

SYMPOSIUM PROGRAM



THE STRESSED BRAIN IN THE 21ST CENTURY

RESEARCH ADVANCES **AND** CLINICAL DIMENSIONS

25th Annual International Symposium

<http://stress.hunter.cuny.edu>

Friday, March 16, 2012, 8:30 AM - 6:00 PM

The Kaye Playhouse at Hunter College | East 68th Street at Lexington Ave, NY | NY 10065

25th Annual International Symposium

Sponsors:

Hunter College of the City University of New York,
Center for Study of Gene Structure and Function

Weill Cornell Medical College,
Clinical and Translational Science Center

Additional CTSC member institutions:

Memorial Sloan-Kettering Cancer Center, Hospital for Special Surgery,
Hunter College School of Nursing, Cornell University Cooperative Extension

The 25th Annual International Symposium of the Center for Study of Gene Structure & Function at Hunter College, with Weill Cornell Medical College Clinical and Translational Science Center, is supported by the National Institute on Minority Health and Health Disparities, National Institutes of Health - G12-RR-003037 and Clinical Translational Science Award - UL1RR024996



The mission of the National Institute on Minority Health and Health Disparities (NIMHD) is to promote minority health and to lead, coordinate, support, and assess the NIH effort to reduce and ultimately eliminate health disparities. In this effort NIMHD will conduct and support basic, clinical, social, and behavioral research, promote research infrastructure and training, foster emerging programs, disseminate information, and reach out to minority and other health disparity communities. <http://www.nimhd.nih.gov/default.html>



The NIH, a part of the U.S. Department of Health and Human Services, is the primary federal agency for conducting and supporting medical research. Composed of 27 Institutes and Centers, the NIH provides leadership and financial support to researchers in every state and throughout the world. Its mission is science in pursuit of fundamental knowledge about the nature and behavior of living systems and the application of that knowledge to extend healthy life and reduce the burdens of illness and disability. <http://www.nih.gov>



RTRN is strategically positioned to facilitate interdisciplinary clinical and translational research. RTRN has established a solid technological foundation to support intellectual exchange, generate innovative inter- and multi-disciplinary research and facilitate the movement of scientific advances throughout the translational research spectrum.



Center for Study of Gene Structure and Function

The Center for Study of Gene Structure and Function (Gene Center) is a consortium of researchers within Hunter College of The City University of New York - one of the largest public universities in the nation. At the heart of the Gene Center's mission is an imperative to build unique collaborations among biologists, biochemists, bio-psychologists, bio-physicists, bio-anthropologists and researchers from the Hunter School of Social Work and Urban Public Health; to recruit and equip outstanding faculty; to facilitate translational research; to develop and share core research facilities; and to implement strategies for scientific networking.

Since the Gene Center's inception, the growing number of papers published in peer-reviewed journals and the number and amount of grants obtained by the faculty have been the most visible hallmarks of its success. The Gene Center provides a vibrant research environment marked by: workshops on cutting-edge research techniques; frequent research colloquia and seminars by guest scientists; an annual international symposium, which is a major event on the New York scientific calendar; and a strong emphasis on collaborative translational research.

In addition, the Gene Center encourages bright undergraduates, including minorities, to make a career in biomedical, drug abuse/neuroscience research by hosting a Summer Program for Undergraduate Research (SPUR) and supports the professional development of minority scientists through the JustGarciaHill science web site.

The Gene Center is a key partner in the Clinical and Translational Science Center (CTSC), an enterprise that also includes the Weill Cornell Medical College, Memorial Sloan-Kettering Cancer Center, Hospital for Special Surgery and the Hunter College School of Nursing. The CTSC was established in 2007 with the aim of accelerating translational research. The overall goal is to facilitate the transition of laboratory work into state of the art clinical research (T1 research), and to provide research (T2) that improves patient care and health outcomes in the general community (T3). The Gene Center encourages collaborations among its members and with scientists at these and other institutions. The Gene Center also participates in a national consortium, the RCMI Translational Research Network (RTRN), that facilitates collaboration, large-scale projects and sharing of facilities among RCMI institutions.

Visit the Gene Center website: <http://genecenter.hunter.cuny.edu>



Clinical and Translational Science Center

The Clinical and Translational Science Center (CTSC) is a unique collaboration between renowned biomedical and community organizations centered on Manhattan's east side. Weill Cornell Medical College and Graduate School of Medical Sciences is home to the administrative core of the CTSC, led by CTSC Program Director Julianne Imperato-McGinley, MD, Associate Dean of Translational Research and Education at Weill Cornell Medical College (WCMC).

In addition to WCMC, the CTSC partner institutions include:

- Hunter College, Center for Study of Gene Structure and Function
- Hunter College, School of Nursing
- Hospital for Special Surgery
- Memorial Sloan-Kettering Cancer Center
- Cornell University Co-operative Extension in New York City

Affiliated hospitals include New York-Presbyterian Hospital, Lincoln Medical Center, Methodist Hospital, New York Downtown Hospital, New York Queens Hospital, Wyckoff Heights Medical Center, and Brooklyn Hospital.

