Brain Imaging Behavior and Aging Lab
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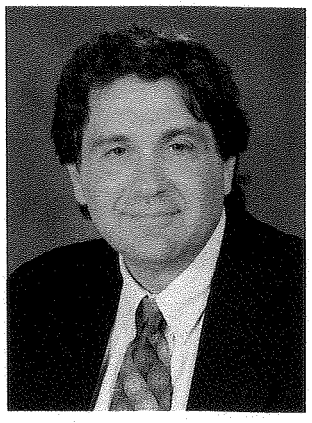

Dr. Alexander's research and academic interests focus on the study of brain-behavior relationships in the context of aging and age-related, neurodegenerative disease. He uses neuroimaging techniques, including structural and functional magnetic resonance imaging (MRI) and positron emission tomography (PET), in combination with measures of cognition and behavior to address research questions on the effects of healthy aging and Alzheimer's disease on the brain. A major focus of his research program includes the use of univariate and multivariate network analysis techniques with multiple neuroimaging methods and measures of neuropsychological function, health status, and genetic risk to help elucidate the mechanisms of human cognitive aging and to advance understanding on how these multiple factors interact to influence cognitive function as we age. Dr. Alexander's research also includes the application of these techniques to non-human animal models of aging and age-related disease. He is Professor in the Clinical and Cognition & Neural Systems Programs and directs the Brain Imaging, Behavior & Aging Lab in the Department of Psychology. Dr. Alexander has a faculty appointment in the Evelyn F. McKnight Brain Institute and directs the MRI Morphology Core of the Arizona Alzheimer's Research Center.

E. Fiona Bailey, Ph.D.
Assistant Professor, Physiology
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Dr Bailey's research examines the voluntary and respiratory-related neuromuscular control of upper airway muscles. Her research program combines behavioral and neurophysiological approaches in human and lower mammalian model systems. Some recent work includes a series of experiments conducted in healthy human subjects that examined the modulation of upper airway motor neuron activities as a function of sleep and wakefulness and during voluntary movement. These experiments have provided insights into the nervous system control of the tongue muscles in healthy human subjects that have implications for the nervous system control of movement more broadly. Specifically, the results of her research provide a much-needed foundation for future studies of upper airway muscle function in critical functions such as swallowing and speaking. These data also provide the necessary comparison data for evaluating and understanding the effect of healthy aging and disease states on oromotor function. Future directions include the assessment of upper airway muscle function in adults with Parkinson's Disease and the effects of menopause in a rodent model of ovarian failure.

Sara N. Burke, M.S.
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The central goal of Sara Burke's thesis research is the question of how functional changes in the perirhinal cortex during aging contribute to a decline in recognition memory. Her approach to investigating this problem involves using both multiple single unit recordings of perirhinal neurons in awake behaving rats, and imaging neural ensembles that were activated during distinct episodes of object exploration. Recently, Sara's research has also shown that a particular age-related deficit in behaviorally induced plasticity could be alleviated with the therapeutic agent memantine.

Monica K. Chawla, Ph.D.
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The primary goal of Dr. Chawla's research is the question of how the brain changes during the normal aging process and the functional consequences of these changes on information processing and memory in the elderly. Her research involves behavioral studies of immediate-early genes and neural plasticity mechanisms using spatial and temporal compartmental analysis in young and old laboratory animals. This work provides a basis for understanding the basic mechanisms of normal aging in the brain and sets a background against which it is possible to assess the effects of pathological changes such as Alzheimer's disease. Dr. Chawla is an Assistant Research Scientist and heads the molecular research team in Dr. Carol Barnes laboratory at the University of Arizona, Evelyn F. McKnight Brain Institute and the ARL Division of Neural Systems Memory and Aging at the University of Arizona.

Haiyin Chen, Ph.D.
Postdoctoral fellow
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Haiyin Chen is a postdoctoral fellow who recently received the National Institute of Aging National Research Service Award postdoctoral training fellowship to work with both Dr. Katalin Gothard and Dr. Carol Barnes on the contribution of emotional memory on social cognition in aging. The broad goal of her project is to determine how emotional memories modify neural encoding of social stimuli in the amygdala. The amygdala has been implicated in the decline of social cognition in the elderly, particularly with regard to facial emotion perception. This study aims to determine how emotional facial expressions are represented in the young and old amygdala, and how these representations are modified by recent or remote social experience. Emotional and social cognition are intimately linked to mental health. The amygdala serves as a 'gate' for human emotional behavior by virtue of its connections to both brain areas that serve higher mental functions and centers that control cardiovascular and immune responses. Abnormal activity in the amygdala has been found in the majority of psychiatric diseases, particularly in anxiety and mood disorders, but also in age-related neurodegenerative diseases. Understanding its normal function and changes between the young and old amygdala will be a critical step toward finding effective treatments for these diseases.

Jean-Marc Fellous, Ph.D.
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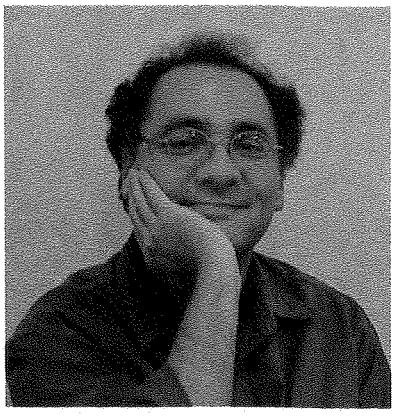

The central goal of Dr. Fellous' research is to understand how large network of neurons communicate effectively in the face of background noise and unreliable synaptic transmission. His research program involves a combination of in vitro patch clamp and in vivo multi-units recordings in the rat, together with computer simulations of biophysical network of neurons. The current focus of his lab is on the dopaminergic system, and its role in neural information processing in the normal and aged rat. Dr. Fellous' teaching program includes a class on the use of modern analysis techniques for neurophysiological data and a class on computational neuroscience as a tool for understanding brain functions.

Ralph F. Fregosi, Ph.D.
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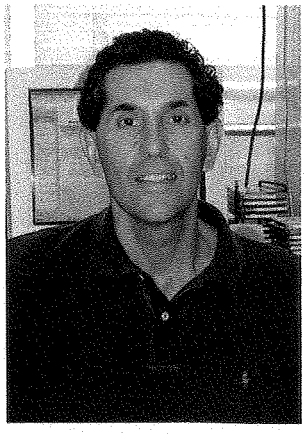

Our laboratory studies how the nervous system controls the muscles of breathing. Under this umbrella, our principle studies are divided into two main themes. Detailed descriptions of these research projects can be found at:
http://www.physiol.arizona.edu/RNLab/index.html.
A brief summary of these projects is given below.

1. We are interested in how inhibitory and excitatory neurotransmitters lead to the periodic excitation and inhibition of respiratory neurons, which underlies the cyclic breathing motions that we all take for granted. We are currently studying how prenatal nicotine exposure alters the development of respiratory neuron structure and function. These studies relate directly to neonatal breathing instability, including the Sudden Infant Death Syndrome (SIDS). Studies are done using electrophysiological methods in the *in vitro* brain stem spinal cord preparation of neonatal rat, and are buttressed by parallel *in vivo* studies in awake neonatal animals.

2. Neural control of the tongue muscles. The tongue is moved, shaped and stiffened by seven different muscles, all of which are innervated by the hypoglossal nerve. The tongue muscles participate importantly in breathing, swallowing, speech and facial expression. We focus principally on the breathing-related control of these muscles, which occurs spontaneously and is driven by central pattern generators located in the brainstem. Malfunction of these muscles is responsible for obstructive sleep apnea, swallowing disorders and speech impediments. Various chronic brain pathologies such as Parkinson's disease, Alzheimers disease, and stroke are associated with altered control of the tongue muscles, leading to the increased incidence of sleep apnea and swallowing disorders observed in these populations.

Andrew J. Fuglevand, Ph.D.
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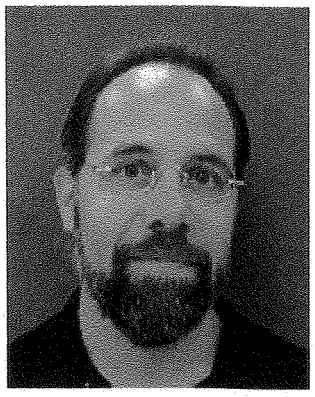

The broad goal of the work carried out in Dr. Fuglevand's laboratory is to understand how the mammalian nervous system controls the action of skeletal muscles to produce coordinated movements. A variety of experimental approaches are used are used in Dr Fuglevand's laboratory, including computer modeling and simulation, single motor unit recording, and microneurographic methods to record and stimulate single sensory and motor axons. His experiments address a range of topics from those related to how individual neurons integrate synaptic information to those associated with the development of methods to restore movement and sensation in paralyzed individuals. Of particular emphasis at present are studies designed to characterize the functional organization of the corticospinal pathways that underlie the control of hand and finger movements. He is presently a member of the Musculoskeletal and Rehabilitation Sciences Study Section of NIH, he serves as associate editor for Neuroscience Letters, and he was the 2006 recipient of the Delsys Prize for innovation in electromyography.

Elizabeth L. Glisky, Ph.D.
Professor, Associate Head, and Director of Graduate Studies
Department of Psychology
Affiliate, Evelyn F. McKnight Brain Institute

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Elizabeth Glisky's research interests include changes in memory and executive function that occur as a result of normal aging or age-related neurological conditions such as Alzheimer's disease. Recent collaborative work has focused on tracking longitudinal changes in cognitive function in a cohort of normally-aging older adults, and relating those changes to measures of brain integrity, genetic predisposition, and other health variables. The goals of this research are to understand the variability in the normal aging process, to identify early indicators of what might be abnormal aging, and to design and implement interventions that might be instrumental in enabling older adults to maintain optimal memory function into the oldest years. Dr. Glisky's work has been supported by the National Institute on Aging, the Arizona Biomedical Research Council, the Arizona Alzheimer's Consortium, and the Evelyn F. McKnight Brain Institute.

Katalin M. Gothard, M.D., Ph.D.
Assistant Professor of Physiology and Neurology
Affiliate, Evelyn F. McKnight Brain Institute

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Katalin Gothard obtained an M.D. degree in Romania, where she trained in neurosurgery, and a Ph.D. in Neuroscience at the University in Arizona. She received postdoctoral training at the California National Primate Research Center at UC Davis where she specialized in primate neurophysiology.

The broad goal of her research is to understand the neural basis of emotion. She uses non-human primates as a model system for normal and pathological emotions generated in the context of social behavior. The experiments involve eliciting emotions in freely behaving monkeys while recording neural activity from several brain areas in conjunction with cardiovascular and other autonomic measurements. These experiments reveal the real-time dynamic interaction of multiple systems implicated in emotion regulation and the mechanisms by which emotional responses produce immediate behavioral effects.

Dr. Gothard trains a postdoctoral fellow, Dr. Haiyin Chen, whose NIH-funded research is focused on the age-related changes in social and emotional behavior in macaque monkeys.

G. Alex Hishaw, M.D.
Evelyn F. McKnight Fellow in Behavioral
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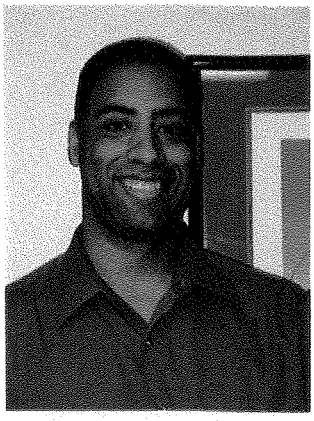

Dr. Hishaw is the current Evelyn F. McKnight Fellow in Behavioral Neurology and Neuropsychiatry. He previously completed a combined residency in Neurology and Psychiatry. His clinical and research activities include: Dementia; Behavioral Neurology; Neuropsychiatry; Intracarotid Amobarbital (Wada) Test; and Head Injury. He has participated in a number of clinical research studies directed at improved diagnosis and management of Alzheimer's Disease.

