

ACNP

51st Annual Meeting Final Program

December 2-6, 2012
The Westin Diplomat
Hollywood, Florida

President: John H. Krystal, M.D.
Program Committee Chair: Anissa Abi-Dargham, M.D.
Program Committee Co-Chair: Randy D. Blakely, Ph.D.



This meeting is jointly sponsored by the Vanderbilt University School of
Medicine Department of Psychiatry and the American College of
Neuropsychopharmacology.



Dear ACNP Members and Guests:

It is a tremendous pleasure to welcome you to the 51st Annual Meeting of the ACNP. We have returned to the wonderful venue of the Westin Diplomat in Hollywood, Florida for the first meeting of the second half-century of our College. This implied mix of tradition and innovation is at the core of a new vitality of the ACNP. Each year we celebrate the strong bonds of friendship and collegiality that are a hallmark of our community. At the same time, our College is reaching out to young scientists and “new science” as never before.



Neuropsychopharmacology is transforming and the excitement of revolution is energizing our field. Our Program Committee, under the leadership of Anissa Abi-Dargham and Randy Blakely, has done an exceptional job of capturing this wonderful progress within our Annual Meeting. They also have refined the program innovations of the past several years, including the Hot Topics sessions, the Mini-Panels, and the Data Blitz. This year’s program also reflects a revised submission process that increases opportunities for members, particularly young scientists, to contribute to the scientific program. Once again, the ACNP Women’s Task Force will host a luncheon to enhance the engagement of female attendees in the life of the College.

It has been an honor to serve as President of the ACNP. It has been a privilege and a pleasure to work closely with Ronnie Wilkins, Executive Director; Sarah Timm, Deputy Director; and the wonderful ACNP staff over the past year.

Welcome to Hollywood! Have a wonderful meeting.

A handwritten signature in black ink that reads "John Krystal". The signature is written in a cursive, flowing style.

*John H. Krystal, M.D.
President, 2012*

Annual Meeting Evaluation

CME Credit

All meeting attendees are urged to complete an evaluation.

Attendees who are requesting CME credit for the meeting are required to complete the evaluation. This form is available online only.

To complete the online form while at the meeting, visit the ACNP Computer Center located in the Diplomat 1 & 2 Foyer

Or

You may access the evaluation on the ACNP website (www.acnp.org) under the “Annual Meeting” tab.

There will be a \$40.00 charge for scientific registrants to obtain CME credits. The evaluation must be completed by January 9, 2013 in order to receive CME credit.