The CTSC is designed to bring together the resources of all partner and affiliate institutions to facilitate novel translational research. Separately, these institutions include superb academic centers of excellence, a diverse patient base, and a unique community-engagement program designed to foster collaboration between community groups and translational research scientists. Each partner and affiliate has an unmistakable character that enhances multi-disciplinary interaction. Integration of these unique resources and intellectual assets will facilitate translation of research findings in the laboratory to clinical research at the bedside and ultimately to best practices within underserved communities.

A Translational Research Support Team and a wide range of services, including core laboratories and professionally staffed patient care inpatient and outpatient units, are available to support clinical and basic science investigators who are interested in translational research. Contact a CTSC Research Facilitator to find out more: jph2003@med.cornell.edu.

The CTSC is funded through the Clinical and Translational Science Awards, a national consortium that is transforming how clinical and translational research is conducted.

For more information about the CTSC, please visit <http://www.med.cornell.edu/ctsc>.

THE STRESSED BRAIN IN THE 21ST CENTURY: RESEARCH ADVANCES AND CLINICAL DIMENSIONS

*25th Annual International Symposium
March 16, 2012*

Overwhelming evidence implicates stress as the cause or precipitant of many health problems such as ulcers and high blood pressure. Less appreciated is the fact that stress exerts powerful effects over brain functions. For example, chronic stress is associated with increases in symptoms of depression, generalized anxiety disorder and post-traumatic stress disorder. This symposium brings together noted basic and clinical neuroscience researchers from the USA and abroad with the goal of advancing our understanding of maladaptive stress responses in order to improve treatments and outcomes. The impact of stress as it relates to race, ethnicity, gender, and socioeconomic status will also be a major focus. Moreover, basic and translational researchers will discuss mechanisms of risk and resilience related to stress-induced disease and disability during development and in adulthood. Both the morning and afternoon sessions will end with an extensive, moderated Question and Answer period. A poster session, open for submissions by the scientific community, will also provide ample opportunity for discussion. Thus, this symposium will provide an excellent opportunity for researchers, clinicians, and caregivers to advance their understanding of the pervasive and damaging physiological insults of continued stress, a condition which is becoming more pervasive in our society as we enter the 21st century.

James S. Jackson, The Daniel Katz Distinguished University Professor at the School of Public Health at University of Michigan, will open the symposium with a focus on understanding population differences in health in relation to stress.

Julio Licinio, Professor and Director of John Curtin School of Medical Research at Australian National University, will discuss depression and obesity as separate or comorbid entities and provide biological underpinnings through clinical studies and animal models.

Elissa Epel, Associate Professor in Psychiatry at UC San Francisco, will present psychological processes and biochemical mediators of stress arousal that are known to affect the telomere/telomerase maintenance system, which in turn impacts successful aging.

Margaret Altemus, Director of the Payne Whitney Women's Program in Psychiatry at Weill Cornell Medical School, focuses on sex differences in responses to stress and how reproductive hormones impact stress responses and vulnerability to psychiatric disorders.

Eric J. Nestler, Professor and Chair of Neuroscience and Director of Friedman Brain Institute at Mount Sinai Medical Center, utilizes an animal model for studying stress and depression that provides unique insights into the molecular mechanisms by which chronic stress produces lasting changes in the brain to cause depression-like symptoms.

BJ Casey, Director of Sackler Institute for Developmental Psychobiology at Weill Cornell Medical College, will present an overview of recent empirical studies employing human imaging, rodent models, and mouse genetics to examine how attention, fear, and stress-related processes differ across development and across individuals.

Roger Pitman, Professor of Psychiatry, Harvard Medical School and Massachusetts General Hospital, will present results of unique neurological and psychological studies of identical twins who are discordant for combat exposure in Vietnam, showing that identified biological risk factors only lead to psychopathology after the occurrence of a traumatic event.

Victoria Luine, Distinguished Professor of Psychology, Hunter College of CUNY, utilizes animal models to assess stress effects on cognitive processes and will highlight novel findings that sex and age may be critical in the etiology and treatment of stress-related diseases.