This summer, Dr. Hishaw will join the faculty at the University of Arizona with a joint position as an Assistant Professor of Clinical Neurology & Psychiatry. He will direct Dementia and Head Injury management on the UPH-Kino campus.

Dr. Hishaw's participation in the Evelyn F. McKnight Brain Institute centers on the interface between normal age-related changes in cognition and alterations in cognition that signify the transition to more ominous entities, e.g., Mild Cognitive Impairment (MCI), Alzheimer's Disease, and other dementing illnesses. He will continue his work in the McKnight Brain Institute through a project with Dr. Gene Alexander involving neuroimaging of subjects being followed for senescent changes in cognition.

Lan T. Hoang
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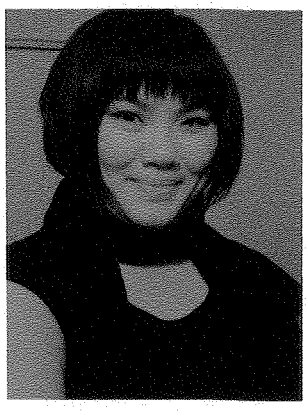

The central goal of Lan's research interests lie in investigating the plasticity of the nervous system. Specifically, Lan's doctoral thesis is aimed at exploring the neural correlates of reward by studying the characteristics of the dopaminergic neurons found in the ventral tegmental area and how these may change during normal aging. High density neural recording ensembles as well as molecular imaging techniques are being utilized to achieve a greater understanding of not only the systems level response to reward, but also to gain insights into the possible processes that underlie age-related cognitive decline.

Nathan Insel
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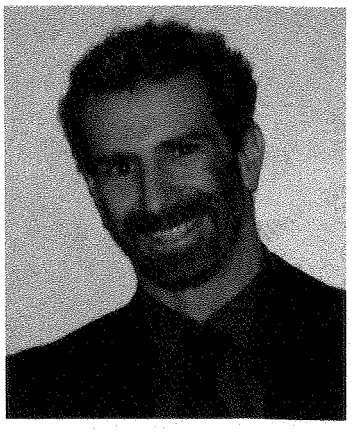

Nathan Insel is currently investigating how old age affects a rat's ability to make a decision between two high-valued alternatives and then learn from the decision's outcome. Nathan's dissertation work has begun not only to assess the behavioral differences between aged and young adult rats in a decision task, but also to use molecular biology imaging techniques to identify brain areas that respond to a situation of increased decision conflict, and electrophysiological techniques to assess the mechanisms underlying age-related changes in overcoming conflict and learning from decision outcomes. These experiments have focused primarily on medial frontal regions of the brain such as the anterior cingulate cortex. Before joining the Barnes lab Nathan received a bachelor's degree in biology and neuroscience at Oberlin College, and has since earned a master's degree in psychology at the University of Arizona.

Alfred W. Kaszniak, Ph.D.
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Dr. Kaszniak received his Ph.D. in clinical and developmental psychology from the University of Illinois in 1976, and completed an internship in clinical neuropsychology at Rush-Presbyterian-St. Luke's Medical Center in Chicago. He is currently Head of Psychology, Director of Clinical Neuropsychology, Director of the Arizona Alzheimer's Disease Center Education Core, an affiliate faculty member of the Evelyn F. McKnight Brain Institute, and a professor in the departments of psychology, neurology, and psychiatry at The University of Arizona. His publications (7 books and over 145 journal articles and scholarly book chapters) focus primarily on neuropsychological aspects of aging and age-related disorders of the central nervous system, particularly Alzheimer's and Parkinson's diseases. Dr. Kaszniak has served on the editorial boards of several journals in neuropsychology and gerontological psychology, has been an advisor to several national institutes and agencies concerned with aging and Alzheimer's disease, and is a Past-President of the Section on Clinical Geropsychology (Division of Clinical Psychology) of the American Psychological Association.

Thabelo Khoboko M.Sc.(Med)
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Thabelo is working with Carol Barnes to study mechanisms underlying neuronal processing of spatial memory in the dentate gyrus during aging. Spatial memory is one of the cognitive functions compromised in normal aging. The dentate gyrus has been found to be the region of the hippocampal formation that is most susceptible to functional alterations that occur during normal aging. Although spatial selective discharge has been described in granule cells there have been no studies conducted to compare ensemble spatial firing characteristics of granule cells in young and old rats. Thabelo hails from the mountain kingdom of Lesotho in Southern Africa. She received her B.Sc. (Hons) degree from the University of Leicester. Thabelo went on to complete her M.Sc.(Med) degree at the University of Cape Town after she was one of two women to be the first to be awarded the Levi Montalcini Fellowship in Neuroscience for African Women from the Fondazione Rita Levi Montalcini and the International Brain Research Organization.

James P. Lister, Ph.D.
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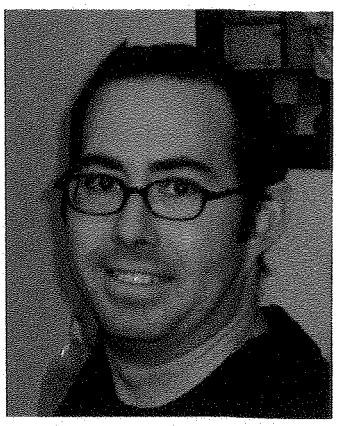

Dr. Lister received his doctoral training at Boston University researching the effects of prenatal protein malnutrition on the neuroanatomy of the adult rat hippocampal formation. After studying structure throughout graduate school, he came to NSMA to learn more about function, and is involved in efforts for automating whole brain imaging as well as projects that use the expression of immediate early genes (such as Arc and Homer) to map behavior-induced neural circuits. Current progress on automated brain imaging has focused on work with collaborators at Rensselaer Polytechnic Institute to automate montaging of high resolution confocal images encompassing entire cortical regions. He is also involved in using 3D catFISH to analyze encoding in the hippocampus and cerebral cortex in young and old animals to assess age-related impairments in the ability of these structures to represent information. 3D catFISH is a technique that combines fluorescent in situ hybridization with high resolution confocal microscopy of immediate-early gene expression to evaluate the exact neural circuits activated by behavior. Behaviorally relevant neuronal activity is known to induce the expression of certain immediate early genes, such as Arc. The localization of Arc mRNA within cellular compartments (nucleus vs. cytoplasm) is consistently time-dependent, allowing the researcher to probe multiple time points within the same animal. Current projects examine the effects of exercise on Arc expression and age-related differences in Arc expression in the hippocampus and entorhinal cortices during behavior.

Bruce L. McNaughton Ph.D.
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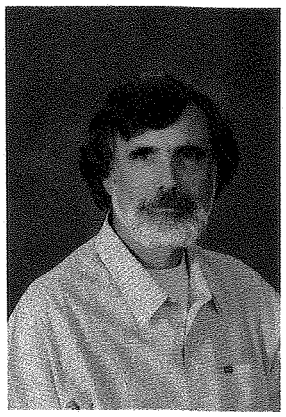

Dr. McNaughton's research focuses on the molecular, cellular and brain system mechanisms of memory and memory disorders associated with aging and brain damage. His early career concentrated on the biophysics of long-term synaptic potentiation and the role of this process in associative information storage. This work was highlighted by the first demonstration that "Hebbian" principles of association, which form the basis of all neural network learning algorithms, are embodied in the actual dynamics of experience dependent synaptic plasticity. In the last 15 years, Dr. McNaughton has been at the forefront of development of methods to study the large-scale interactions of neurons in the intact brain during the encoding, storage, recall and consolidation of memory. Methods developed in his laboratory at the University of Arizona now make it possible to record from several hundred cortical neurons during learning experiments in animals, providing an unprecedented window on how neurons cooperate during cognitive processing. These methods are also being directed towards the development of neuroprosthetic systems that will use direct brain recording to control muscle activity in patients with spinal injury. At the other end of the scientific spectrum, Dr. McNaughton is a key member of an interdisciplinary team involved in the development of immediate-early gene activation markers of neural activity in the brain. This method permits visualization of the recent history of activity in the brain at cellular resolution, thus allowing identification of not only which areas of the brain are activated during cognitive processing, but which specific neurons. This method will provide an important complement to non-invasive, but lower resolution, functional neuroimaging studies using magnetic resonance. Dr. McNaughton teaches courses in Neural Coding and Computation and Mammalian Neurophysiology and supervises postdoctoral associates and graduate students in Psychology, Neuroscience, Physiological Sciences, Applied Mathematics and Biomedical Engineering. He also provides research mentorship in the neurosciences to numerous undergraduate research assistants through the Undergraduate Biology Research Program (UBRP) at the University of Arizona.

Lynn Nadel, Ph.D.
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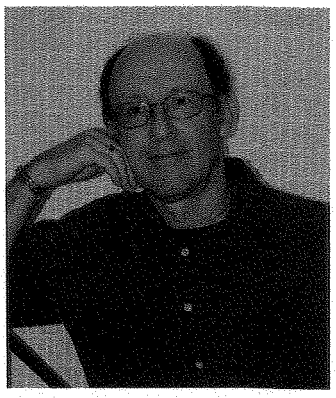

Dr. Nadel's research program focuses on the role of the hippocampal formation in memory and spatial cognition. Current projects involve: (1) neural activation, as measured with fMRI, during processing encoding and retrieval of episodic information; (2) memory reconsolidation; (3) the effects of stress on memory; (4) the role of sleep in memory consolidation; (5) the neural and cognitive basis of Down syndrome. Dr. Nadel is a Regents' Professor at the University of Arizona and recipient of the Grawemeyer Prize in Psychology (2006).

Marsha R. Penner, M.S.
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In general, my primary research interest is directed at understanding how the brain changes during the normal aging process and how these changes may contribute to impaired memory function. My dissertation research has focused on whether age-associated changes in memory promoting immediate-early genes occur in aged rats compared to adult rats. Recent work has also been directed at the question of whether age-associated changes in immediate-early gene expression may be regulated by DNA methylation.

Mary A. Peterson, Ph.D.
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Dr. Peterson investigates how we perceive the world visually. Specifically, she uses cognitive neuroscience techniques (e.g., ERPs and behavioral methods) to investigate:

- the competitive processes producing the perception of shape, and how they are affected by context;
- the relationship between perception and memory;
- the processes used to learn new objects and to recognize familiar ones;
- how attention affects perception;
- the illusory perception of color in synesthesia.
- how brain damage and aging affect the perception of and memory for objects and faces

Dr. Peterson is a Fellow of the American Psychological Association and the Association for Psychological Research. She is also an elected member of the Society of Experimental Psychologists.

