

8:00 a.m. – 11:30 a.m.
President's Plenary
Grand Ballroom

President's Plenary

Welcoming Remarks and Moment of Silence

John Krystal
President

Presentation of Honorific Awards

Eric Nestler
Chair, Honorific Awards Committee

Taking Stock of the Connectome

- 8:30 a.m. Order and Complexity in the Development of Synaptic Connectivity
Jeffrey Lichtman
- 9:15 a.m. Integrating Clinical, Genetic and Neuroimaging Data
Eric Schadt
- 10:00 a.m. Connectomics, Psychiatry and Drug Development
Edward Bullmore
- 10:45 a.m. Making Sense of the Functional Connectomes
Bruce Rosen

PL

8:00 a.m. – 11:30 a.m.

President's Plenary

Grand Ballroom

PL

Order and Complexity in the Development of Synaptic Connectivity

Jeff Lichtman

Harvard University

Jeff Lichtman, M.D., Ph.D., Professor of Molecular and Cellular Biology at Harvard University. Dr. Lichtman characterizes the processes that shape the development of synaptic connectivity in the developing brain. His research yields a unique record of the way that the formation and elimination of synapses with the context of life experiences yields distinct patterns of connectivity that constitutes a way in which these experiences are encoded. He is a remarkably engaging speaker on this topic as his mastery of imaging tools and presentation technologies is quite distinctive. This presentation highlights the enormous complexity and variability of synaptic connectivity at the “micro level”, a critical caution to overly facile interpretation of connectivity at the “macro level” of human MRI research.

A Systems Framework for Understanding the Complexity of Human Diseases

Eric Schadt

Mount Sinai School of Medicine

PL

Common human diseases and drug response are complex traits that involve entire networks of changes at the molecular level driven by genetic and environmental perturbations[1]. Changes at the molecular level can induce changes in biochemical processes or broader molecular networks that affect cell behavior, and changes in cell behavior can affect normal tissue or whole organ function, eventually leading to pathophysiological states at the organism level that we associate with disease. While the vast majority of previous efforts to elucidate disease and drug response traits have focused on single dimensions of the system, achieving a more comprehensive view of common human diseases requires examining living systems in multiple dimensions and at multiple scales[1-3].

Studies focused on identifying changes in DNA that correlate with changes in disease or drug response traits, changes in gene expression that correlate with disease or drug response traits, or changes in other molecular traits (e.g., metabolite, methylation status, protein phosphorylation status, and so on) that correlate with disease or drug response are fairly routine and have met with great success in many cases. However, to further our understanding of the complex network of molecular and cellular changes that impact disease risk, disease progression, severity, and drug response, we can more formally integrate these different data dimensions[4]. Here I present an approach for integrating a diversity of molecular and clinical trait data to uncover models that predict complex system behavior. By integrating diverse types of data on a large scale I demonstrate that some forms of common human diseases like diabetes are most likely the result of perturbations to specific gene networks that in turn causes changes in the states of other gene networks both within and between tissues that drive biological processes associated with disease[5-8]. These models elucidate not only primary drivers of disease and drug response, but they provide a context within which to interpret biological function, beyond what could be achieved by looking at one dimension alone[4-6, 9-11]. That some forms of common human diseases are the

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A Systems Framework for Understanding the Complexity of Human Diseases

Eric Schadt (continued)

result of complex interactions among networks has significant implications for drug discovery: designing drugs or drug combinations to impact entire network states rather than designing drugs that target specific disease associated genes[2].

Dr. Eric Schadt recently joined Mount Sinai Medical School as Chairman and Professor, Department of Genetics and Genomic Sciences and as Director, Institute of Genomics and Multiscale Biology (effective 1 August 2011). Previously, Dr. Schadt had joined Pacific Biosciences as Chief Scientific Officer in June 2009 to oversee the scientific strategy for the company, including creating the vision for next-generation sequencing applications of the company's technology. Dr. Schadt is also a founding member of Sage Bionetworks, an open access genomics initiative designed to build and support databases and an accessible platform for creating innovative, dynamic models of disease. Dr. Schadt's current efforts at Mount Sinai to generate and integrate large-scale, high-dimension molecular, cellular, and clinical data to build more predictive models of disease so that we may better diagnose and treat disease, were motivated by the genomics and systems biology research he led at Merck to elucidate common human diseases and drug response using novel computational approaches applied to genetic and molecular profiling data. His research helped revolutionize a field in statistical genetics (the genetics of gene expression), has energized the systems biology field, and has led to a number of discoveries relating to the causes of common human diseases. At the time Dr. Schadt left Merck in 2009, greater than 50% of all new drug discovery programs at Merck in the metabolic space were derived from Dr. Schadt's work. Dr. Schadt was also recently appointed as Fellow to the Institute of Systems and Synthetic Biology, Imperial College London. Dr. Schadt received his B.S. in applied mathematics/computer science from California Polytechnic State University, his M.A. in pure mathematics from UCD, and his Ph.D. in bio-mathematics from UCLA (requiring Ph.D. candidacy in molecular biology and mathematics).