MORNING SESSION

- 9:00** Introduction and welcome:
Robert P. Dottin, Professor of Biological Sciences at Hunter College, CUNY and Principal Investigator of the Center for Study of Gene Structure and Function
Jennifer J. Raab, President, Hunter College, CUNY
Julianne Imperato-McGinley, Associate Dean of Translational Research, Weill Cornell Medical College
- 9:15** **James S. Jackson, Ph.D., Keynote Speaker**
University of Michigan
Environmental Affordances Framework of Health Disparities
- 10:00** **Julio Licinio, M.D., F.A.P.A.**
John Curtin School of Medicine Research at Australian National University
Translational Approaches to the Shared Biology of Stress, Depression, and Obesity
- 10:30** Coffee Break and Poster Session
- 11:00** **Elissa S. Epel, Ph.D.**
University of California, San Francisco
Stress and Resilience Factors for Cellular Aging
- 11:30** **Margaret Altemus, M.D.**
Weill Cornell Medical College
Sex Differences in Stress Responses and Psychiatric Disorders
- 12:00** **Panel discussion with Q&A led by Tracy Dennis, Ph.D.**

- 12:30** Lunch for **pre-registered** participants
Speednetworking session for registered clinicians and researchers
Poster session

AFTERNOON SESSION

- 2:00** **Robert P. Dottin**, Professor of Biological Sciences at Hunter College, CUNY and Principal Investigator of the Center for Study of Gene Structure and Function
- 2:05** **Remarks by Sponsoring Agency**
- 2:20** **Eric Nestler, M.D., Ph.D., Keynote Speaker**
Mount Sinai Medical Center
Transcriptional and Epigenetic Mechanisms of Depression
- 3:05** **BJ Casey, Ph.D.**
Weill Cornell Medical College
Basic and Translational Studies on the Developmental Psychobiology of Risk for Anxiety and Stress Related Disorders
- 3:35** Coffee Break and Poster Session
- 4:05** Poster Awards Ceremony
- 4:15** **Roger Pitman, M.D.**
Massachusetts General Hospital
Studies of Identical Twins Discordant for Combat Exposure in Vietnam
- 4:45** **Victoria Luine, Ph.D.**
Hunter College, City University of New York
Accounting for Sex and Age in Neural Responses To Stress
- 5:15** **Panel discussion with Q&A led by Peter Serrano, Ph.D.**
- 5:45** **Closing Remarks**
Presentations will be available via the symposium web site.

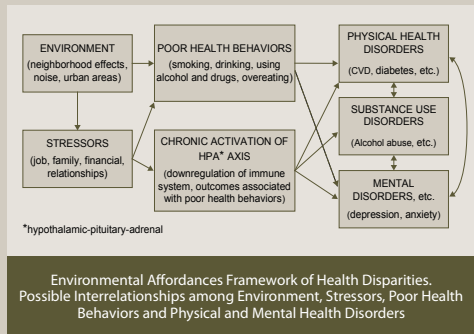
James S. Jackson, Ph.D.

University of Michigan

Environmental Affordances Framework of Health Disparities



Abstract: This lecture will present an overview of the *Environmental Affordances Framework of Health Disparities*; this is a comprehensive theoretical framework that addresses a compelling empirical paradox: non-Hispanic whites have better physical health and substance use disorders than black Americans, and in contrast, on most measures, black Americans have the same or lower rates of mental disorders than non-Hispanic whites. Succinctly, I propose that under stressful living conditions (e.g. poverty, poor housing, financial difficulties, etc.) individuals engage in accessible negative health behaviors (e.g. smoking, alcohol use and abuse, drug use, and overeating) in an attempt to cope with the stressors of daily life. The exposure to stress inducing events is more consciously accessible than the biological degenerations (e.g. growth in tumors, atherosclerosis, etc.) that eventuate in physical health ailments and chronic health conditions (e.g. heart disease, cancer). Thus, I hypothesize that when individuals are confronted with chronic stressful conditions of life they will engage in behaviors that alleviate the resulting symptoms of stress. These behaviors may interfere with or mask the physiological cascade of stressful responses through the hypothalamic-pituitary-adrenal cortical (HPA) axis that eventuate in serious mental disorders. However, these same behaviors silently affect physical health morbidity and mortality. The framework postulates that the environment serves as a source of stressors as well as a source of structures and opportunities that facilitate or afford the availability of negative health behaviors. In addition, the environment may have direct effects on the development of physical disorders and perhaps psychological health disorders as well.



Bio: James S. Jackson is the Daniel Katz Distinguished University Professor of Psychology, Professor of Health Behavior and Health Education, School of Public Health, and Director and Research Professor of the Institute for Social Research. He is the past Chair, Social Psychology Training Program and Director of the Research Center for Group Dynamics, the Program for Research on Black Americans, and the Center for Afroamerican and African Studies, all at the University of Michigan. He is past-Chair of the Section on Social, Economic, and Political Sciences (K) of the American Association for the Advancement of Science (AAAS). He is a former Chair of the Section on Social and Behavioral Sciences, and the Task Force on Minority Issues of the Gerontological Society of America, and the Committee on International Relations and the Association for the Advancement of Psychology of the American Psychological Association. He is a former National President of the Black Students Psychological Association and the Association of Black Psychologists. He is current President of the Society for the Psychological Study of Social Issues.