Naomi E. Rance, M.D., Ph.D.
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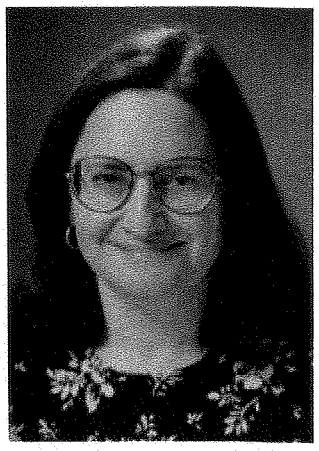

Dr. Rance currently divides her time between clinical practice as a Neuropathologist, research in Neuroendocrinology and teaching in the College of Medicine. The overall objective of Dr. Rance's laboratory is to characterize and understand the events that occur in the human central nervous system in response to the ovarian failure of menopause. An equally important goal is to contribute to our knowledge of the neuroendocrine mechanisms that regulate human reproduction. Although complex, the neural basis of reproductive function is amenable to experimentation because of the known feedback circuits. Control mechanisms can be studied at many different levels, ranging from plasma hormone levels, to gene expression in subsets of hypothalamic neurons. In the last 20 years, our laboratory has made considerable progress in characterizing the effects of menopause on neuronal morphology and neuropeptide gene expression in the human hypothalamus. This research has provided information on the site of estrogen negative feedback on LH secretion in the human and neuroendocrine regulation in postmenopausal women.

Eric M. Reiman, M.D.
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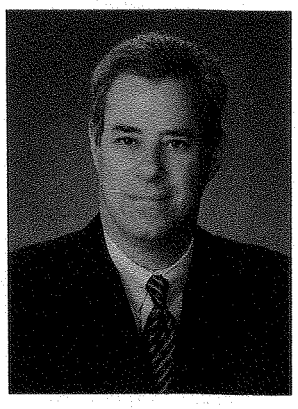

Dr. Reiman is Executive Director of the Banner Alzheimer's Institute, Clinical Director of the Neurogenomics Division at the Translational Genomics Research Institute (TGen), Professor and Associate Head of Psychiatry at the University of Arizona, and Director of the Arizona Alzheimer's Consortium. His research interests include brain imaging, genomics, the unusually early detection and tracking of Alzheimer's disease (AD), and the rigorous and rapid evaluation of promising Alzheimer's disease-slowing and prevention therapies. He and his colleagues have used imaging techniques to detect and track brain changes in cognitively normal carriers and non-carriers of the apolipoprotein E (APOE) ε4 allele, a common Alzheimer's susceptibility gene. They have shown how imaging techniques could help identify effective primary prevention therapies without having to study a many healthy volunteers or wait many years to determine if they go on to develop symptoms. They recently published the first genome-wide association studies of Alzheimer's disease and the individual variation in normal human memory. Using the latter information, they have begun to evaluate treatments to improve memory in older people.

Linda L. Restifo, M.D., Ph.D.
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The primary focus of Dr. Restifo's research program is the genetic control of brain development, with an emphasis on how genetic mutations and environmental exposures cause developmental brain disorders associated with mental retardation and autism. The long-term goal is to devise strategies for identifying safe and effective treatments for patients with brain-development disorders. Dr. Restifo further proposes that risk factors for aging-related neurodegeneration will be found among the genetic and environmental influences on brain development. The Restifo lab uses bioinformatics, molecular genetics, and cell biology, primarily in the fruit fly model system. Dr. Restifo and colleagues have developed a novel cellular bioassay, based on primary culture of developing brain cells, that can reveal neuronal defects caused by mutations or toxins. They are currently conducting a proof-of-concept drug screen and will be doing cross-species validation studies in the near future. Dr. Restifo is a Professor of Neurobiology and Neurology at the University of Arizona.

Lee Ryan, Ph.D.
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Dr. Lee Ryan received a Ph.D. in Cognitive and Clinical Psychology at the University of British Columbia in 1992. She is currently a faculty member of the Evelyn F. McKnight Brain Institute at the University of Arizona as well as the Director of the Cognition and Neuroimaging Laboratories, making magnetic resonance imaging (MRI) technology available to cognitive neuroscience researchers on campus. Her research focuses on the neural basis of memory and understanding how age-related changes in brain function affect memory in older adults. She has a special interest in memory disorders such as Alzheimer's Disease, and is currently conducting research using various MRI methods as a tool for detecting subtle markers of change in brains of individuals with risk for Alzheimer's disease prior to the onset of memory impairments.

As an associate professor in the Cognition and Neural Systems program and the Clinical Neuropsychology program at the University of Arizona's Department of Psychology, Dr. Ryan teaches undergraduate classes in human memory and graduate level courses such as Human Brain Behavior Relationships, Cognitive Neuroscience, and Principles of Neuroanatomy. As a clinical psychologist, Dr. Ryan works with individuals and families who are coping with chronic and progressive diseases that effect cognitive functioning, including multiple sclerosis, Parkinson's disease, and Alzheimer's disease.

Rachel Samson, Ph. D.
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Supervisor: Dr. Carol A. Barnes

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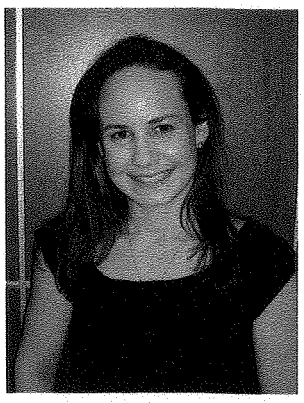

Dr. Samson's research examines the effects of normal aging on the network activity underlying emotional learning, with particular attention aimed at the age-related modifications on amygdala physiology and function. Older adults use different affective-cognitive strategies and show greater memory for positive stimuli, as compared to younger adults. Her project combines methods of fluorescent *in situ* hybridization using the CATFISH technique and multiple single cell recordings *in vivo*. The reinforcer devaluation task is used as an animal model for goal-directed behaviour, allowing measurement of the differences in network activity while the incentive value of a reward changes. By comparing results from young adult and old rats, her work will provide insight into the mechanisms of age-related modifications in emotional perception and processing. This will help distinguish between the normal aging process and pathological changes in cognitive function associated with AD.

Lesley A. Schimanski, Ph.D.
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Dr. Schimanski's research focuses on age-related changes in hippocampal neurophysiology and spatial memory. She is examining whether aged rats learn and remember locations in an environment differently from young rats using a spatial version of a classical eyeblink conditioning task. Her project will determine whether properties of "place cells" in the hippocampus are altered in aged rats compared to young rats during this task. This work will shed light on the link between encoding of spatial information by hippocampal cells, and use of this information by aged animals in the context of a behavioral task. Dr. Schimanski was trained as an electrophysiologist and behavioral neuroscientist, and is currently a Post-Doctoral Associate at the University of Arizona and the Evelyn F. McKnight Brain Institute.

Robert S. Sloviter, PhD
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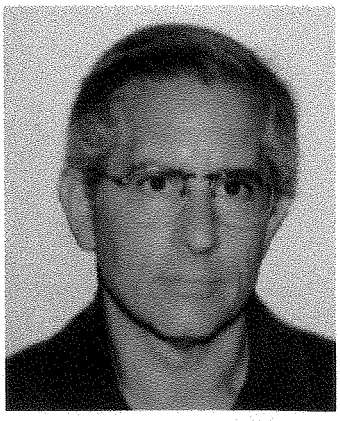

Dr. Sloviter's laboratory focuses on the structure and function of the hippocampus, with special emphasis on the role of hippocampal malfunction in temporal lobe epilepsy. The methods used include neuroanatomy, electrophysiology, and immunocytochemistry. One planned avenue of research will address the vulnerability of the aged brain to injury and its possibly altered propensity to develop epilepsy.

Alexander Thome, B.A
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Mr. Thome's research focuses on studying populations of neurons in the temporal lobe of awake and freely behaving primates using molecular imaging techniques as well as multiple single unit recordings. He is using freely navigating primates in real and virtual environments in combination with molecular imaging techniques. In particular, he is seeking to understand whether ensembles of neurons in navigation related structures show patterns of activation similar to those seen in rodents. In addition, the project aims to understand whether there exist differences in patterns of ensemble activity between real and virtual environments. This work will clarify basic questions regarding primate temporal lobe function and provide insight into the extensibility of findings in rodents to higher primates. A second set of experiments, using data from young and old primates, is aimed at understanding the functional role of oscillations in the primate temporal lobe and whether these change with age. Mr. Thome received an interdisciplinary B.A in Cognitive Science at the University of Arizona.

Michelle Valfre, B.A.
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Michelle's current research examines relationships between decision-making and aging. Of particular interest are factors that are likely to influence decisions made by older adults, such as age-related changes in cognition and social behavior. In order to investigate how older adults' individual differences affect decision-making, scores from neuropsychological tests measuring executive function are correlated with gain-optimizing performance on a decision-making task. Use of theory of mind (the ability to impute mental states to others) is assessed by self-report measures and contrasted to the frequency and degree of its use by younger adults. Future research will investigate neural correlates of decision-making in older adults. Michelle's other interests include age-related changes in memory, gender differences in the brain, and the application of magnetic resonance techniques to the early detection of pathological aging.

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Katrin Walther is a postdoctoral fellow in the Cognition & Neuroimaging Laboratories. She is working on a project assessing the utility of diffusion-weighted magnetic resonance imaging (DWMRI) in identifying early neuropathological markers of Alzheimer's disease (AD) in healthy older adults with increased risk for AD, which might enable early diagnosis and early treatment. She earned her Ph.D. in Psychology at the University of Leipzig in Germany where her research was focused on the development of assessment and intervention methods for individuals with neurological impairment.

Evelyn F. McKnight Brain Institute Meeting Participants
University of Alabama at Birmingham

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Director, Evelyn F. McKnight Brain Institute

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Michael Brenner, Ph.D.
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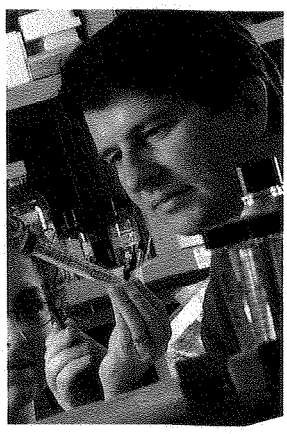

Dr. Sweatt's interest is in understanding the biochemical mechanisms underlying learning and memory. One of the major advances in neurobiology in the last century was the formulation of the general theory that changes in synaptic connections between neurons underlie information storage in the CNS. One powerful offshoot of this general theory is that it allows a reductionist approach by studying the mechanisms of long-lasting synaptic plasticity in vitro insights can be gained into the mechanisms of learning and memory in vivo. Using this rationale, for the last decade Dr. Sweatt's laboratory has been investigating the biochemical mechanisms subserving the induction and maintenance of long-term potentiation in the hippocampus. In general the lab's focus has been on signal transduction, with a particular emphasis on the role of protein kinases in LTP. Dr. Sweatt's lab and many other labs, has indicated that four protein kinases play particularly prominent roles in LTP: PKA, PKC, CaMKII, and erk MAPK. By and large PKC and CaMKII have achieved notoriety for their roles as molecular information storage devices; autonomously active forms of these kinases subserve the maintenance of early LTP. In contrast, PKA and MAPK appear predominantly to be involved in triggering the induction of early and late stages of LTP. While Dr. Sweatt's lab has investigated all of these protein kinase cascades over the last decade, of late they have focused most of their effort on studies of the MAPK and PKC cascades.

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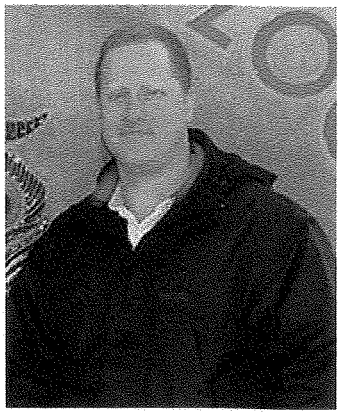

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Jon's dissertation research deals with evaluating and understanding the role of potassium channel interacting proteins (KChIPs) in hippocampal-dependent learning and memory. He uses knockout mice to characterize the importance of two specific proteins, KChIP2 and KChIP3. To address their specific roles in learning and memory, he uses behavior, extracellular electrophysiology, and molecular methodology. Overall, his research will aid in the understanding of neuronal excitability and its effects on learning and memory.