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Connectomics, Psychiatry and Drug Development

Edward Bullmore

The University of Cambridge/GlaxoSmithKline (GSK)

PL

Psychiatric disorders are increasingly recognized to emerge from abnormally connected human brain networks. The recent growth of connectomics as a way of looking at brain organization is therefore a positive development for psychiatry. I will briefly review concepts from graph theory that have been used to map normal human brain networks, and I will show how some of the key topological features of human fMRI and DTI networks are characteristic also of nervous systems at cellular scale and in different species. The first complex network studies of patients with neuropsychiatric disorder have demonstrated abnormalities of network organization, some of which can be related to impairments of cognition and to generative models of abnormal network development in schizophrenia, or to degenerative network models of dementia. There are several ways in which the connectomic approach could support CNS drug development in future. Recent data suggest that fMRI connectomics can provide theoretically principled markers of pro-cognitive drug effects; can be used to test differentiation between in-class competitor drugs at the level of human brain function; and, more speculatively, may be useful as a translational predictor of a drug's effects on human brain function and cognition based on its effects on network properties of animal nervous systems.

Ed Bullmore trained in clinical medicine at the University of Oxford and St Bartholomew's Hospital in London, then worked as a Lecturer in Medicine at the University of Hong Kong, before specialist clinical training in psychiatry at St George's Hospital and then the Bethlem Royal & Maudsley Hospital London. His research career started in the early 1990s as a Wellcome Trust (Advanced) Research Fellow and was initially focused on mathematical analysis of neurophysiological time series. Since moving to Cambridge as Professor of Psychiatry in 1999, his interest in human brain function and structure has increasingly focused on complex brain networks identified in MRI and other brain scanning data. Since 2005, he has worked half-time for GlaxoSmithKline as Head of GSK's Clinical Unit in Cambridge and Vice-President, Experimental

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Connectomics, Psychiatry and Drug Development

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Edward Bullmore (continued)

Medicine. He is Deputy Director of the Wellcome Trust/GSK funded training programme in Translational Medicine and Therapeutics, Clinical Director of the Wellcome Trust/MRC funded Behavioural & Clinical Neuroscience Institute, and an honorary Consultant Psychiatrist and Director of R&D in Cambridgeshire & Peterborough Foundation NHS Trust. He has published about 300 scientific papers (see http://scholar.google.co.uk/citations?hl=en&user=It_G4zsAAAAJ for bibliography) and he has been elected as a Fellow of the Royal College of Physicians, the Royal College of Psychiatrists, and the Academy of Medical Sciences.

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Making Sense of the Functional Connectomes

Bruce Rosen

Harvard University

PL

Dr. Rosen is Professor of Radiology at the Harvard Medical School and Professor of Health Science and Technology at the Harvard-MIT Division of Health Sciences and Technology. He is Director of the Athinoula A. Martinos Center for Biomedical Imaging at Massachusetts General Hospital.

Dr. Rosen's research over the past thirty years has focused on the development and application of physiological and functional NMR techniques, most recently, on the fusion of fMRI data with information from other modalities, including positron emission tomography (PET), magnetoencephalography (MEG) and noninvasive optical imaging. Dr. Rosen leads the activities of several large interdisciplinary and inter-institutional research programs including the NIH Blueprint-funded Human Connectome Project, the NCCR Regional Resource Center, the Center for Functional Neuroimaging Technologies (CFNT), and the Biomedical Informatics Research Network (BIRN) Collaborative Tools Support Network. He is Principal Investigator/Program Director for two neuroimaging training programs, and he has mentored dozens of graduate students and research fellows through the years.

Dr. Rosen is a Fellow and Gold Medal winner of the International Society for Magnetic Resonance in Medicine, a Fellow of the American Institute for Medical and Biological Engineering, and a member of the Institute of Medicine of the National Academies.