He served on the National Advisory Mental Health Council of the National Institute of Mental Health and the National Institute on Aging Advisory Council and the Board of Scientific Counselors of NIA. He was recently named to the NIH Advisory Council to the Director. He is a fellow of the Gerontological Society of America, Society of Experimental Social Psychology, American Psychological Association, Association of Psychological Sciences, International Demographic Association, and the American Association for the Advancement of Science. He is the recipient of the Distinguished Career Contributions to Research Award, Society for the Psychological Study of Ethnic Minority Issues, American Psychological Association, the James McKeen Cattell Fellow Award for Distinguished Career Contributions in Applied Psychology, the Association for Psychological Sciences, Presidential Citation, American Psychological Association, and the Medal for Distinguished Contributions in Biomedical Sciences, New York Academy of Medicine. He is an elected member of the Institute of Medicine of the National Academies of Sciences, and a Fellow of the American Academy of Arts and Sciences

<http://www.lsa.umich.edu/psych/people/directory/profiles/faculty/?uniqueusername=jamesjs>



Julio Licinio, M.D., F.A.P.A

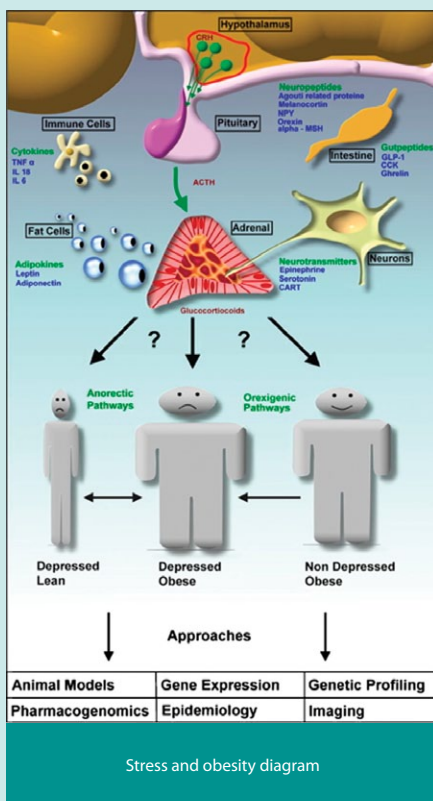
John Curtin School of Medical Research at Australian
National University

Translational Approaches to the Shared Biology of
Stress, Depression, and Obesity

Abstract: Depression and obesity are both major public health problems worldwide that often co-exist, yet they are most commonly studied as two distinct, unrelated entities. However, there is considerable clinical and biological overlap between these two conditions. Clinically, depressed mood can

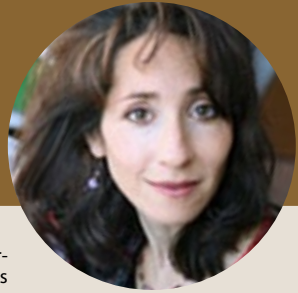
be a side effect of anti-obesity treatments and weight gain can be the outcome of antidepressant use. Furthermore, both disorders are independent risk factors for cardiovascular disease. Biologically, several neuroendocrine and neurotransmitter systems, involving molecules such as corticotropin-releasing hormone, neuropeptide Y, serotonin, norepinephrine and leptin are involved in the regulation of mood as well as body weight. Among shared biological mechanisms, chronic dysregulation of the stress response with hyperactivity of the hypothalamic-pituitary-adrenal axis can lead to both depression and obesity, either as separate entities or co-morbidly. This interface can be explored clinically and in animal models through a variety of approaches that include pharmacogenomics, genetic profiling, gene and protein expression studies, imaging and epidemiology. The translational outcomes of this line of investigation are of considerable importance as treatment of co-morbid obesity and depression is likely to fail if both conditions are not simultaneously and effectively addressed.

Bio: Julio Licinio, M.D., F.A.P.A., is Professor and Director, John Curtin School of Medicine, The Australian National University, and head of the Department of Translational Medicine. He is also a Research Professor at the University of Southern California, in Los Angeles. Professor Licinio is originally from Brazil and lived for over 25 years in the United States, where he had clinical and research training in endocrinology and psychiatry at University of Chicago and Cornell. He then held academic positions at Yale, NIH, and UCLA, where he was Professor and Vice-Chair of Psychiatry and Director of the Center for Pharmacogenomics. Prior of moving to Australia, he was Miller Professor, Chairman of Psychiatry and Associate Dean at University of Miami. His work on the fundamental endocrine and pharmacogenomic mechanisms at the interface of obesity and depression has been extensively funded by NIH, and it is highly cited in the scientific literature. Dr. Licinio is the Founding Editor of three Nature Publishing Group journals, The Pharmacogenomics Journal, Translational Psychiatry and Molecular Psychiatry, which has an Impact Factor of 15, the highest in its field worldwide.



Elissa S. Epel, Ph.D.