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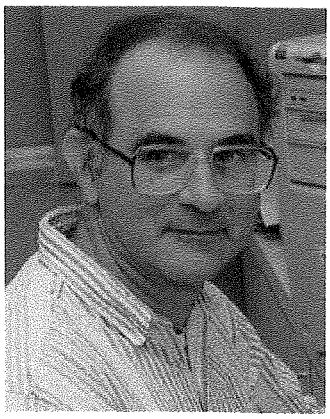

Dr. Brenner's laboratory studies the molecular biology of astrocytes, the most common cell type in the central nervous system (CNS). Astrocytes are responsible for many of the homeostatic controls in the CNS, such as maintaining the blood-brain barrier and proper neurotransmitter levels. Astrocyte serve as precursors for neurons and oligodendrocytes during development, and also serve as stem cells for the production of these cell types in the adult. CNS injury stimulates astrocytes to undergo a reactive response, which contributes to healing but can also lead to further damage. The work focuses on the transcriptional regulation of a gene encoding an intermediate filament protein specific to astrocytes, glial fibrillary acidic protein (GFAP), and on the biological role of this protein. The GFAP gene is of interest because it is turned on as astrocytes mature, and its activity increases dramatically during the reactive response. Thus, study of GFAP transcription will yield insights into mechanisms governing development, reaction to injury, and cell specificity, ultimately allowing these processes to be manipulated.

Dr. Brenner's laboratory has also discovered that heterozygous coding mutations in the GFAP gene are responsible for Alexander disease, a rare but fatal neurological disorder. Interestingly, although this establishes that the primary genetic defect in this disease is in astrocytes, the infantile form of Alexander disease is marked by massive myelination defects, and the later onset forms by neuronal dysfunction. Thus the study of this disorder not only has direct clinical implications, but also will reveal critical interactions between astrocytes and oligodendrocytes and between astrocytes and neurons that occur throughout the life span.

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As the Scientific Director of the Electrophysiology Neuroscience Blueprint Core Facility at UAB, Dr. Campbell examines electrophysiological phenotype of various genetically engineered mouse models in terms of cellular biophysics, baseline synaptic transmission and plasticity. The Core offers investigators at UAB and other institutions access to a state-of-the-art facility for mouse characterization. As a postdoctoral fellow in Dr. J. David Sweatt's laboratory, Dr. Campbells' research focuses on the effects of altering epigenetic mechanisms on synaptic transmission and ion channel activity. Her research involves the use of electrophysiological techniques utilizing *in vitro* brain slice preparations to ascertain how alterations in DNA methylation and histone acetylation affect the function of ion channels involved in long-term plasticity. Her broader questions are aimed at discerning if modifications in epigenetic mechanisms lead to changes in the expression or function of ion channels in neurodegenerative diseases.

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Dr. Dobrunz's research program uses electrophysiological approaches to study synaptic transmission and regulation of presynaptic properties at synapses in the hippocampus. Using hippocampal brain slices and cultured hippocampal neurons from rodents, the lab studies short-term plasticity and the cellular and molecular mechanisms underlying the activity dependent modulation of neurotransmitter release. Projects in the lab include the study of mechanisms and effects of target-cell specific short-term plasticity, including the role of postsynaptic influences on the formation and function of presynaptic terminals. The lab also studies the changes that occur in presynaptic function during normal postnatal development and during normal aging.

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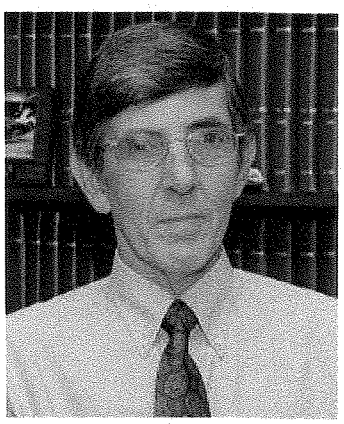

Dr. Hablitz's research is centered on understanding control of activity in local cortical circuits. He is using studies on synaptic transmission to further understand basic biophysical properties of mammalian central neurons, as well as to explore the pathophysiology of experimental epilepsy. Whole-cell voltage-clamp recordings from visually identified neurons are used in *in vitro* brain slice preparations. The goal of these studies is to determine the types of synaptic interactions present among pyramidal cells and interneurons in neocortex and how these patterns change over the lifespan. A particular goal is to understand how dopamine, an important modulator of working memory, affects excitability of individual neurons in prefrontal cortex. Additional studies involve the use of imaging techniques to directly visualize activity in presynaptic nerve terminals. These studies examine modulation of neurotransmitter release in normal neocortex and animal models of cortical dysplasia.

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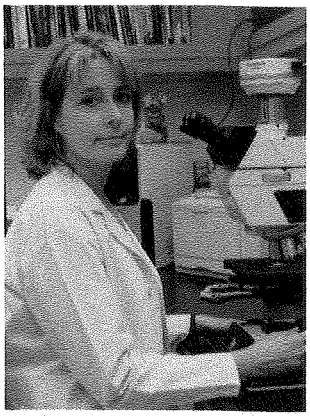

The main goal of Dr. Kadish's research is to elucidate the role of white matter pathology in age-related cognitive deficits.

Her studies indicate that relatively early in the aging process changes occur in the crosstalk between astrocytes and oligodendrocytes that ultimately lead to malfunctioning of oligodendrocytes, and demyelination of axons. The studies also indicate that cholesterol metabolism is disturbed early in the aging process in the white matter, specifically in astrocytes, and since the astrocytes are the main source of cholesterol in the brain, and the main source of cholesterol for oligodendrocytes, this likely leads to changes in the myelin sheath. The lab investigates these changes in astrocyte functioning and communication in the white matter, and the development of cognitive impairments, using behavioral, immunohistochemical and molecular biology approaches.

A secondary research interest is the role of vascular (white matter) pathology in Alzheimer's disease. The relationship between small infarcts and cognitive decline is still not clearly defined. The lab has shown that small ischemic infarcts increase parenchymal Aß deposition and worsen cognitive outcome. Further, they have found that infarcts involving the white matter have a significantly worse outcome.

Part of the current studies includes the use of therapeutic agents that may be promising in the alleviation or delay of neural and cognitive changes that occur with age.

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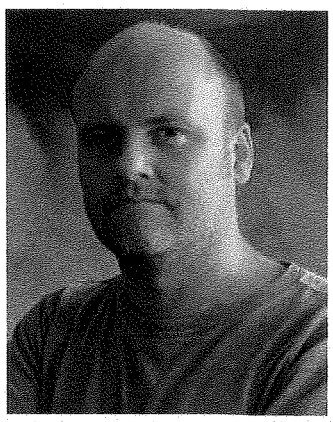

Dr. Lester's lab has been researching the role of CNS nicotinic acetylcholine receptors (nAChRs) in tobacco addiction and central synaptic transmission. nAChRs are ligand-gated ion channels composed of five individual protein subunits that cause neuronal excitation when bound and activated by synaptically released neurotransmitter, acetylcholine, or exogenous drugs like nicotine. In respect to drug addiction, they have been studying how exposure of these receptors to nicotine *in vivo* leads to persistent changes in hippocampal neuronal network activity following long-term withdrawal of the drug. In addition they have uncovered a unconventional form of diffuse synaptic signaling through nAChRs in the brain implying that this transmitter system may participate in volume transmission. Molecular biological studies have characterized at least ten receptor subunits that can be assembled together in numerous combinations giving rise to a wide variety of nAChRs with distinct functional roles. It is because of this diversity that nAChRs have been implicated in a range of CNS behaviors from pain sensation to learning and memory, and multiple pathological states such as aging, epilepsy and schizophrenia.

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Dr. Lubin's research is primarily directed towards characterizing the role of epigenetic mechanisms, such as histone modifications, DNA methylation, and signaling cascades that mediate the interaction of the NF-κB transcription factors to chromatin and determine how they participate in the regulation of gene expression as they relate to learning and memory and memory deficits associated with epilepsy. Her research program focuses on neurons and synapses in the hippocampus, an area of the brain that plays an important role in learning and memory and the development of epilepsy in humans. She is investigating the epigenetic regulation of brain derived neurotrophic factor (BDNF) transcripts during memory formation. This has lead to the discovery that exon-specific gene regulation of BDNF transcripts are dynamically regulated by DNA methylation and specific histone modifications in hippocampus during memory consolidation. Current work also includes an assessment of histone deacetylase (HDAC) inhibitors and demethylating agents that may be promising in the mitigation or disruption of cognitive changes that occur with epilepsy.

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My lab is currently investigating the role of estradiol in hippocampal synapse density, synaptic plasticity and learning. We are particularly interested in determining how loss of estradiol during aging impacts hippocampal function and whether hormone replacement therapy can activate estradiol-dependent mechanisms to restore normal synaptic function in hippocampus as well as hippocampal dependent learning and memory. Ovariectomized female rats treated with estradiol at various intervals following ovariectomy are used as a model system. Experiments involve electrophysiological measurements of NMDA currents, synaptic transmission, and long-term plasticity in acute brain slices. We have recently reported that estradiol increases NMDA transmission mediated by NR2B containing receptors and that is causally related to the heightened LTP induced by estradiol. Determining how estradiol and hormone replacement affects hippocampal function could lead to development of therapies to alleviate hormone-dependent memory loss in aging.

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As Scientific Director of the UAB Behavior Core, Dr. Miller provides expertise in the design, implementation and analysis of behavior experiments, with a primary focus on learning and memory. The Behavior Core encourages investigators from UAB, as well as external collaborators at universities across the country, to take advantage of our facilities to investigate the behavioral effect of molecular and genetic models of a variety of cognitive disorders, such as Alzheimer's disease. As a postdoctoral fellow in Dr. Sweatt's laboratory, Dr. Miller is investigating the role of DNA methylation in the long-term storage of memories. This line of research has important implications because DNA methylation has been linked to normal memory formation, memory decline with age, and schizophrenia. In addition, Dr. Miller collaborates with another UAB McKnight investigator, Dr. Gavin Rumbaugh, on a project in his laboratory aimed at understanding the molecular basis of the earliest stages of memory formation. One goal of this research is to develop therapeutic agents that will enhance the formation of new memories and the maintenance of old memories.

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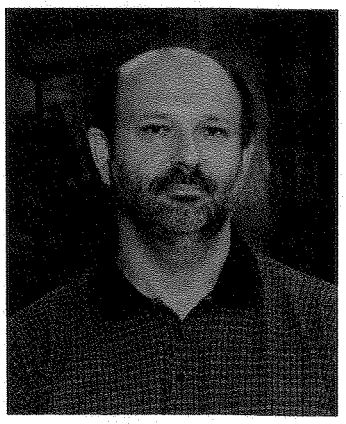

Glial cells were long considered to serve merely as the supporting cast and scenery against which the starring neuronal roles would be played out. Relatively recent evidence, however, indicates that glial cells are intimately involved in many of the brain's functions, including its computational power. Our research has been instrumental in demonstrating a novel functional role for glial cells. Hence, astrocytes, a sub-type of glial cell, can exocytotically release the neurotransmitter glutamate and, in turn, that glutamate released from astrocytes can signal to adjacent neurons. Indeed, by releasing glutamate, astrocytes can modulate synaptic transmission in response to experimental stimuli. Since intracellular calcium ion levels critical for secretion from astrocytes are within the physiological range, this release of glutamate from astrocytes could represent an additional site for modulation of synaptic transmission and integration in the CNS. Some current work also employs atomic force microscopy (AFM) in examining the interactions between pairs of molecules such as syntaxin and synaptobrevin that reside astrocytes (and within synaptic junctions) and that are critical for glutamate release.