ACNP

AMERICAN COLLEGE OF NEUROPSYCHOPHARMACOLOGY

51st

ANNUAL MEETING

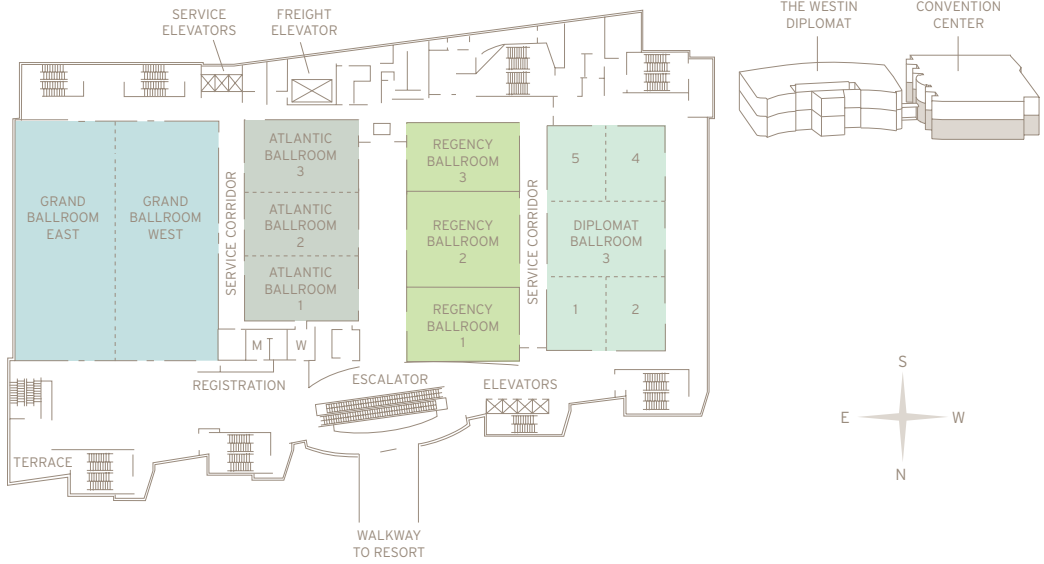
FINAL PROGRAM

HOLLYWOOD, FLORIDA
WESTIN DIPLOMAT RESORT & SPA

DECEMBER 2-6, 2012

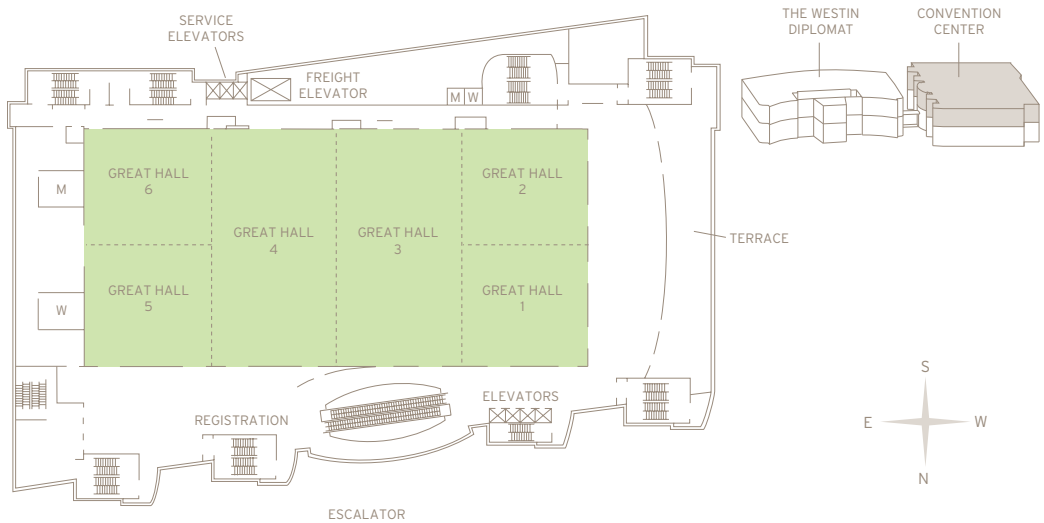
A link to disclosures for 2012 speakers (mini-panels, panel, study group, and plenary) and poster presenters may be found online at: www.acnp.org/annualmeeting/dates.aspx (choose "Abstracts")