University of California San Francisco
Stress and Resilience Factors for Cellular Aging



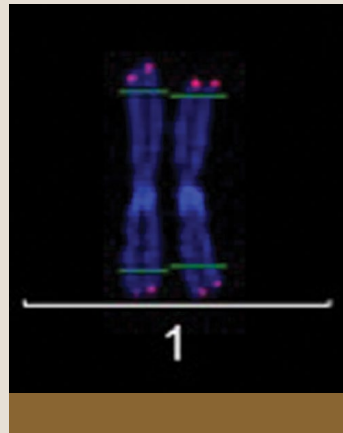
Abstract: This lecture will review the psychological and behavioral correlates of aspects of cellular aging, including oxidative stress and mainly telomere length, as well as interventions that have examined changes in cell aging. There has long been a search for 'psychobiomarkers'—measures that index psychosocial stress and well-being, and precede and predict early disease and mortality. Telomeres appear to be such a psychobiomarker. There have been many studies that link telomere shortness to psychological stress, as well as to states of severe distress. This lecture will discuss possible commonalities underlying these states of emotional distress that may be promoting telomere attrition, both psychological processes, such as threat appraisal, that promote stress arousal, as well as biochemical mediators of stress arousal that are known to affect the telomere/telomerase maintenance system.

Telomere length appears partly under personal control, as a number of health behaviors have now been linked to telomere length—including activity, nutrition, and sleep. Exercise in particular may serve not only as a main effect but may also buffer the effect of stress on telomere attrition.

As a psychobiomarker, the telomere/telomerase system may serve as a possible barometric health marker for behavioral change interventions. Although few intervention studies have been done to date, preliminary data indicate that interventions may impact rate of telomere attrition. Human studies are needed to understand telomere dynamics over time, and modifiers, but ultimately basic in vitro and animal studies are needed to test mechanisms of telomere regulation, experimentally.

Bio: Elissa Epel is an Associate Professor in the Department of Psychiatry at UCSF. Dr. Epel received her training in psychology from Stanford and Yale University, with a focus on health psychology and behavioral medicine, and subsequently completed clinical training, focusing on behavioral medicine, at the Palo Alto Veterans Administration Health Care System, and a postdoctoral fellowship (in Psychology and Medicine) at UCSF. She is a faculty member of the Health Psychology program, the UCSF Osher Center for Integrative Medicine, the Robert Wood Johnson Health and Society Scholars fellowship program, the Assistant Director of the Center for Health and Community, and Director of Research for the UCSF Center on Obesity.

She has interests in the impact of stress physiology on 'metabolic health,' including food intake, insulin resistance, obesity, and premature aging at the cellular level, and how health enhancing interventions might enhance regulation in these systems. Along with Elizabeth Blackburn and colleagues, she demonstrated novel links between stress and stress arousal with markers of cellular aging (telomere length and telomerase activity). She aims to understand, from a psychological and biological perspective, why some people are vulnerable and others are resilient to chronic stress, and how much of accelerated aging is due to changes in metabolism and eating behavior. She focuses on mothers of children with chronic conditions, mainly autism, and older people caring for family members with dementia. She is also involved in clinical trials examining how stress reduction interventions might reverse or slow cellular aging.



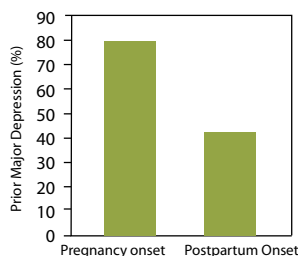
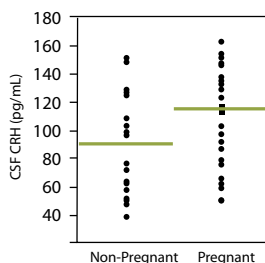


Margaret Altemus, M.D.

Weill Cornell Medical College
Sex Differences in Stress Responses and
Psychiatric Disorders

Abstract: The lecture will provide an overview of sex differences in stress responses, with a focus on sex differences in psychiatric disorders. Depression and anxiety disorders are well known to be more common in women, and the course of these

disorders are influenced by reproductive events, including puberty, the menstrual cycle, pregnancy and menopause. Sex differences in physiological stress responses in humans also have been identified, but the mechanisms through which sex differences in stress responses may impact vulnerability to affective illness are not well understood. Potentially more important than sex differences in vulnerability to depression and anxiety disorders, are marked individual differences in the nature of behavioral responses to reproductive hormones. Better characterization of reproductive related psychiatric disorders is likely to provide more biologically distinct subtypes of depression and anxiety disorders which can more readily be linked to pathophysiological mechanisms. In addition, better understanding of the role of reproductive hormones in stress physiology and psychiatric illness is likely to lead to new approaches to prevention and treatment.