11:30 a.m. – 1:00 p.m.
Women's Luncheon
Great Hall 5 & 6

PL

Women's Luncheon

Looking Back in Amazement:
What I Learned on the Way to this Luncheon

Co-Chairs: Karen F. Berman and Linda S. Brady

Presented by:
Huda Akil

11:30 a.m. – 1:00 p.m.
Women's Luncheon
Great Hall 5 & 6

Looking Back in Amazement: *What I Learned on the Way to this Luncheon*

Huda Akil

University of Michigan

PL

Huda Akil, Ph.D. is the Gardner Quarton Distinguished University Professor of Neuroscience and Psychiatry and the co-Director of the Molecular & Behavioral Neuroscience Institute at the University of Michigan. Dr. Akil has made seminal contributions to the understanding of the brain biology of emotions, including pain, anxiety, depression and substance abuse. She and her colleagues provided the first physiological evidence for a role of endorphins in the brain; and showed that endorphins are activated by stress and cause pain inhibition.

Dr. Akil's current research investigates the genetic, molecular and neural mechanisms underlying stress, addiction and mood disorders. She is engaged in large-scale studies to discover new genes and proteins that cause vulnerability to major depression and bipolar illness. She is the author of over 500 original scientific papers, and has been recognized as one of the most highly cited neuroscientists by the ISI Citation Index.

Dr. Akil's scientific contributions have been recognized with numerous honors and awards. These include the Pacesetter Award from the National Institute on Drug Abuse in 1993 and the Pasarow Award for Neuroscience Research in 1994. In 1998, she received the Sachar Award from Columbia University and the Bristol Myers Squibb Unrestricted Research Funds Award. She is also the recipient of the Society for Neuroscience Mika Salpeter Lifetime Achievement Award and the NARSAD Patricia Goldman-Rakic Prize for Cognitive Neuroscience (2007), and most recently the Paul Hoch Distinguished Service Award from the American College of Neuropsychopharmacology (2010).

In 1994, she was elected to the membership of the Institute of Medicine (IOM) of the National Academy of Science. She was elected as a Fellow of the American Association for the Advancement of Science in 2000. In 2004, she was elected to the American Academy of Arts and Sciences. In 2011 she was elected to the National Academy of Sciences.

11:30 a.m. – 1:00 p.m.

Women's Luncheon

Great Hall 5 & 6

PL

Looking Back in Amazement: *What I Learned on the Way to this Luncheon*

Huda Akil (continued)

Dr. Akil has served on numerous boards and scientific councils, numerous non-profit national and international organizations to promote scientific and brain health awareness nationally and globally. She is the past President of the American College of Neuropsychopharmacology (1998) and the past President of the Society for Neuroscience (2004) the largest neuroscience organization in the world. She has co-chaired the Neuroscience Steering Committee at the Foundation for the National Institute of Health, and served two terms on the Council of the Institute of Medicine of the US National Academy of Sciences.

**1:30 p.m. – 3:00 p.m.
Distinguished Lecture
Grand Ballroom**

Distinguished Lecture

Leptin and the Biologic Basis of Obesity

Presented by:
Jeffrey Friedman

PL

1:30 p.m. – 3:00 p.m.
Distinguished Lecture
Grand Ballroom

Leptin and the Biologic Basis of Obesity

PL

Jeffrey Friedman
Rockefeller University

The discovery of leptin has led to the elucidation of a robust physiologic system that maintains fat stores at a relatively constant level. Leptin is a peptide hormone secreted by adipose tissue in proportion to its mass. This hormone circulates in blood and acts on the hypothalamus to regulate food intake and energy expenditure. When fat mass falls, plasma leptin levels fall stimulating appetite and suppressing energy expenditure until fat mass is restored. When fat mass increases, leptin levels increase, suppressing appetite until weight is lost. By such a mechanism total energy stores are stably maintained within a relatively narrow range.

Recessive mutations in the leptin gene are associated with massive obesity in mice and some humans. Treatment with recombinant leptin markedly reduces food intake and body weight. The low leptin levels in patients with leptin mutations are also associated with multiple abnormalities including infertility, diabetes and immune abnormalities all of which are corrected by leptin treatment. These findings have established important links between energy stores and many other physiologic systems and led to the use of leptin as a treatment for an increasing number of other human conditions including a subset of obesity, some forms of diabetes including lipodystrophy and hypothalamic amenorrhea, the cessation of menstruation seen in extremely thin women. Identification of a physiologic system that controls energy balance establishes a biologic basis for obesity and further establishes links between leptin and numerous other physiologic responses. Recent studies have explored the relationship between leptin and the reward value of food. In addition, new methods for identifying neurons activated by leptin and other stimuli have been developed.