2nd Floor Convention Center

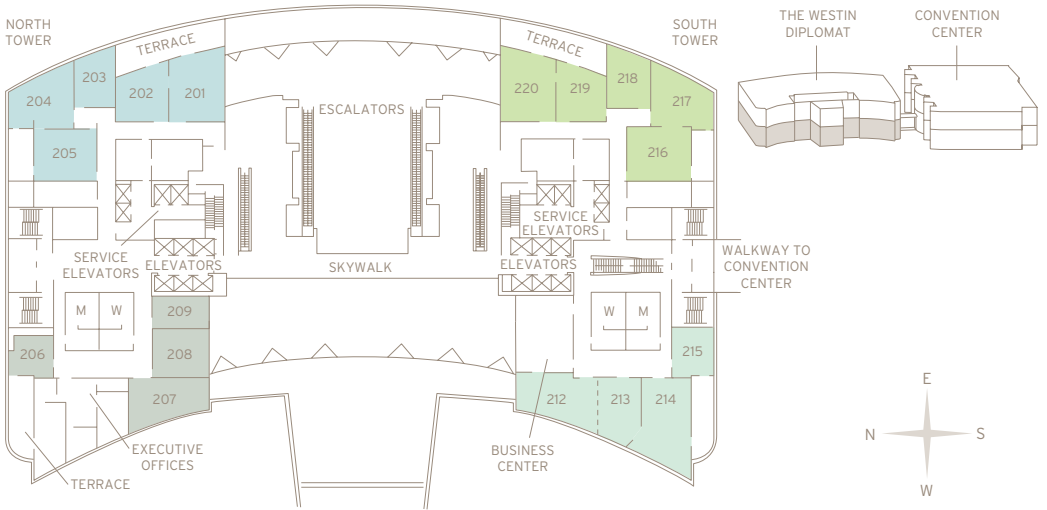


Meeting rooms for panels, mini-panels, plenaries, and study groups are on the 2nd floor of the Convention Center (map above). Poster sessions and group lunches are on the 3rd floor (map below).

3rd Floor Convention Center

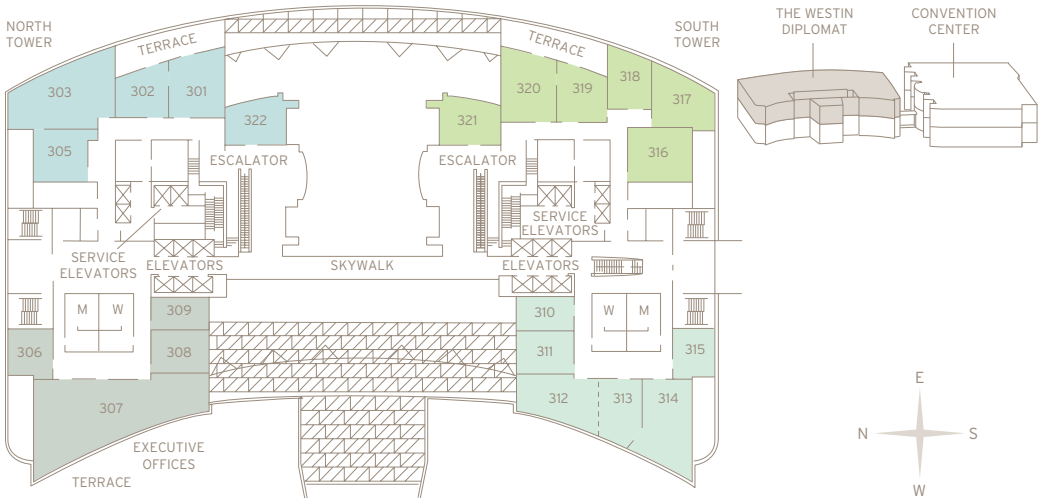


Resort, Second Floor



Conference rooms for committee and board meetings are located on the 2nd and 3rd floor of the hotel. Most small meetings have been scheduled on the hotel side.

Resort, Third Floor



ACNP 51st Annual Meeting • Final Program

Program at a Glance

Saturday, December 1, 2012

8:00 AM – 3:00 PM Room 319 & 320
ACNP Council Meeting

8:00 AM – 5:00 PM Diplomat Ballroom 5
ACNP Membership Committee Meeting

9:30 AM – 12:00 PM Room 307
iSPOT-D Meeting

2:00 PM – 4:00 PM Room 208
Neuropsychopharmacology & Neuropsychopharmacology Reviews
EIC & Deputy Editors Meeting

4:00 PM – 5:30 PM Room 208
ACNP Publications Committee Meeting

4:00 PM – 5:30 PM Room 202
ACNP Ethics Committee Meeting

6:30 PM – 8:30 PM Great Hall 5
ACNP Travel Award Reception (*by invitation only*)

Sunday, December 2, 2012

7:00 AM – 8:30 AM Room 307
ACNP Public Information Committee

8:30 AM – 11:30 AM Regency Ballroom 1 & 2
Neuropsychopharmacology Reviews Plenary:
“Epigenetics”

11:30 AM – 1:00 PM Room 219-220
ACNP Past Presidents’ luncheon

11:30 AM – 1:00 PM Diplomat Ballroom 5
ACNP Program Committee Meeting

11:30 AM – 1:00 PM Diplomat Ballroom 4
ACNP Liaison Committee Meeting

11:30 AM – 1:00 PM Room 205
U19 Scientific Advisory Board Meeting

1:00 PM – 2:30 PM Regency Ballroom 1 & 2
NIH Institutes Directors’ Session

2:30 PM – 6:30 PM Regency Ballroom 1 & 2
Hot Topics

6:30 PM – 7:30 PM Room 201-202
Associate Member Reception (*by invitation only*)