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Dr. Roth's research focuses on applying integrative methods to examine many aspects of spatial ecology. He uses molecular, neurophysiological, behavioral, and ecological techniques to address questions related to spatial environmental interactions, animal navigation, and spatial learning and memory. Currently he is examining the role of molecular epigenetic mechanisms in maintaining spatial representations of the environment within the hippocampus.

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The goal of Dr. Roth's research is to determine how early social experiences influence the developing brain. She is particularly interested in the lasting effects of early-life adversity on molecular mechanisms of synaptic plasticity, and whether this contributes to deficits in cognition and emotion across the lifespan. Her current research efforts utilize a rodent model of caregiver maltreatment to understand the relationship between early-life adversity and the dysregulation of epigenetic molecular mechanisms underlying gene expression and memory formation. Behavioral and biochemical approaches are used in infant, adolescent, and adult animals to address this relationship.

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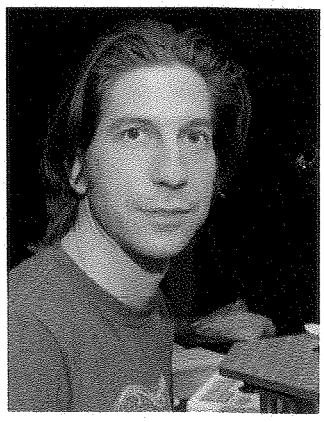

Dr. Rumbaugh's lab focuses on signaling pathways that are triggered by activation of NMDA receptors following vesicular release of glutamate from synaptic terminals. Activation of these receptors is critical for long-term changes in synaptic strength, which is believed to be one of many neural mechanisms that contribute to learning and memory in animals. Many of these signaling pathways (an example would be Ras/ERK activation) can regulate trafficking of AMPA receptors (AMPARs) to and from the synapse. Because AMPAR trafficking underlies certain forms of long-term synaptic plasticity, understanding the signaling pathways that regulate this trafficking will likely uncover mechanisms utilized during acquisition, consolidation and retrieval of memories. In addition, emerging evidence from several lines of research suggests that many common neurological disorders including Alzheimer's, schizophrenia and familial mental retardation are associated with synaptic dysfunction. Therefore, understanding signaling mechanisms that subserve synaptic plasticity and learning may help unravel the pathophysiology of common brain afflictions.

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Many diseases are linked to dysregulation of second messenger signaling cascades. One important second messenger system is the phosphoinositide (PI) system, in which inositol lipids function as second messengers and cofactors for many cellular activities stimulated by growth and trophic factors, hormones, cytokines, and neurotransmitters. Dr. Theibert's research focuses on investigating the intracellular targets for several of the PI second messengers in the nervous system. They are particularly interested in the function of PtdInsP3 in neurons and glia, since they have demonstrated that this lipid is required for cells to extend processes, termed neurites, in response to trophic factors and extracellular matrix. Neurites eventually form mature axons and dendrites, which contact each other at synapses, and allow for information transfer between neurons. Using biochemical and molecular techniques, they have isolated and cloned several novel phosphoinositide receptors from brain. One of these receptors is involved in regulating vesicle trafficking and the actin cytoskeleton, two activities which are involved in neurite outgrowth and new synapse formation. Studies are underway to determine the role of these receptors in neuronal development and synapse formation, and the molecular mechanisms which regulate receptor expression, targeting to intracellular compartments, and modulation of activity. Several potential homologues of these receptors are present in the genetically tractable organism, Saccharomyces cerevisiae, which allows us to use yeast genetics to complement the biochemical and molecular approaches in dissecting the function of these brain phosphoinositide receptors.

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The research in the laboratory of Dr. Linda Overstreet-Wadiche is focused on understanding the role of adult generated neurons in a region of the brain that is associated with learning and memory. Most neurons are generated during embryogenesis, but in the hippocampus newborn neurons are continuously produced throughout adulthood and growing evidence suggests that they participate in hippocampal-dependent cognitive and emotive functions. The proliferation, survival and integration of newborn neurons are regulated by many factors including aging and environmental enrichment, thus adult neurogenesis provides a link between experience and neural regeneration in the adult brain. Dr. Overstreet-Wadiche's lab uses transgenic mouse models and electrophysiological techniques to explore how experience-dependent factors control adult neurogenesis and how newborn neurons in turn participate in hippocampal network activity.

Scott Wilson, Ph.D.
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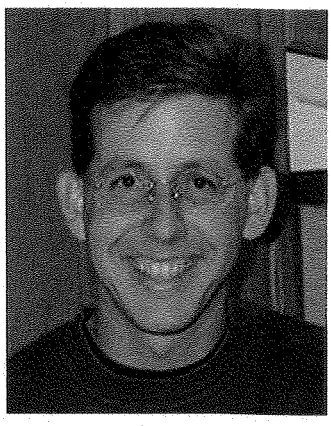

The focus of Scott Wilson's research is to investigate how regulated protein turnover by the ubiquitin proteasome pathway controls nervous system development and function. By using a combination of genetics, biochemistry, electrophysiology and behavioral analyses, the Wilson laboratory has investigated ubiquitin-signaling events that are required for synapse maturation, the induction of synaptic plasticity, learning and memory in mice. Results from these studies demonstrate the importance of localized ubiquitin recycling to maintain efficient protein turnover at synapses and indicate that changes in ubiquitin homeostasis may contribute to neurodegenerative diseases.

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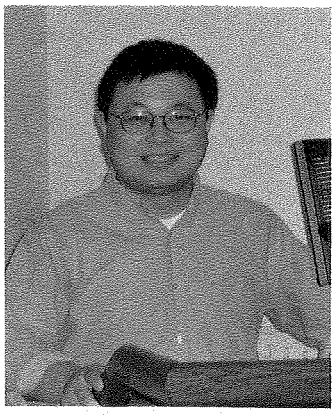

High resolution and high sensitivity molecular imaging provides critical insights in understanding cellular and molecular interactions in living tissues. Imaging depth and molecular contrast are two essential problems that need to be solved in *in vivo* microscopy. Having worked in the field of ultrafast laser spectroscopy for many years, I am now focusing on developing imaging techniques that can provide increased imaging depth with abounding molecular contrasts in optical microscopy by taking advantage of achievements from nonlinear optics and laser spectroscopy. For example, one of our approaches is using near infrared ultrafast laser pulses to generate fluorescence of fluorophores through multiphoton excitation, which provides imaging depth up to 500 micron (in brain slices) with sub-micron resolution and additional benefits, such as high photo-bleach thresholds and confined photo-damages. The multiphoton fluorescence microscopy makes it possible for us to image mouse brains in vivo through thinned skulls or cranial windows. There are certainly a lot more nonlinear optical processes that can provide useful molecular contrasts in imaging; for example, second harmonic, third harmonic, coherent anti-Stocks Raman scattering and transient absorption have been explored in various cell and tissue imaging. Through the newly established Neuroimaging Core in the Alabama Neuroscience Blueprint Core Center, we will try our best to make the most advanced microscopy techniques available to the neuroscience research community.

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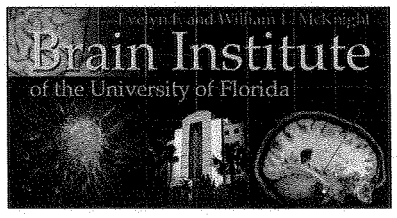

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Dr. Steindler received his doctorate in Anatomy and neurosciences from the University of California, San Francisco. After postdoctoral studies at the Max-Planck-Institute for Biophysical Chemistry in Germany, Dr. Steindler began his studies of brain development and injury as an Assistant Professor of Anatomy at Michigan State University. He is currently the Executive Director of the McKnight Brain Institute of the University of Florida, and the Joseph J. Bagnor/Shands Professor of Medical Research, a member of the Program in Adult Stem Cell Biology and Regenerative Medicine of the University of Florida College of Medicine, and an advisor to the California Institute of Regenerative Medicine. He also serves on the scientific advisory board for the Michael J. Fox Foundation for Parkinson's Research. Besides directing a large developmental neurobiology group, and teaching medical school neuroscience, Dr. Steindler has been studying the growth and transplantation of brain and stem cells for over 25 years. He is also responsible for reviewing manuscripts and grants for a variety of journals and funding agencies, including formerly chairing a brain repair and stem cell-related review panel at the National Institute of Neurological Diseases and Stroke in the National Institutes of Health, and he retains a position on the editorial boards of the following journals: The Journal of Neuroscience, GLIA, Experimental Neurology, and Brain Research. His recent papers in the international journals of medicine and science, The Lancet, and Proceedings of the National Academy of Sciences, set forth plans for the use of stem cells and regenerative medicine for a variety of neurological diseases, including Parkinson's Disease, age-related memory loss and cancer.

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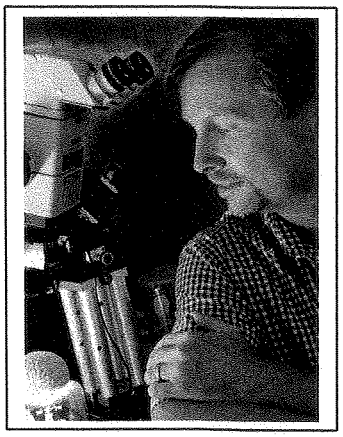

My research program utilizes a combination of behavioral characterization with biochemical, molecular, and electrophysiological techniques to obtain a vertically integrated perspective on neural aging, from the molecular to the cognitive level. The two main goals of the lab are to identifying mechanisms for age-related memory impairment and test treatments to alleviate memory deficits. Regulation of Ca^{2+} is thought to play a role in age-related neurodegeneration and the synaptic plasticity that underlies memory. Our research shows that rapid forgetting is associated with a shift in the threshold for Ca^{2+}-dependent synaptic plasticity, LTP and LTD, indicating that the shift in the balance of LTD/LTP contributes to synaptic and cognitive impairment with age. Subsequent research clarified the mechanisms and showed that age-related changes in synaptic plasticity are due to altered Ca^{2+} regulation involving voltage-dependent Ca^{2+} channels and intracellular Ca^{2+} stores interacting with processes for cell excitability. Moreover, a shift in the activity of LTD/LTP signaling mechanisms (e.g. phosphatase/kinase activity) was found to underlie the decrease in synaptic strength and correlate with memory impairment. These signaling cascades impinge on transcriptional regulation and recent work has examined gene expression associated with memory decline during aging. This body of work characterizes several biological markers of age-related memory impairment and provides a model linking age-related memory decline and a major hypothesis for aging, altered Ca^{2+} homeostasis, through a change in Ca^{2+} signaling cascades to markers of brain aging including the shift in synaptic plasticity, increased susceptibility to neural toxicity, and altered gene regulation. Our current research directed at testing the effectiveness of treatments in ameliorating memory decline and preventing/reversing markers of brain aging includes behavioral treatments (exercise, environmental enrichment), diet (vitamin E, high fat, caloric restriction) and lentiviral gene delivery (superoxide dismutase, estrogen receptor, growth factors).