Dr. Jeffrey Friedman is a physician scientist studying the genetic mechanisms that regulate body weight. Dr. Friedman's research on various aspects of obesity received national attention in late 1994, when it was announced that he and his colleagues had isolated the mouse ob gene and its human homologue. They subsequently found that injections of the encoded protein, leptin, decreases body weight of mice by reducing food intake and increasing energy expenditure.

1:30 p.m. – 3:00 p.m.
Distinguished Lecture
Grand Ballroom

Leptin and the Biologic Basis of Obesity

Jeffrey Friedman (continued)

PL

Current research is aimed at understanding the genetic basis of obesity in human and the mechanisms by which leptin transmits its weight reducing signal.

He is currently a Professor at the Rockefeller University, an Investigator at the Howard Hughes Medical Institute and the Director of the Starr Center for Human Genetics. Dr. Friedman's affiliation with The Rockefeller University began in 1980, where he was awarded a Ph.D. degree in 1986. He was appointed Assistant Investigator with the Howard Hughes Medical Institute at Rockefeller in 1986, promoted to Associate Investigator in 1991, and Investigator in 1997. Dr. Friedman received an MD. degree from Albany Medical College in 1977 and completed a medical residency at Albany Medical College in 1980. Dr. Friedman was born in Orlando, Florida, on July 20, 1954, and grew up in North Woodmere, Long Island. He graduated from Rensselaer Polytechnic Institute magna cum laude and, at the age of 22, received his medical degree from Albany medical College of Union University in Albany, New York. While at Albany Medical College, he was elected to Alpha Omega Alpha, the medical honor society. After completing a residency in Internal Medicine at Albany Medical Center Hospital, Dr. Friedman came to Rockefeller as a postgraduate fellow and associate physician in 1980. From 1980 to 1981, he also served as a postgraduate fellow at Cornell University Medical College. In 1986, he received a Ph.D. under the tutelage of Professor James E. Darnell, was appointed assistant professor, and became an assistant investigator at the Howard Hughes Medical Institute. Dr. Friedman was appointed Professor at Rockefeller in 1995 after serving as Associate Professor and Head of Laboratory of Molecular Genetics at the Institution since 1991 and in 1998 awarded the Marilyn M. Simpson Professorship. In 1995 he was appointed Director of the Starr Foundation Center for Human Genetics.

Dr. Friedman was elected to the National Academy of Science in 2001. His work was referred to in Time Magazine's Best of Science Section in 1995 and 1996. He has also received Popular Science's, Best of Science Award in 1995, the Alumnus of the Year Award, 1996, from Albany Medical College, the Heinrich Wieland Prize, 1996, the Jacobaeus Prize, University of Goteborg, 1997, the Steven C.

1:30 p.m. – 3:00 p.m.
Distinguished Lecture
Grand Ballroom

Leptin and the Biologic Basis of Obesity

PL

Jeffrey Friedman (continued)

Beering Award, Indiana University School of Medicine, 1999, the Janssen Award for Special Achievement in Gastroenterology, 1999, the Endocrinology Transatlantic Medal, Society for Endocrinology, United Kingdom, 2000, the Osborne Mendel Award, American Society for Nutritional Sciences, 2000, the Rolf Luft Award, Karolinska Hospital, Stockholm, Sweden, 2000, and the Bristol-Myers Squibb Award for distinguished Achievement in Metabolic Research. He has delivered the Shelton Lecture, Harvard University, 1996, the Peters' Lecture, Yale University, 1996, the Carl Vernon Moore Lecture, Washington University, 1997, the Allan D. Bass Lecture, Vanderbilt University, 1997, the Priscilla White Lecture, Joslin Diabetes Center, 1998, the Chilton Foundation Lecture, University of Texas, 1998, the Jack Gross Memorial Lecture, Israel, 1998, the Van Wyck Lecture, University of North Carolina, 1999, the Verna and Marrs McLean Lecture, Baylor College of Medicine, 1999, the Banting Lecture of the British Diabetes Association, 2002 the Passano Award, 2005, elected to The Royal Swedish Academy of Sciences, Foreign Member, 2005, the Gairdner International 2005, Kovalenko Medal, 2006, Honorary Doctorate, Molecular Genetics, Maastricht University, The Netherlands, 2006, Danone International Prize for Nutrition, 2007, Keio Medical Science Prize, Keio University, 2009, Shaw Prize for Life Sciences and Medicine, 2009, Thomson Reuters Citation Laureate, 2010, Pasarow Foundation Award, 2010, and the Albert Lasker Basic Medical Research Award, 2010.