7:00 PM – 9:00 PM Infinity Pool “T” Area
Opening Night Reception

Monday, December 3, 2012

7:00 AM – 8:00 AM Room 207
ACNP Leadership & Institute Directors
Meeting

7:00 AM – 8:00 AM Diplomat Ballroom 4
JAMA Network Meeting

8:00 AM – 11:30 AM Grand Ballroom
President’s Plenary: Taking Stock of the
Connectome

11:30 AM – 1:00 PM Room 312-313
FNH Biomarkers Consortium Neuroscience
Steering Committee

Monday, December 3, 2012

11:30 PM – 1:00 PM Great Hall 5 & 6
Women’s Luncheon

11:30 AM – 1:30 PM Room 201
Consortium on the Genetics of Schizophrenia
(COGS)

1:30 PM – 3:00 PM Grand Ballroom
Distinguished Lecture – Jeffrey Friedman
Leptin and the Biologic Basis of Obesity

Mini-Panel Sessions

3:00 PM – 4:15 PM Diplomat Ballroom 1 & 2
Renaissance in Opioid Biology: From
Preclinical Concepts to Clinical Practice

4:15 PM – 5:30 PM Diplomat Ballroom 1 & 2
Interaction of Ontogeny and Environment in
Adolescent Substance Abuse

Panel Sessions

3:00 PM – 5:30 PM Regency Ballroom 2
Neuroplasticity Deficits in Neuropsychiatric
Illness: New Targets for Cognitive
Enhancement

3:00 PM – 5:30 PM Atlantic Ballroom 1
Common Neural Mechanisms across
Dimensions of Pediatric Psychopathology

3:00 PM – 5:30 PM Regency Ballroom 3
Unraveling the Genetic Architecture of Mental
Illness with Whole Genome Sequence Data

3:00 PM – 5:30 PM Regency Ballroom 1
De-risking the Pathway of Treatment
Development for Autism Spectrum Disorders

3:00 PM – 5:30 PM Atlantic Ballroom 3
Links between Activity, Sleep and Mental
Function: Translational Models

3:00 PM – 5:30 PM Atlantic Ballroom 2
Glial Regulation of Synaptic Pathology: Novel
Mechanisms of Neuropsychiatric Disease
and Avenues for Repair

5:30 PM – 7:30 PM Great Hall 1-4
Poster Session I with Reception

Study Groups

7:30 PM – 9:00 PM 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?

7:30 PM – 9:00 PM Regency Ballroom 1
Practical, Societal, Ethical, and Legal
Challenges for Modern Brain and Biobanking:
Experiences from America and Europe

7:30 PM – 9:00 PM Regency Ballroom 2
The Role of Corticotropin-Releasing Factor
(CRF) in the Pathophysiology of Mood and
Anxiety Disorders: A Tribute to Wylie Vale

7:30 PM – 9:00 PM Atlantic Ballroom 2
NIMH Research Domain Criteria Project: How
will the Criteria Work for Studies of Diagnosis
and New Drug Development?

Tuesday, December 4, 2012

7:00 AM – 8:30 AM Diplomat Ballroom 4
ACNP Education & Training Committee
Meeting

7:00 AM – 8:30 AM Room 203
ACNP Membership Advisory Task Force
Meeting

7:00 AM – 8:30 AM Room 220
American Journal of Psychiatry Editorial
Board Meeting

7:00 AM – 8:30 AM Room 207
CME Institute Executive Director’s Meeting

7:30 AM – 8:30 AM Room 202
SOBP Biological Psychiatry Journal Editors’
Meeting

Panel Sessions

8:30 AM – 11:00 AM Regency Ballroom 2
Neuroscience and the Future of Psychiatric
Diagnosis: Updates on Development of the
Fifth Edition of Diagnostic and Statistical
Manual of Mental Disorders