Postpartum Depression may be a distinct subtype of Major Depression

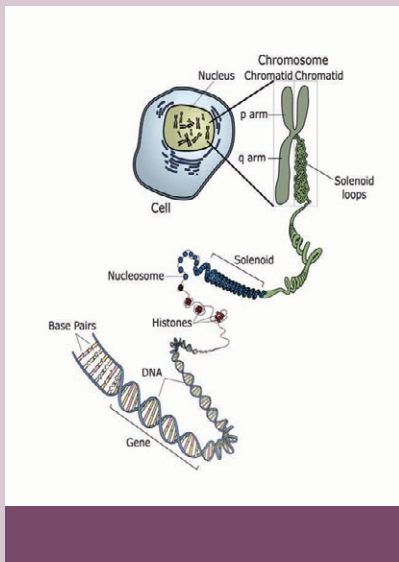
Bio: Margaret Altemus, M.D. is an Associate Professor of Psychiatry and Complementary and Integrative Medicine at the Weill Medical College of Cornell University in Manhattan. Dr. Altemus had clinical and research training in psychiatry at Yale and the NIH Intramural Research Program before coming to Cornell. Dr. Altemus directs a neuroendocrinology research laboratory focused on the physiological interplay between stress, reproductive hormones, and affective disorders. She also is the director of the Payne Whitney Women's Program, which provides clinical care and clinical training in addition to research in reproductive-related psychiatric disorders. Dr. Altemus is the Core Director of the Participant and Clinical Interactions Resource of the Weill Cornell CTSC.

Eric J. Nestler, M.D., Ph.D.

Mount Sinai Medical Center
Transcriptional and Epigenetic
Mechanisms of Depression



Abstract: Depression is a common, chronic, and debilitating syndrome. Only about half of depressed patients show a complete remission to available treatments, which underscores the need for more effective agents. The mechanisms that precipitate depression, such as stress, are incompletely understood. One mystery of depression is its long-lasting nature and delayed response to antidepressant treatment, phenomena which might be mediated in part by transcriptional and epigenetic regulation.



We have used chronic social defeat stress as an animal model of depression that mimics certain symptoms of human depression. Prolonged exposure to an aggressor induces lasting changes in mouse behavior such as social avoidance, which are reversed by chronic (but not acute) treatment with standard antidepressant treatments. At a molecular level, we have documented changes in chromatin remodeling at particular genes in the nucleus accumbens, a key brain reward region, in response to chronic social defeat stress. This work has involved the examination of candidate genes, ChIP (chromatin immunoprecipitation) methods to obtain a genome-wide view of epigenetic modifications, and studies of transcription factors, for example, CREB, that mediate these effects. Interestingly, roughly a third of animals subjected to chronic defeat stress avoid most of its deleterious consequences, which has made it possible to study the molecular basis of resilience as well, including the role of certain transcription factors such as Δ FosB in mediating this adaptive response. We have directly related several changes in gene expression and chromatin remodeling to the behavioral changes observed. Moreover, we have demonstrated stress regulation of chromatin-modifying enzymes, including certain

histone deacetylases and methyltransferases and DNA methyltransferases, which appear to mediate some of the lasting effects of social defeat stress on gene expression within this brain region.

This work provides novel insight into the molecular mechanisms by which chronic stress produces lasting changes in brain to cause depression-like symptoms. The findings also suggest novel leads for the development of new antidepressant treatments, including mimicking coping mechanisms mounted by resilient individuals.

Bio: Dr. Nestler is the Nash Family Professor of Neuroscience at the Mount Sinai School of Medicine in New York, where he serves as Chair of the Department of Neuroscience and Director of the Friedman Brain Institute. He received his B.A., Ph.D., and M.D. degrees, and psychiatry residency training, from Yale University. He served on the Yale faculty from 1987-2000, where he was the Elizabeth Mears and House Jameson Professor of Psychiatry and Neurobiology, and Director of the Division of Molecular Psychiatry. He moved to Dallas in 2000 where he served as the Lou and Ellen McGinley Distinguished Professor and Chair of the Department of Psychiatry at The University of Texas Southwestern Medical Center until moving to New York in 2008. Dr. Nestler is a member of the Institute of Medicine and a Fellow of the American Academy of Arts and Sciences. The goal of Dr. Nestler's research is to better understand the molecular mechanisms of addiction and depression based on work in animal models, and to use this information to develop improved treatments of these disorders.

<http://www.mountsinai.org/profiles/eric-j-nestler>



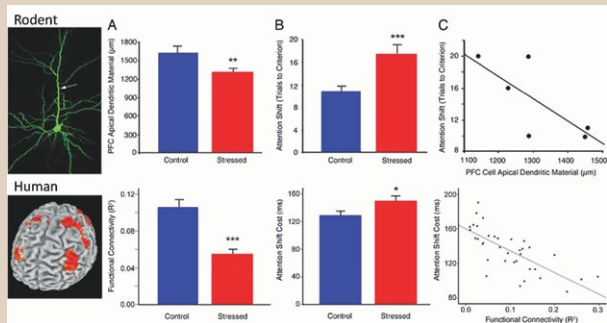
Photo provided by WCMC

BJ Casey, Ph.D.