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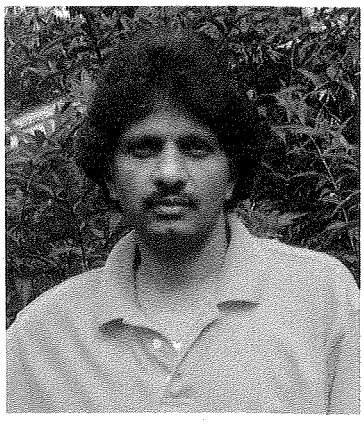

Dr. Kumar studies the role of Ca^{2+} dysregulation in altered synaptic function and its implication in age-related memory loss. Dysregulation of the Ca^{2+} homeostasis during aging contributes to various biological markers of brain aging including shift in synaptic plasticity and decreased neuronal excitability due to an augmented afterhyperpolarization (AHP) and spike frequency accommodation. Aging is associated with a shift in synaptic plasticity favoring long-term depression (LTD) over long-term potentiation (LTP) and Dr. Kumar has shown that the magnitude of the Ca^{2+} -dependent, K^{+} mediated AHP plays a critical role in setting the threshold for induction of synaptic plasticity. Recently, Dr. Kumar has shown that Ca^{2+} release from intracellular Ca^{2+} stores and voltage gated Ca^{2+} channels contributes to the enhanced AHP and thus the threshold for LTP induction.

Dr. Kumar's research also involves delineating the impact of environmental enrichment and exercise on biological markers of brain aging and its effect on cognitive performance during senescence. Recently published results suggest that environmental enrichment reduced the augmented AHP in aged animals. In addition, Dr. Kumar's research also focused on unraveling the pharmacological and biochemical signaling pathways involved in chemically induced LTD by activation of group I metabotropic glutamate receptor and cholinergic receptor agonists. Findings published last year indicate that the magnitude of mGluR-induced LTD is enhanced in senescent animals and depends on activation of both mGluR 1 and 5 subtypes and requires Ca^{2+} from L-type Ca^{2+} channels.

Finally, Dr. Kumar also studies effects of estrogen on hippocampal function across the lifespan and his results indicate that estrogen rapidly increases neuronal excitability, decreases AHP, and augments the strength of synaptic transmission. Thus, taken together Dr. Kumar's research interest is to delineate the pharmacological, biochemical, and molecular mechanisms underlying biological markers of brain senescence, which underlie cognitive impairments associated with aging. Dr. Kumar earned his Master of Science from University of Lucknow and his Doctor of Philosophy in Pharmacology from Central Drug Research Institute, and is currently employed as a Research Assistant Professor and working in the laboratory of Dr. Thomas C. Foster at the University of Florida.

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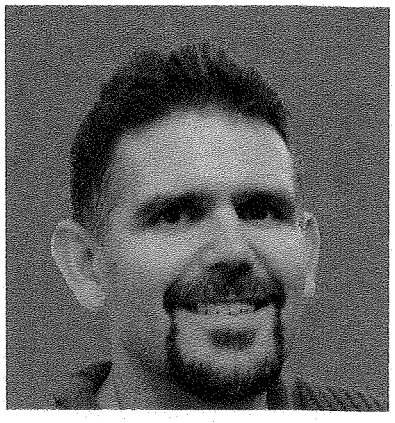

Dr. Laywell's laboratory has three major areas of emphasis: 1) neural stem cell biology, with particular interest in the role of multipotent astrocytes in neurogenesis; 2) the use of thymidine analogs as negative regulators of cancer cell proliferation; and 3) the role of microglia in modulating neurogenesis and neurodegenerative diseases.
Dr. Laywell received a bachelor's degree from the University of Michigan, and a Ph.D. in Neuroscience from the University of Tennessee. He did postdoctoral training at the University of Utah and University of Tennessee before moving to the University of Florida where he is currently an assistant professor in the Department of Anatomy & Cell Biology.

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Research in the Luesch lab lies at the interface of chemistry and biology and addresses multiple aspects of the drug discovery process ranging from assay development, identification and structure determination of bioactive small molecules, to studies toward the mechanism of action of small-molecule drug candidates and the discovery of novel putative drug targets. In our quest for small molecules with biomedical utility we mainly scrutinize natural products derived from marine cyanobacteria and eukaryotic algae. Active compounds are isolated using bioassay-guided fractionation and their structures determined using a combination of spectroscopic techniques. Subsequently, we use various genomic, proteomic and metabolomic profiling techniques to elucidate the mechanism of action of these compounds. We are also carrying out studies to disclose the function and action of genes/proteins that are putatively involved in cancer, aging, and neurodegeneration. Candidate genes are identified by high-throughput genome-wide screening. Validated genes are then subjected to molecular and biological characterization. The ultimate goal is to modulate gene or protein function with small molecules which could then be translated into valuable chemical biology tools or even novel drugs. Our recent focus has been on gene products that modulate oxidative stress levels through activation of the antioxidant response element (ARE). In humans, the ARE regulates the expression of a number of cytoprotective antioxidant enzymes and scavengers which contribute to the endogenous defense against oxidative stress. The activation of the ARE in the absence of general oxidative stress could provide a novel therapeutic approach for the treatment of various neurodegenerative diseases, stroke and aging. Dr. Luesch received his *Diplom* in Chemistry at the University of Siegen (Germany) in 1997. He attended the University of Hawaii at Manoa to study marine natural products chemistry and obtained his Ph.D. in Chemistry under the supervision of Prof. Richard E. Moore in 2002. He then undertook three years of postdoctoral studies as an Irving S. Sigal Fellow at The Scripps Research Institute in La Jolla under the guidance of Prof. Peter G. Schultz in the area of functional genomics. In 2005 he joined the faculty of the Department of Medicinal Chemistry at the University of Florida.

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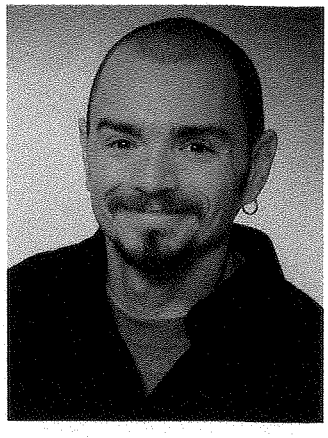

The central goal of Dr. Marshall's research is elucidate the connection between microglia (both intrinsic and bone marrow derived) and adult neurogenesis. His research involves the isolation and expansion of microglia from neurogenic regions of the brain and the subsequent transplantation of these cells back into the brains of aged mice. As a hematopoietic corollary to this work, bone marrow chimeras are generated using transgenic bone marrow from younger animals in order to ascertain the link between bone marrow derived microglia and age-related declines in neurogenesis. This work holds the potential to provide insights into determining the cellular cause for the well documented age-related declines in neurogenesis, as well as potential therapies for both cognitive decline and neurodegenerative disease states. Dr. Marshall is a postdoctoral fellow currently working under a NIH research training grant with the Center for the Neurobiology of Disease in the College of Pharmacy at the University of Florida.

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Received Ph.D. in Physiology at the Institute of Developmental Biology in 1989. Joined the UF faculty in 1998

Research interests within the MBI-UF Programmatic Matrix: Genomic Bases of Neuronal Identity and Plasticity; Learning & Memory Mechanisms, Age-related Memory Loss; Epigenetic Control of Neuronal Phenotypes in Networks, Developmental NeuroBiology, NeuroGenomics.

Searchable neuro-related research terms and diseases: Genomics, proteomics, single-cell genomic and microchemical analysis, evolution of nervous systems, cell-cell communication, synaptic physiology, nanotechnology, neural circuit reconstruction in vitro.

Research Program

My laboratory works to characterize basic mechanisms underlying the design of nervous systems and evolution of neural circuits and signaling mechanisms. The major questions are: (1) why are individual neurons so different from each other, (2) how do they maintain such precise connections between each other, (3) how does this fixed wiring result in such enormous neuronal plasticity and (4) how does this contribute to learning and memory mechanisms? By taking advantage of relatively simpler nervous systems of invertebrate animals as models (e.g. the mollusk *Aplysia*, *Cancer*), we combine neuroscience, genomics, bioinformatics, evolutionary theory, zoology, molecular biology, micro analytical chemistry and nanoscience to understand how neurons operate, remember and learn.

Current Projects

1. Genomic Bases and Principles of Organization of Neural Circuits Controlling both Stereotyped (Locomotion & Feeding central pattern generators) and Learned Behaviors (a simpler memory-forming network involved in a defensive behavior of *Aplysia*); Mechanisms of Generation of Neuronal Diversity in these Neural Circuits
2. Genomic Bases of Neuronal Homeostasis and Mechanisms of Maintenance of Unique Neuronal Phenotypes in Networks; Reconstruction of simpler neural networks in cell culture
3. Genomic bases of long-term plasticity: Genomic dissection of a simpler memory forming network
4. Massive Parallel Sequencing Technologies for Single-neuron Genomics: Digital Gene Expression Profiling and Integrated Single-Cell Microchemical Analysis using capillary electrophoresis and MS.
5. Gaseous Signaling in Nervous System: The role of NO in memory-forming and feeding circuits.
6. Parallel Evolution of Nervous Systems: Polyphyletic Origin of Neurons and Neural Circuits

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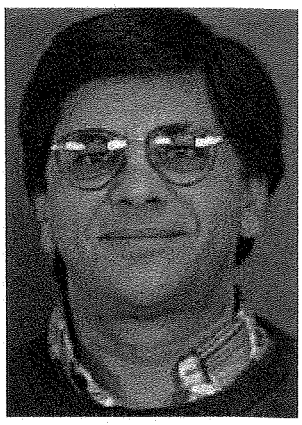

Nick Muzyczka was trained as a biochemist in DNA replication. As a postdoctoral fellow in Dan Nathans's laboratory, he developed the first SV40 vectors. When he moved to Florida, he then developed the first AAV vectors. For most of his career he has studied the basic molecular biology and biochemistry of AAV and worked out many of the technical issues related to the use of AAV for gene therapy. One of the technical problems that was solved was the modification of the coding sequence of the green fluorescent protein (GFP) gene so that it could be used in higher eukaryotes. This is now widely used by many laboratories. Muzyczka's lab also developed most of the production, purification and quantitation protocols currently in use today. In addition, Muzyczka's lab mapped the transcriptional signals of AAV, created the first genetic map of AAV, identified the components of the AAV origin for DNA replication, characterized the biochemical activities of the AAV non-structural Rep proteins, completely reconstructed AAV DNA replication in vitro with purified cellular components, and most recently, helped to crystallize and characterize mutants in the AAV capsid proteins. He is currently working on AAV capsid assembly and trafficking and trying to develop vectors that are specifically targeted to particular tissues or organs. Muzyczka has also collaborated with a number of investigators to bring rAAV vectors to clinical trials including the current trial for alpha-1-antitrypsin deficiency (a genetic pulmonary disorder) and the current trial for rpe65 deficiency (a form of congenital blindness). As part of a collaboration with Ron Mandel at the University of Florida to bring therapies for Parkinson Disease to the clinic, Muzyczka's lab has begun studying the function of alpha-synuclein, the major protein in Lewy bodies. His lab recently published work showing that the non-phosphorylated form of alpha-synuclein is non-toxic suggesting that Lewy bodies protect against Parkinson degeneration rather than promote it. In the learning and memory area, Muzyczka's lab first developed rAAV vectors that could quantitatively transduce the hippocampus. He then used a learning paradigm and microarrays to identify a number of genes in the hippocampal CA1 and dentate region that were likely to be involved in learning and memory. Most recently, he has tested three of the CA1 genes in the rat hippocampus by using rAAV vector gene transfer to overexpress these genes. All three genes (cycD1, pctk1, and tcf12) produced significant learning deficits in the radial arm water maze paradigm. Finally, Muzyczka's lab is also collaborating with Dave Morgan at USF to develop therapies for Alzheimer's Disease.