8:30 AM – 11:00 AM Regency Ballroom 3
Developmental Programming of the Brain:
Implications for Shared Mechanisms Across
Neuropsychiatric Disorders

8:30 AM – 11:00 AM Regency Ballroom 1
One Size Doesn’t Fit All: Molecular
Mechanisms Underlying Diverse Estradiol
Signaling in the Brain

8:30 AM – 11:00 AM Diplomat Ballroom 1 & 2
Optimizing Cognitive Interventions for
Schizophrenia: Predictive Biomarkers and
Pharmacologic Enhancement

8:30 AM – 11:00 AM Atlantic Ballroom 2
The Developmental Trajectory of Cannabis
Effects on Neurobiological Functioning

8:30 AM – 11:00 AM Atlantic Ballroom 3
Multi-level Classification of Schizophrenia
and Bipolar Disorder: New Evidence and
Controversies

8:30 AM – 11:00 AM Atlantic Ballroom 1
Immune Modulation of Neurodevelopment in
Schizophrenia and Autism

11:00 AM – 12:30 PM Room 220
U19 Committee Meeting: Emory-MSSM-
Baylor-SFVA

11:00 AM – 12:30 PM Diplomat Ballroom 4
Corporate Liaison Luncheon
(*by invitation only*)

11:15 AM – 1:30 PM Regency Ballroom 2
Data Blitz Session

12:00 PM – 1:00 PM Room 209
ACNP Website Editors Meeting

1:30 PM – 3:00 PM Regency Ballroom 1
Associate Member Session: “Ask the Experts:
Peer Review”

Program at a Glance

Tuesday, December 4, 2012

Mini-Panel Sessions

3:00 PM – 4:15 PM Diplomat Ballroom 1 & 2
Rescuing Novel Mechanisms: Minimizing Placebo Response and Optimizing Signal Detection in Proof of Concept Trials

4:15 PM – 5:30 PM Diplomat Ballroom 1 & 2
Exploring Therapeutic Use of Psilocybin, A Classic Hallucinogen

Panel Sessions

3:00 PM – 5:30 PM Regency Ballroom 1
New Perspectives on the Role of Glutamatergic Neurotransmission in Alcoholism and Drug Addiction

3:00 PM – 5:30 PM Atlantic Ballroom 2
High Anxiety: Endocannabinoid Regulation of the Stress Response and Emotional Behavior

3:00 PM – 5:30 PM Regency Ballroom 3
Longitudinal Neuroimaging of Emerging Substance Use: Brain Indicators of Early Risk and Effects of Use

3:00 PM – 5:30 PM Regency Ballroom 2
Neuropeptide Receptor Ligands in Psychiatric Diseases: New Hopes after Multiple Failures

3:00 PM – 5:30 PM Atlantic Ballroom 1
Are We at a Turning Point in Psychiatric Genetics?

3:00 PM – 5:30 PM Atlantic Ballroom 3
The Ups and Downs of AKT Signaling: A Nexus of Risk for Psychiatric Disorders

5:30 PM – 7:30 PM Great Hall 1-4
Poster Session II with Reception

6:00 PM – 11:00 PM Room 318
ACNP Committee Chairs Waiting Room

6:00 PM – 11:00 PM Room 319-320
ACNP Council Meeting

Wednesday, December 5, 2012

7:00 AM – 8:30 AM Room 201
ASCP Board of Director's Meeting

Mini-Panel Sessions

8:30 AM – 9:45 AM Diplomat Ballroom 1 & 2
Behavioral Paradigms to Improve Signal Detection in Trials of Cognition Enhancing Drugs

9:45 AM – 11:00 AM Diplomat Ballroom 1 & 2
Pathology Driven Biomarker Development for Major Depressive Disorder: Bridging Central to Peripheral Markers

Panel Sessions

8:30 AM – 11:00 AM Regency Ballroom 2
Molecular and Cellular Mechanisms Underlying Resilience in Mood and Other Social-psychological Stress-related Disorders: New Avenue for Novel Therapeutics?