Weill Cornell Medical College
Basic and Translational Studies on the
Developmental Psychobiology of Risk
for Anxiety and Stress Related Disorders

Abstract: Anxiety and stress related disorders are the most common of the psychiatric disorders with a heightened increase during adolescence and affecting as many as 10-20% of our youth. One of the most commonly used therapies to treat these disorders is exposure-based cognitive behavioral therapy that relies on basic principles of

fear learning and extinction. A substantial portion of patients improves with this therapy, but 40-50% do not. This presentation will provide an overview of our recent empirical studies employing human imaging, rodent models and mouse genetics to examine how attention, fear and stress related processes differ across development and across individuals. Behavioral, genetic and brain imaging data will be provided to offer insight for whom may be at risk for anxiety and for whom and when, during development, exposure based treatment may be most effective for treating individuals with anxiety and stress related disorders.



Liston, McEwen & Casey PNAS 2009

Bio: Dr. BJ Casey is the Sackler Professor for Developmental Psychobiology. She holds appointments in the Departments of Psychiatry, Neurology and Neuroscience at Weill Cornell Medical College and in the Department of Psychology at Cornell University. She directs the Sackler Institute and Neuroscience Graduate Program in addition to directing the NIMH funded Center for Brain, Genetic and Behavioral (CBGB) research across development.

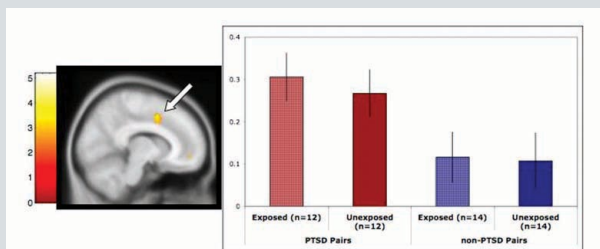
Casey is a world leader in pediatric neuroimaging and its use in typical and atypical development. She skillfully uses brain imaging to uniquely examine transitions into and out of developmental periods, such as the period of adolescence. Her work is grounded in translational studies from rodent to human, developing models for several psychiatric disorders and potential treatments. She is a member of several advisory boards including the NIMH Board of Scientific Counselors, the Scientific Advisory Board for NARSAD, the National Research Council Board of Children, Youth and Families and Committee for Assessing Juvenile Justice Reform.

Roger Pitman, M.D.

Massachusetts General Hospital
Studies of Identical Twins Discordant
for Combat Exposure in Vietnam



Abstract: Cross-sectional studies of biological markers for post-traumatic stress disorder (PTSD) are unable to reveal their origins. A marker a) may represent a familial risk factor for PTSD, or b) have been acquired as a result of the traumatic event or the PTSD that followed it. We have studied male identical twin pairs discordant for combat exposure in Vietnam. Some of the exposed (Ex) twins developed combat-related PTSD (ExP+), and some did not (ExP-). The unexposed (Ux) co-twins of combat veterans with PTSD (UxP+) are deemed "high-risk," whereas the unexposed co-twins of combat veterans without PTSD (UxP-) are deemed "low-risk." If a PTSD biomarker represents a familial risk factor, it should be present in UxP+ but not UxP- co-twins. If acquired, a biomarker should be present in ExP+ twins but not their UxP+ co-twins. The following biomarkers have been found to represent familial risk factors for PTSD: increased neurological soft signs, decreased hippocampal volume, and increased resting metabolism in dorsal anterior cingulate cortex. The following biomarkers have been found to represent acquired PTSD abnormalities: increased heart rate response to startling stimuli, diminished P3



Resting Hypermetabolism in Dorsal Anterior Cortex/Mid-Cingulate Cortex in Identical Twins Discordant for Combat Exposure in Vietnam. (Shin et al. Arch Gen Psychiat 2009;66:1099-1107)

event-related potential response, increased P2 amplitude-intensity slope, failure to retain extinction of a conditioned fear response, and decreased pregenual anterior cingulate gray matter volume. Despite the presence of biological risk factors, UxP+ co-twins, in contrast to their ExP+ twins, do not show substantial psychopathology on psychometric testing. PTSD comprises a mix of familial biological risk factors and acquired abnormalities. Identified biological risk factors only lead to psychopathology after the occurrence of a traumatic event.

Bio: Dr. Pitman is a psychiatrist at Massachusetts General Hospital and Professor of Psychiatry at Harvard Medical School, Boston, MA. He served as a psychiatrist in the U.S. Navy during the Vietnam War and went on to complete a 30-year career in the Department of Veterans Affairs prior to moving to MGH. He is the recipient of the International Society for Traumatic Stress Studies' Award for Outstanding Scientific Achievement in the field of PTSD and its Lifetime Achievement Award. Dr. Pitman's research into the psychobiology of post-traumatic stress disorder (PTSD) spans more than 25 years. He has more than 100 peer-reviewed publications on PTSD and more than 200 overall publications in the general psychiatric and medical literature. He has authored or co-authored numerous structural and functional neuroimaging studies of PTSD. For the past 15 years, he has been conducting a large-scale, psychobiologic investigation of a national sample of monozygotic twins discordant for combat exposure in Vietnam.