3:00 p.m. – 4:15 p.m.
Mini Panel
Diplomat Ballroom 1 & 2

Renaissance in Opioid Biology: From Preclinical Concepts to Clinical Practice

Chair: Floyd E. Bloom

- 3:00 p.m. Molecular Basis for Kappa Opioid Receptor Antagonism:
Implications of Ligand-directed Signaling for the Development
of Novel Antidepressants
Charles Chavkin
- 3:25 p.m. The Place of Opiates in the Cortico-basal Ganglia Reward
Circuit
Suzanne Haber
- 3:50 p.m. New Clinical Research in Opioid Modulation Indicates Novel
Utility in Treating Resistant Depression
Elliot W. Ehrich

MP

4:15 p.m. – 5:30 p.m.

Mini Panel

Diplomat Ballroom 1 & 2

Interaction of Ontogeny and Environment in Adolescent Substance Abuse

Chair: Cynthia Kuhn

Co-Chair: Sari Izenwasser

MP

- 4:15 p.m. Adolescent Response to Reward and Adversity
Cynthia Kuhn
- 4:40 p.m. Intersection of Environment, Individual and Drug in
Development of Substance Abuse in Adolescence
Sari Izenwasser
- 5:05 p.m. Environmental Stressors and Risk for Alcohol Problems:
A Longitudinal GxE GWAS in Community Samples
William Copeland

3:00 p.m. – 5:30 p.m.
Panel
Regency Ballroom 2

Neuroplasticity Deficits in Neuropsychiatric Illness: New Targets for Cognitive Enhancement

Chair: Daniel Javitt

- 3:00 p.m. Learning Mechanisms in Obsessive-compulsive Disorder: Bias to Stimulus-Response Habit Learning
Trevor W. Robbins
- 3:30 p.m. Experience Dependent Cortical Long-term Synaptic Potentiation (LTP) and Sequelae in the Intact Visual System
Mark Bear
- 4:00 p.m. Induction of Neuroplasticity in Humans by Transcranial Direct Current Stimulation: Clinical Applications and Methodological Advancements
Michael A. Nitsche
- 4:30 p.m. Neurophysiological Basis of Auditory/Motor Plasticity Deficits and tDCS Effects in Schizophrenia
Daniel Javitt
- 5:00 p.m. Discussant: *Richard Keefe*

PA

3:00 p.m. – 5:30 p.m.

Panel

Atlantic Ballroom 1

Common Neural Mechanisms across Dimensions of Pediatric Psychopathology

Chair: Danny Pine

Co-Chair: Kate D. Fitzgerald

PA

3:00 p.m. Human Amygdala Development Following Early-life Stress
Nim Tottenham

3:30 p.m. Childhood Disruptive Behavior Disorders and Risk for
Adolescent Substance Use
Iliyian Ivanov

4:00 p.m. Functional and Structural MRI Studies of the Neural Circuits that
Mediate Self-regulation over Development in Bulimia Nervosa
Rachel Marsh

4:30 p.m. Neural Response to Social Threat: Disease Specificity in
Adolescents with Generalized Anxiety Disorder, Social Phobia,
and at Risk Populations
Jarcho M. Johanna

5:00 p.m. Discussant: *Francisco Xavier Castellanos*

3:00 p.m. – 5:30 p.m.

Panel

Regency Ballroom 3

Unraveling the Genetic Architecture of Mental Illness with Whole Genome Sequence Data

Chair: Carrie Bearden

- 3:00 p.m. The Utility of Whole Genome Sequencing in Human Pedigrees
for Identifying Genes Underlying Human Quantitative Trait Loci
John Blangero
- 3:30 p.m. Endophenotypes, Normal Variation and Whole Genome
Sequence Data in Pedigrees: Insights into the Genetics of
Psychotic Illnesses
David C. Glahn
- 4:00 p.m. Rare Variants in Genes Involved in Neurotrophin Signaling
Identified by Genome Sequencing in Bipolar Disorder
John R. Kelsoe
- 4:30 p.m. Transcriptional Profiling in ASD: A Systems Biology Approach
Daniel H. Geschwind
- 5:00 p.m. Discussant: *Raquel E. Gur*

PA

3:00 p.m. – 5:30 p.m.