8:30 AM – 11:00 AM Regency Ballroom 1
Reward/Motivation Deficits in Attention Deficit Hyperactivity Disorder (ADHD) and the Effects of Medication

Wednesday, December 5, 2012

8:30 AM – 11:00 AM Regency Ballroom 3
Opioid and Cannabinoid Mechanisms in Alcohol Addiction: Recent Evidence from Functional Brain Imaging

8:30 AM – 11:00 AM Atlantic Ballroom 2
Beta-amyloid Neuropathology in Cognitively Normal Individuals: Preclinical Alzheimer's Disease or Cognitive Resilience?

8:30 AM – 11:00 AM Atlantic Ballroom 3
Inhibition of Phosphodiesterases to Treat Psychiatric Disorders: Advances through Innovation in Preclinical Models and Feedback from the Clinic

8:30 AM – 11:00 AM Atlantic Ballroom 1
Dendritic Spine Plasticity in Depression and Addiction

11:15 AM – 12:30 PM Regency Ballroom 2
ACNP Business Meeting (*ACNP Fellows, Members, and Associate Members Only*)

12:30 PM – 2:00 PM Diplomat Ballroom 4
SOBP Program Committee Meeting

12:30 PM – 2:00 PM Great Hall 5
Travel Awardee Luncheon (*by Invitation Only*)

Panel Sessions

3:00 PM – 5:30 PM Regency Ballroom 1
Circadian Rhythms and Mood Disorders: Clock Genes and New Treatment Implications

3:00 PM – 5:30 PM Atlantic Ballroom 1
Neuronal Circuit Regulation of Ventral Tegmental Area Neurons

3:00 PM – 5:30 PM Regency Ballroom 3
Applying Translational Research and Imaging to Treatment Strategies in Alcoholism

3:00 PM – 5:30 PM Atlantic Ballroom 2
Lesson from Animal Studies of Genetic Risk Factors for Psychiatric Disorders of Neurodevelopmental origin: How can we Move Forward with our Research for Novel Treatment Interventions?

3:00 PM – 5:30 PM Atlantic Ballroom 3
Glucocorticoid Receptors as Pharmacologic Targets in Psychiatry

3:00 PM – 5:30 PM Diplomat Ballroom 1 & 2
Army STARRS Suicide Research: From Bench to Battlefield

3:00 AM – 5:30 PM Regency Ballroom 2
Anxiety Disorders: New Evidence for Structural and Functional Connectivity Abnormalities

5:30 PM – 7:30 PM Great Hall 1-4
Poster Session III with Reception

7:30 PM – 9:00 PM Diplomat Ballroom 4
Neuropsychopharmacology Editorial Board

Thursday, December 6, 2012

7:00 AM – 8:00 AM Room 212
ACNP/AsCNP/CINP/ECNP/JSNP Meeting

Thursday, December 6, 2012

Mini Panel Sessions

8:00 AM – 9:15 AM Diplomat Ballroom 1 & 2
Beyond Ketamine, Can Selective Targeting of the NMDA Receptor Produce Antidepressant Response without Psychotomimetic Effects: Clinical Results with Three Novel Compounds

9:15 AM – 10:30 AM Diplomat Ballroom 1 & 2
Beyond the NMDA Receptor-Alternative Glutamatergic Targets for Antidepressant Treatment

Panel Sessions

8:00 AM – 10:30 AM Regency Ballroom 3
Hippocampus and Addiction: New Neurons, New Circuits, and New Responses

8:00 AM – 10:30 AM Regency Ballroom 2
Neural Networks across Development in Health, Anxiety/Depression, and Treatment Implications

8:00 AM – 10:30 AM Regency Ballroom 1
Harnessing Cortical Plasticity for Therapeutic Purposes

8:00 AM – 10:30 AM Atlantic Ballroom 3
Balancing Benefits, Risks and Cost for New Treatments in Vulnerable Populations: Lessons from Child Psychiatry on the Need for a New Standard of Diverse Methodologies

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Metabotropic Glutamate Receptors (mGluRs) and Addiction

8:00 AM – 10:30 AM Atlantic Ballroom 1
Affective Neuroscience of Young Monkeys to Developing Population: Translational Studies of Brain Function Informing Interventions