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My laboratory investigates how protein homeostatic mechanisms, specifically the chaperone and autophagy-lysosomal pathways, are involved in the subcellular pathogenesis of neurodegenerative paradigms and in the age-related decline of neural function. While we examine the contribution of these basic mechanisms to disease processes, we are also exploring dietary and pharmacologic approaches to modulate these pathways. The main model system we use for our studies is Charcot-Marie-Tooth disease type 1A, an autosomal dominant demyelinating disorder of the peripheral nervous system. Using this paradigm, we are examining how cells respond to the accumulation of damaged and misfolded proteins and investigating approaches to prevent and reverse these events. Currently we are expanding these studies to rodent models of aging. Along with our investigations concerning age-, toxinand genetically-linked alterations in the nervous system, we are interested in understanding mechanisms that mediate the differentiation and interactions of neurons and glia. A recently discovered mechanism for differential gene expression in related cells is the utilization of microRNAs. Ongoing experiments are aimed at determining which specific miRNAs regulate the expression of key glial proteins at specific stages of neural development.

Dr. Notterpek received a B.A. in Anatomy-Physiology from the University of California at Berkeley. She obtained her Ph.D. in Neuroscience at the University of California at Los Angeles working with Dr. Leonard H. Rome. Her postdoctoral training was under the guidance of Dr. Eric Shooter at Stanford University. Currently, Dr. Notterpek is an Associate Professor in the Department of Neuroscience at the McKnight Brain Institute of the University of Florida, with tenure. She is recipient of the 2004 Jordi Folch-Pi Memorial Award, from the American Society of Neurochemistry, to a young scientist for research excellence. She has authored and coauthored nearly forty peer-reviewed publications. She is actively involved in the educational and research missions of the College of Medicine at the University of Florida. Her research efforts are being supported by the NIH, the National Muscular Dystrophy Association and the National Multiple Sclerosis Society.

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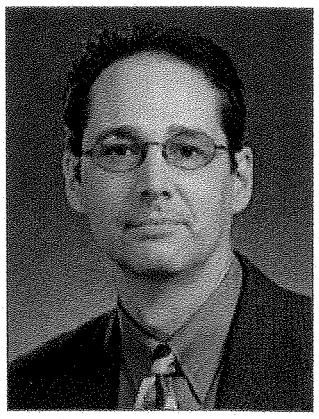

Dr. Ogle's area of research is how memory functions. What genes are activated by the process of memory? How is this process disrupted by stress and aging? We have originally approached the problem by comparative analysis of gene expression during memory formation in two regions of the brain during chronic-stress. This has allowed us to develop an initial list of candidate genes and pathways that are involved in memory formation.

While the study of memory is very important work, what we consider more important is the study of how memory fails. Stress and ageing can lead to a decline in the ability of an individual to store memories. We would like to understand the events that lead to memory decline and reverse them. We are also trying to understand age related memory decline at the level of gene expression? We have been able to examine the differences in genes expressed in the brains of laboratory rats between old animals with good memory recall and poor memory recall. We believe we see positive compensatory changes in gene expression in the animals with good memory recall. This observation is remarkable in that we see age related compensation and that in some animals this compensation maintains healthy memory with increased age.

We also know that stress effects memory and we know that stress effects neurogenesis. Some evidence suggests neurogenesis may play a role in memory formation. So, can we regulate or modulate neurogenesis and with this modulation restore memory? We are approaching this problem by applying a pharmacological approach and a genetic approach. We are directly modulating the effects of stress hormones on neuronal precursors by using engineered proteins that can modulate the cellular stress response. We are introducing these proteins into neuronal precursors and then determining what effects these engineered proteins have on the process of neurogenesis.

Dr. Ogle received his Bachelor of Science in Biological Sciences (Invertebrate Zoology) and Chemistry (Biochemistry) from California State University, Hayward a Master in Science (Biochemistry) and a Doctor of Philosophy (Virology) from the Department Biochemistry and Molecular Biology at the University of Chicago. Before becoming a faculty in the College of Engineering he was a Post-Doctoral Fellow at Stanford University (Neurobiology) and is an Ellison Medical Foundation New Scholar in Aging.

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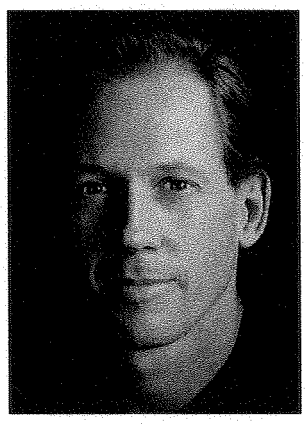

Brent Reynolds is an Associate Professor at the McKnight Brain Institute in the Department of Neurosurgery at the University of Florida. He received his Ph.D. in 1994 from the University of Calgary during which time he and Sam Weiss discovered the existence of a stem cell in the adult central nervous system, challenging a century old dogma that the adult brain was unable to produce new neurons. He co-founded NeuroSpheres Ltd. where he was Vice-President of Research and in 1999 published the first report on the transdifferentiation of cells derived from one germ layer into functional cells of another germ layer. Dr. Reynolds holds 16 US patents related to neural stem cells and his lab is currently focused on the *in situ* manipulation of neural stem cells and understanding the role that solid tissue cancer stem cells play in tumor initiation and metastasis.

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Dr. Ross studies the role of bromodeoxyuridine (BrdU) in the age-related induction of senescence in neural stem and progenitor cells. Her work has shown that a brief exposure of BrdU leads to a senescent state that involves the upregulation of proteins associated with prominent p53- and Rb-dependent senescence pathways. Current studies are designed to expand these findings and gain a better understanding of BrdU-mediated senescence signaling pathways. She is further interested in determining what effects BrdU has at the DNA level that lead to the activation of senescence signaling events and the elucidation of the parallels this shares with genomic instability related to aging. The goal of this work is to study the molecular mechanisms governing physiological aging and recurrent neurogenesis through the utilization of BrdU as a rapid and reproducible *in vitro* model of aging. Dr. Ross is also interested in the amelioration of recurrent glioblastoma multiforme (GBM) using BrdU. This work aims to study the efficacy of BrdU treatment to target the cancer stem cell and/or highly invasive residual tumor cells. Dr. Ross earned her Master of Physical Therapy from East Carolina University in 2000 and her Doctor of Philosophy in Anatomy and Neurobiology from Virginia Commonwealth University in 2006, and is currently employed as a Postdoctoral Associate in the laboratory of Dr. Eric Laywell at the University of Florida.

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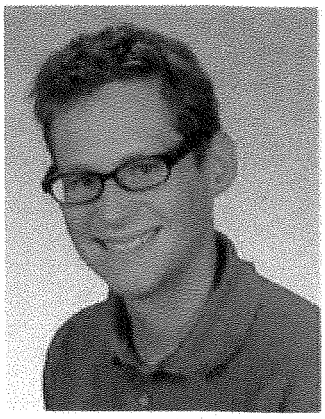

The main focus of Dr. Siebzehnrubls research are neurodegenerative disorders of the striatum, i.e. Parkinson's and Huntingtons disease. A main goal is the development of an experimental, adult stem cell-based therapy for Parkinson's disease. Employing genetically modified adult human neuroprogenitors that release GDNF, this therapy is aimed at preserving and restoring dopaminergic innervation of the striatum. This program involves stem cell grafting in rodent models of Parkinson's disease, as well as behavioral testing and basic neuroscience research techniques.

Another area of interest targets adult subventricular zone stem cells in Huntington's disease. Functional anomalies of stem cell migration and differentiation are analyzed in a rat model of Huntington's disease. Using organotypic slice cultures as a platform for time-lapse imaging as well as fate mapping, this work aims to provide a basis for the search of stem cell-targeting therapeutic compounds.

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Dr. Zheng's major research interest is the characterization of postnatal and adult sources of transplantable cells that may eventually be appropriate as therapeutics for neurological injury and disease. Our previous studies have demonstrated that certain regionally and temporally restricted astrocytes display neural stem cell characteristics *in vitro*. These Multipotent Astrocytic Stem Cells (MASCs) from both subependymal zone (SVZ) and non-SVZ regions have the abilities to respond to intrinsic environmental cues by anatomically integrating into the plastic SEZ-RMS-olfactory bulb system. Dr. Zheng is exploiting the possibility of MASCs as a therapeutic source for neurological disorders by grafting these cells into animal models of neonatal or adult disease. She is also interested in approaches that would enhance the survival, differentiation and migration of the grafted cells-including homing factors, biomaterial scaffolding, and growth factors. Her current research also involves the use of live cell tracking and novel imaging approaches to track stem cell's growth, migration and differentiation both in vitro and in vivo.

Evelyn F. McKnight Center for Age-Related Memory Loss Meeting Participants
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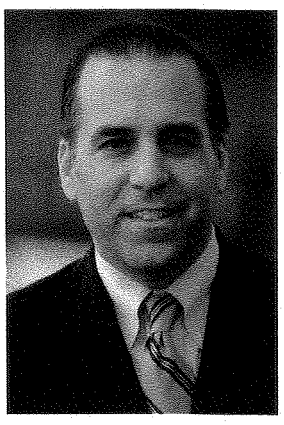

Ralph L. Sacco, MD, MS, is the Chairman of Neurology and Miller Professor of Neurology, Epidemiology, and Human Genetics at the Miller School of Medicine, University of Miami and Chief of the Neurology Service at Jackson Memorial Hospital. He was the former Professor of Neurology and Director of the Stroke and Critical Care Division at the Neurological Institute of Columbia University College of Physicians and Surgeons, the Mailman School of Public Health, and the Sergievsky Center.

Dr. Sacco graduated from Cornell University with distinction, received his medical degree cum laude from Boston University School of Medicine in Massachusetts, and a master's degree in epidemiology from Columbia University, School of Public Health. Dr. Sacco completed a residency in neurology at Presbyterian Hospital of the City of New York. He completed his postdoctoral training in stroke and Epidemiology at Columbia under a NINDS-funded neuroepidemiology training grant.

Dr. Sacco's clinical research activities began in 1980 when he participated in the Framingham Heart Study. Since 1990, he has been the Principal Investigator of the Northern Manhattan Study an NIH-funded community-based, epidemiologic study designed to determine stroke incidence, risk factors, and prognosis in an elderly, multi-ethnic, urban population living in northern Manhattan in New York City. This study now includes a separate NINDS-funded project, the Northern Manhattan Family Study, to evaluate potential genetic determinants of stroke risk factors.

Dr. Sacco was also the founding principal investigator of the NY Columbia Collaborative Specialized Program in Translational Research in Acute Stroke. He is also co-investigator of six other NINDS grants. He has been involved in the design and conduct of multiple randomized trials including the co-principal investigator of the Warfarin Aspirin Recurrent Stroke Study, the principal investigator of the Glycine Antagonist in Neuroprotection Trial, and the current co-chair of the international PRoFESS Study (Prevention Regimen For Effectively avoiding Second Strokes). He serves on the Data Safety and Monitoring Boards of a number of NIH and pharmaceutical-sponsored clinical trials. In addition, Dr. Sacco is on the editorial board of *Stroke, Neuroepidemiology*, and *Nature Clinical Practice Neurology*. He has published extensively in the areas of stroke prevention, treatment, risk factors and stroke recurrence, with more than 475 original

articles, case reports, book chapters, abstracts and communications to his credit. He has been a principal author on numerous evidence based guidelines from the AHA and ACCP. He has helped train numerous fellows in stroke and epidemiology. He has been awarded the 2006 Feinberg Award for Excellence in Clinical Stroke and the 2007 Chairman's Award from the American Heart Association.