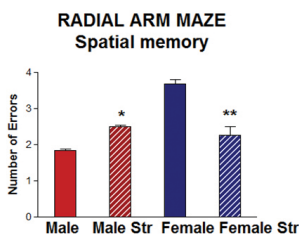
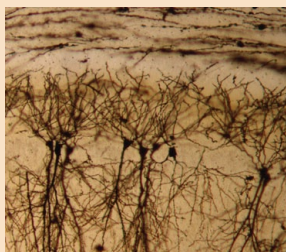


Victoria Luine, Ph.D.

Hunter College, City University of New York
Accounting for Sex and Age in Neural
Responses To Stress

Abstract: The debilitating effects of chronic stress have been documented in systems ranging from neural cells in culture to laboratory rodents, sub-human primates and humans. While there is general agreement on the adaptive properties of acute stress vs. the deleterious nature of chronic stress, it is remarkable to note that the vast majority of studies were conducted in males only. Thus, it is only recently that the response of females to stress has been investigated and surprisingly, responses in females can be dramatically different than in males, especially in relation to cognitive function. For ex., chronic stress impairs male rats' cognitive performance whereas female rodents show enhanced performance on the same memory tasks following stress. Anxiety responses are also dependent on sex - chronic stress is anxiolytic in males and anxiogenic in females. These sexually differentiated responses to stress appear early in development, become somewhat larger through adolescence and adulthood and then abate somewhat at advanced ages. Moreover, different behavioral responses to chronic stress are found in developing as well as aged subjects as compared to adults. In aged rats, chronic stress enhances some aspects of cognition in both sexes, and anxiety effects are opposite to young adults. When pregnant dams are exposed to chronic stress, their offspring display yet different consequences of stress on anxiety and cognition, and, in contrast to adulthood when most effects of stress are reversible over time, prenatal stress effects are enduring. These behavioral changes are mediated by stress effects on neuronal morphology, neurotransmitters and synaptic function. Thus, current research suggests that theories of stress dependent modulations in CNS function - developed mainly in male models, focused mainly on peripheral physiological processes and tested in adult males - may require revision when applied to a more diverse population. Moreover, sex and age should be considered in the etiology and treatment of stress-related illnesses.

ous nature of chronic stress, it is remarkable to note that the vast majority of studies were conducted in males only. Thus, it is only recently that the response of females to stress has been investigated and surprisingly, responses in females can be dramatically different than in males, especially in relation to cognitive function. For ex., chronic stress impairs male rats' cognitive performance whereas female rodents show enhanced performance on the same memory tasks following stress. Anxiety responses are also dependent on sex - chronic stress is anxiolytic in males and anxiogenic in females. These sexually differentiated responses to stress appear early in development, become somewhat larger through adolescence and adulthood and then abate somewhat at advanced ages. Moreover, different behavioral responses to chronic stress are found in developing as well as aged subjects as compared to adults. In aged rats, chronic stress enhances some aspects of cognition in both sexes, and anxiety effects are opposite to young adults. When pregnant dams are exposed to chronic stress, their offspring display yet different consequences of stress on anxiety and cognition, and, in contrast to adulthood when most effects of stress are reversible over time, prenatal stress effects are enduring. These behavioral changes are mediated by stress effects on neuronal morphology, neurotransmitters and synaptic function. Thus, current research suggests that theories of stress dependent modulations in CNS function - developed mainly in male models, focused mainly on peripheral physiological processes and tested in adult males - may require revision when applied to a more diverse population. Moreover, sex and age should be considered in the etiology and treatment of stress-related illnesses.



Hippocampal Neurons & Stress Effects On Memory

Bio: Dr. Luine is a Distinguished Professor of CUNY and holds appointments in the Dept. of Psychology and the Gene Center at Hunter College, The Graduate Center of CUNY and Rockefeller University. She is Director of the NIH funded RISE and SCORE Research Programs for students and faculty, respectively. She received her PhD in Pharmacology from SUNY at Buffalo and was a post doctoral fellow and faculty member at Rockefeller University before joining Hunter College in 1987. Luine's research, primarily in animal models, has shown that hormones of adrenal and gonadal origin alter neural function and contribute to changes in cognition and mood over the lifespan. Of particular interest is understanding how gonadal hormones, estradiol in females and testosterone in males, contribute to sex differences in responses to stress, psychoactive drugs and the aging process. She has served on numerous government and private review and advisory panels and is currently Secretary of the Federation of Associations in Brain and Behavioral Sciences and Chair of the Membership Comm. for the Organization for the Study of Sex Differences. Prof. Luine has received the CUNY Chancellor's Award numerous times as well as an Outstanding Woman Scientist award from the Association for Women in Science and the Society for Neuroscience's Bernice Grafstein Award for Mentoring in 2009.

<http://www.hunter.cuny.edu/psychology/faculty/the-faculty-folder/luine>

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