9:00 AM – 12:00 PM Room 319-320
ACNP Council Meeting

Panel Sessions

12:00 PM – 2:30 PM Diplomat Ballroom 1 & 2
Functional and Structural Alterations in the Insula are Central to the Pathophysiology of Both Anorexia Nervosa and Obesity

12:00 PM – 2:30 PM Atlantic Ballroom 2
Neuroimaging Predictors of Treatment Effects in High-risk and Bipolar Individuals across the Lifespan

12:00 PM – 2:30 PM Atlantic Ballroom 3
Sink or Swim: Take Your Raft and Fyns Down the STEPs to Navigate NMDA Receptor Pools in Neuropsychiatric Disorders

12:00 PM – 2:30 PM Regency Ballroom 2
Non-invasive Brain Modulation to Enhance Inhibitory Control and Drive in Psychiatric Disorders: a Translational Approach with a Focus on Dopaminergic Modulation of Fronto-striatal Pathways

12:00 PM – 2:30 PM Atlantic Ballroom 1
Neuronal Mechanisms for Behavioral and Psychiatric Vulnerability in Adolescents

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12:00 PM – 2:30 PM Regency Ballroom 1
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Acknowledgments

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Council

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Program Committee

2012 Program and Scientific Communications Committee

<i>Chair</i>	Anissa Abi-Dargham
<i>Co-Chair</i>	Randy Blakely
<i>Council Liaison</i>	David A. Lewis

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Paul Kenny	Etienne Sibille	
Henry Kranzler		

General Information

Dates and Location

Dates Sunday, December 2, 2012 - Thursday, December 6, 2012
Location The Westin Diplomat, Hollywood, Florida

Program Book

All scientific registrants will receive a Program Book as part of their registration material. The Program Book is also available on the ACNP website, www.acnp.org.

Itinerary Planner


All scientific registrants will be able to access the itinerary planner for the 51st ACNP Annual Meeting at www.EventScribe.com/2012/ACNP.

ACNP Executive Office

ACNP Executive Office
5034A Thoroughbred Lane
Brentwood, Tennessee 37027 USA

Phone: 615-324-2360
Fax: 615-523-1715
E-mail: acnp@acnp.org

Continuing Medical Education

 The 2012 ACNP Annual Meeting is jointly sponsored by the Vanderbilt University School of Medicine and the ACNP. This activity has been planned and implemented in accordance with the Essentials Areas and Policies of the Accreditation Council for CME (ACCME) through the joint sponsorship of Vanderbilt University School of Medicine and the ACNP.

Vanderbilt University School of Medicine is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education for physicians.

Vanderbilt University School of Medicine designates this live activity for a maximum of 36.25 *AMA PRA Category 1 Credit(s)*[™]. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

There will be a \$40.00 charge for scientific registrants to obtain CME credits. CME instructions will be available at the meeting registration desk and on the ACNP website (www.acnp.org).

It is the policy of Vanderbilt University School of Medicine to require disclosure of financial relationships from individuals in a position to control the content of a CME activity; to identify and resolve conflicts of interest related to those relationships; and to make disclosure information available to the audience prior to the CME activity. Presenters are required to disclose discussions of unlabeled/unapproved uses of drugs or devices during their presentations.

Program Overview/Statement of Need

The Annual Meeting of the American College of Neuropsychopharmacology is designed to meet the educational needs of ACNP members and invited non-member colleagues. Current data suggests that in any given year more than 20% of the U.S. adult population suffers from a diagnosable mental disorder. Four of the ten leading causes of disability in the U.S. are psychiatric disorders, including schizophrenia, depression, bipolar disorder, and obsessive-compulsive disorder. ACNP members have been among the leaders in identifying underlying mechanisms for these disorders and developing new treatment strategies. The desired results for the meeting are that ACNP members and their invited guests learn of the latest developments in preclinical and clinical research being performed by their colleagues and world experts in order to 1) enhance understanding of the neurobiological bases of current best practice approaches, 2) enhance understanding of neurobiological and clinical science underpinnings in development of novel therapeutic strategies, particularly for treatment-resistant forms of illness, and 3) lead to improvements in study designs for proposed clinical and basic studies.