Dr. Sacco is a fellow of the Stroke and Epidemiology Councils of the American Heart Association, a Fellow of the American Academy of Neurology, a member of the American Neurological Association, past chair of the Clinical Research Committee of the American Academy of Neurology, and on the Medical Advisory Board of the Hazel K. Goddess Fund for Stroke Research in Women. He is a past member of the Epidemiology and Disease Control-3 NIH Study Section, NINDS Neuroscience Training Review Committee, and FDA Advisory Panel for Central and Peripheral Nervous System Drugs. He is on the Board of Directors for the American Heart Association and American Academy of Neurology and past president of the New York City AHA Board. He is a member of the Stroke Prevention Advisory Panel of the National Stroke Association and chair of the Stroke Advisory Committee of the American Stroke Association.

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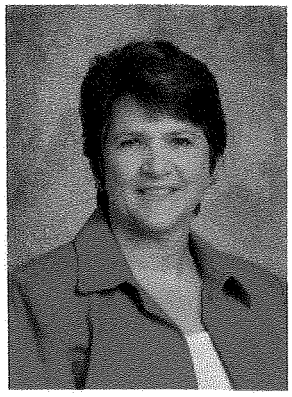

Dr. Blanton's primary research has focused on the mapping of Mendelian and complex diseases. Deafness, retinal diseases, skeletal dysplasias, cleft lip/palate, and club foot are among the diseases which she currently studies. She has recently begun collaborating on studies of stroke and genetic risk factors. She has also been involved in developing and implementing genetic education materials for Federal and appellate level judges and science writers in an ELSI sponsored project. Her current research also involves determining the level of genetic knowledge and attitudes towards genetic testing among the deaf. Dr. Blanton is Associate Director of Communications and Compliance at the MIHG, Associate Professor of Medicine and the Project coordinator for the GGMI.

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Dr. Gardener is an epidemiologist whose research focuses on environmental and genetic risk factors for chronic diseases, with a particular interest in neurological diseases. She is currently examining predictors of carotid atherosclerosis, stroke, and other vascular events using data from the Northern Manhattan Study, and ongoing prospective cohort study. Dr. Gardener graduated from the Harvard School of Public Heath with a doctorate of science in epidemiology in November, 2007. At Harvard her research focused on prenatal and early life risk factors for Parkinson's disease, multiple sclerosis, and autism. She is currently a Post-Doctoral Associate at the University of Miami Miller School of Medicine in the Department of Neurology.

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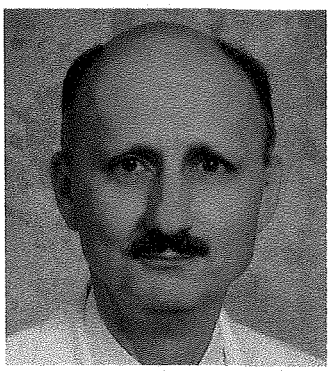

Dr. Horwath is Professor of Psychiatry, Epidemiology and Public Health at the University of Miami Miller School of Medicine; Executive Vice Chair of the Department of Psychiatry and Behavioral Sciences; Executive Medical Director of UMBH; Fellow of the American Psychiatric Association; Chief of Psychiatry Service at University of Miami Hospital; Associate Chief of Psychiatry at Jackson Memorial Hospital.

A native of Austria, Dr Horwath received his medical degree from the University of Chicago Pritzker School of Medicine and completed his residency in psychiatry at Columbia University and the New York State Psychiatric Institute. He was a fellow in psychiatric epidemiology and received his MS in epidemiology from the Columbia University School of Public Health.

Dr. Horwath is a highly experienced clinical expert in the treatment of patients with schizophrenia and other psychotic disorders, bipolar disorder, depression, anxiety and AIDS. He is the former Director of the Acute Inpatient Unit that is the primary clinical and teaching service for the Columbia University Department of Psychiatry.

Dr. Horwath is the former Research Director of the Washington Heights Community Service at the New York State Psychiatric Institute and Columbia University Department of Psychiatry. He has published widely on the epidemiology of depression and anxiety disorders, the prevalence of HIV infection among those with severe mental illness and the neuropsychiatric manifestations of AIDS. His articles have appeared in *Archives of General Psychiatry*, *American Journal of Psychiatry* and other prestigious scientific journals.

As former medical director of the Columbia University HIV Mental Health Training Project, he has lectured widely to medical and mental health audiences concerning the psychiatric and neurologic evaluation and treatment of persons with HIV/AIDS and was the chief psychiatric consultant to the Woodycrest Center. A health care facility for patients with AIDS.

Richard S. Isaacson, M.D.
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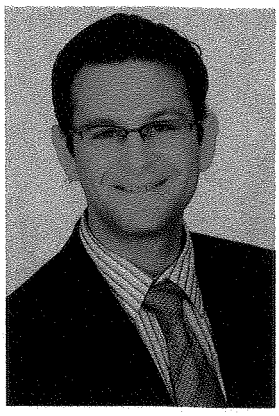

A graduate of the accelerated 6-year B.A./M.D. program at the University of Missouri at Kansas City School of Medicine, Dr. Isaacson currently serves as Director of the Neurology Residency Program at the University of Miami Miller School of Medicine. He completed a three-year residency in Neurology at Beth Israel Deaconess Medical Center/Harvard Medical School in Boston, MA, and completed his medical internship at Mount Sinai Medical Center/University of Miami. Prior to leaving Boston to re-join his family in South Florida, he was appointed as clinical fellow in neurology and instructed neuroanatomy laboratory at Harvard Medical School.

Dr. Isaacson has conducted laboratory and clinical research at the Laboratory of Central Nervous System Studies, part of the National Institute of Neurological Disorders and Stroke, National Institutes of Health in Bethesda, MD with Nobel laureate Dr. D. Carlton Gajdusek and the late Dr. Clarence J. Gibbs, Jr. Dr. Isaacson is the author of numerous abstracts and publications, and his research in neurology and medical education has been presented at scientific meetings both nationally and internationally. His clinical interests include cognitive impairment, cognitive aging and Alzheimer's disease, and he has been involved in a number of clinical and epidemiologic studies on the diagnosis and treatment of disorders. Dr. Isaacson leads the American Academy of Neurology Undergraduate Education Subcommittee working group in cognitive disorders, which is responsible for making recommendations of what is taught to medical students around the country. Dr. Isaacson was recently awarded the American Academy of Neurology 2008 Education Research Award for his project "Evaluating the effectiveness of *Continuum*: Dementia as a teaching tool for medical students".

Bonnie E. Levin, Ph.D.
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Dr. Levin is a neuropsychologist whose research examines neurocognitive and affective changes associated with neurodegenerative disease and the normative again process. Her work examines the inter-relationship between behavioral and motor symptoms in Parkinson's disease and the neural circuitry underlying memory and age related cognitive decline. Her current work is aimed to advance our understanding of frontal striatal circuit function in cognition and to generate data that will improve our knowledge of key clinical parameters associated with differential rates of cognitive decline. Current projects include: imaging and clinical correlates of white matter changes associated with the aging process and structural and metabolic markers underlying different symptom profiles in neurodegenerative disease. Dr. Levin is an Associate Professor of Neurology and Psychology and is the Director of the Division of Neuropsychology within the Department of Neurology at the University of Miami Miller School of Medicine.

Tatjana Rundek, MD PhD
Associate Professor of Neurology
Director, Clinical Translational Research Division

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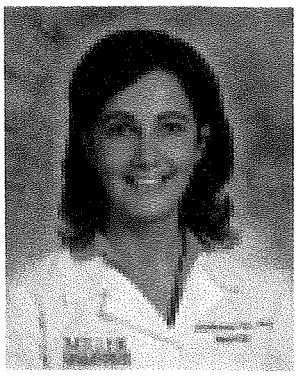

Dr. Tatjana Rundek, MD PhD is an Associate Professor of Neurology at the Department of Neurology of the University of Miami and an Adjunct Faculty at Columbia University in the City of New York, NY. Dr. Rundek is a director of the Clinical Translational Research Division in Neurology at the University of Miami. She was and Assistant Professor of Neurology at the Department of Neurology of the Columbia University, NY and a Director of Research Neurosonology Laboratory at Columbia University, NY. Dr. Rundek graduated from University of Zagreb, Croatia, where she received her medical degree. She received a PhD degree in neuroscience at Max-Planck Institute in Munich, Germany, and trained in neurology at the Grossharden Spital in Munich and at the University Hospital Sisters of Mercy in Zagreb. She trained in neurosonology at the University of Ulm. In the USA, she received her research fellowship training in stroke and neuroepidemiology at the Columbia University, NY. Dr. Rundek research is directed towards the use of ultrasound in early detection, intervention and prevention of functional and structural changes of arterial wall inflammation. Dr Rundek in a clinical researcher and an investigator on several large longitudinal NIH funded grants. She is a PI of the Genetic determinants of carotid atherosclerosis project; co-investigator of the Northern Manhattan Stroke Study and Family Study; and a site PI of the Oral infection and risk of CVD project, the CVD manifestation of primary hyperparathyroidism grant, and Aortic arch and risk of stroke study. Dr. Rundek published extensively on stroke and CVD risk using non-invasive vascular ultrasound in various vascular risk populations. Dr. Rundek was the Fulbright Scholar and the recipient of the research awards from the Hazel K. Goddess Found to study the relationship between vascular risk factors, subclinical atherosclerosis by ultrasound and stroke in women and the Dr. Gilbert Baum Award (AIUM) for expanding clinical applications of vascular ultrasound. Dr Rundek serves on the editorial boards of several professional journals including Stroke, Journal of Cardio Metabolic Syndrome, and Therapy. She is a member of the American Heart Association, American Academy of Neurology, and American Institute of Ultrasound in Medicine.

Clinton B. Wright, M.D.
Current Position:
Assistant Professor of Neurology, Columbia University
Effective July 2008:
Associate Professor of Neurology
Scientific Director, McKnight Center

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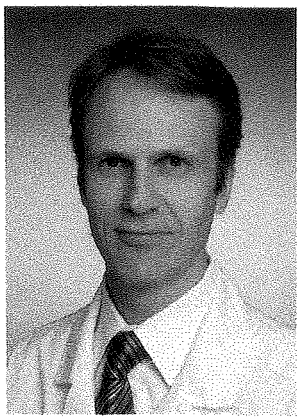

Dr. Wright will be moving to the Miller School of Medicine at the University of Miami in July 2008 from the College of Physicians and Surgeons of Columbia University. He will be appointed to Associate Professor of Neurology and Scientific Director for the McKnight Center. He received his undergraduate degree from George Washington with a degree in psychology and his M.D. degree from Columbia University, where he also completed residency training in neurology and a vascular neurology fellowship and master's in epidemiology. Dr. Wright's work is supported by the American Heart Association and the National Institutes of Neurological Disorders and Stroke and is focused on race-ethnic disparities and the effects of vascular risk factors on brain structure and function, with an emphasis on early cognitive changes. He is chair of the neuroimaging and cognitive components of the Northern Manhattan Study. Recent studies include the determinants of subclinical vascular brain damage as measured by magnetic resonance imaging, and the effects of white matter hyperintensity volume as well as subclinical infarction on psychomotor speed and cognitive flexibility.