Panel

Regency Ballroom 1

De-risking the Pathway of Treatment Development for Autism Spectrum Disorders

Chair: Linda Brady

Co-Chair: Robert H. Ring

PA

3:00 p.m. Measures of Clinical Meaningful Change, a Summary of the
Recent Meeting on Outcome Measures Consensus Statements for
Clinical Trials in ASD

Evdokia Anagnostou

3:30 p.m. Quantifying Social Deficits in Autism via Eye-Tracking
Measures of Social Engagement

Warren Jones

4:00 p.m. Electrophysiological Signatures of Language Impairment in
Autism Spectrum Disorders - Biomarkers, Neurobiological
Insight and Potential Early Signals of Efficacy:
Magnetoencephalographic (MEG) Investigations

Timothy Roberts

4:30 p.m. Using DSM-5 Criteria to Assess Core Symptoms of Autism
Spectrum Disorders for Diagnosis and Evaluation of Treatment
Outcomes

Susan Swedo

5:00 p.m. Discussant: *Geraldine Dawson*

3:00 p.m. – 5:30 p.m.

Panel

Atlantic Ballroom 3

Links between Activity, Sleep and Mental Function: Translational Models

Chair: Kathleen R. Merikangas

- 3:00 p.m. Objective Assessment of Rhythms and Inter-relationships of Activity, Sleep and Mood in a Community Based Family Study of Affective Spectrum Disorders
Kathleen R. Merikangas
- 3:30 p.m. Links between Anxiety and Activity in Nonhuman Primates
Judy Cameron
- 4:00 p.m. Sleep-wake Cycle, Patterns of Physical Activity and Circadian Rhythm Disruption in Young People with Emerging Mood Disorders
Ian Hickie
- 4:30 p.m. Seasonal Effects on Sleep, Activity and Behavior in Migratory Birds
Ruth Benca
- 5:00 p.m. Discussant: *Joseph S. Takahashi*

PA

3:00 p.m. – 5:30 p.m.

Panel

Atlantic Ballroom 2

Glial Regulation of Synaptic Pathology: Novel Mechanisms of Neuropsychiatric Disease and Avenues for Repair

Chair: Peter Kalivas

PA

- 3:00 p.m. Role of Astrocytes in Synaptic Development
Cagla Eroglu
- 3:30 p.m. Lactate-mediated Coupling between Astrocytes and Neurons Controls Memory Consolidation
Cristina Alberini
- 4:00 p.m. Control of Drug Seeking Behavior by Modulation of Astroglial Glutamate Transport
Kathryn Reissner
- 4:30 p.m. Schizophrenia and Astrocytes: The Importance of System xc – to Preclinical Models of PFC Dysfunction
David A. Baker
- 5:00 p.m. Discussant: *Peter Kalivas*

7:30 p.m. – 9:00 p.m.
Study Group
Atlantic Ballroom 1

**‘If We Thought our Field was in Trouble Before...’
Is Ethical Mental Health Care Possible in the
Second Decade of the 21st Century?**

Chair: Ellen Frank
Co-Chair: John G. Csernansky

Participants:
Ellen Frank
Kenneth L. Davis
Howard H. Goldman
William Z. Potter
Alan F. Schatzberg

SG

7:30 p.m. – 9:00 p.m.
Study Group
Regency Ballroom 1

**Practical, Societal, Ethical, and Legal Challenges for Modern
Brain and Biobanking: Experiences from America and Europe**

Chair: Thomas Schulze
Co-Chair: Francine M. Benes

Participants:
Thomas Schulze
Francine M. Benes
Thomas Insel
Joel E. Kleinman
Camilla Stoltenberg
Peter G. Falkai
Shawn HE. Harmon
Robert H. Ring

7:30 p.m. – 9:00 p.m.

Study Group

Regency Ballroom 2

**The Role of Corticotropin-Releasing Factor (CRF)
in the Pathophysiology of Mood and Anxiety Disorders:
A Tribute to Wylie Vale**

Chair: Charles Nemeroff

Participants:

Florian Holsboer

Tracy Bale

George F. Koob

Dimitri Grigoriadis

Charles Nemeroff

Elizabeth Flandreau

Alon Chen

SG

7:30 p.m. – 9:00 p.m.

Study Group

Atlantic Ballroom 2

**NIMH Research Domain Criteria Project: How will the Criteria
Work for Studies of Diagnosis and New Drug Development?**

Co-Chairs: William Carpenter, Bruce Cuthbert

Participants:

William Carpenter

Bruce Cuthbert

James Waltz

Wayne Drevets

Mark Smith