Continuing Medical Education (continued)

Target Audience

The target audience includes members of the American College of Neuropsychopharmacology and invited experts. The audience includes physicians, psychologists, and basic neuroscientists from across the United States as well as Europe and Asia. The physicians include a number of specialties, with psychiatrists representing the majority of attendees, and neurologists next most common. Psychologists include clinical psychologists and neuropsychologists.

Learning Objectives:

After participating in this CME activity, participants should be able to:

- Describe and discuss how the results of recent or ongoing basic science and/or clinical studies of psychiatric disorders in your area of interest or a related area impact your current or potential future research projects.
- Describe and discuss how you will change or modify a current approach or strategy in your current or potential future research projects based on what you learned from the results of recent or ongoing basic science and/or clinical studies of psychiatric disorders in your area of interest or a related area.
- Describe and discuss how recent progress in identifying genetic variations that are risk factors for the development of psychiatric disorders affect your current or potential future research projects.

Americans with Disabilities Act

It is the policy of Vanderbilt University School of Medicine not to discriminate against any person on the basis of disabilities. If you feel you need services or auxiliary aids mentioned in this act in order to fully participate in this continuing education activity, please call the Executive Office at 615-324-2360 or send an email to acnp@acnp.org.

Meeting Evaluation

All meeting attendees are urged to complete an evaluation of the meeting. Attendees who are requesting CME credit for the meeting **are required** to complete the evaluation. This form is available online only. You may complete the evaluation in the ACNP Computer Center located in Diplomat 1 & 2 foyer or on-line at www.acnp.org (click the Annual Meeting tab). All evaluations must be completed by January 9, 2012.

Scientific Program

VIDEOTAPING SESSIONS

Attendees may not videotape, audiotape, or photograph (camera or camera phone) presentations at the Annual Meeting without prior permission from the panel chair.

PLENARY SESSIONS

NEUROPSYCHOPHARMACOLOGY REVIEWS PLENARY This year's title is "Epigenetics" and will be chaired by Eric Nestler and Schahram Akbarian. The speakers include David Sweatt, Marcelo Wood, Lisa Monteggia, Anne Schaefer, and Johannes Bohacek.

Sunday, December 2nd

8:30 a.m. - 11:30 a.m.

This session is located in Regency Ballroom 1 & 2

NIH INSTITUTES DIRECTORS' SESSION Join NIA, NIAAA, NIDA, and NIMH directors for this interactive question & answer panel session where the directors will be responding to audience generated questions related to the status of funding and future plans of the institutes.

Sunday, December 2nd

1:00 p.m. - 2:30 p.m.

Located in the Regency Ballroom 1 & 2

HOT TOPICS Each presentation will last approximately ten minutes with a five-minute period for questions and open discussion. Basic/Translational and Clinical topics have been integrated into one session.

Sunday, December 2nd

2:30 p.m. - 6:30 p.m.

Located in the Regency Ballroom 1 & 2

PRESIDENT’S PLENARY This year’s President’s Plenary, chaired by ACNP President, John Krystal, is entitled, “**Taking Stock of the Connectome.**”

Monday, December 3rd 8:00 a.m. - 11:30 a.m.

Located in the Grand Ballroom

DISTINGUISHED LECTURE This plenary is chaired by John Krystal. The lecture will be presented by Jeffrey Friedman. The title of his talk is, “**Leptin and the Biologic Basis of Obesity.**”

Monday, December 3rd 1:30 p.m. - 3:00 p.m.

Located in the Grand Ballroom

DATA BLITZ SESSION This session is comprised of rigorously timed 5-min presentations by 17 early career investigators that are linked to poster presentations.

Tuesday, December 4th 11:15 a.m. – 1:30 p.m.

Located in Regency Ballroom 2

SPECIAL SESSION Associate Member Session: “Ask the Experts”

Tuesday, December 4th 1:30 p.m. – 3:00 p.m.

Located in Regency Ballroom 1

CONCURRENT SESSIONS

PANELS The title and location of each panel session is indicated in the program. Presentations in each session are scheduled at approximately 30-minute intervals, allowing for 20-minute presentations and 10-minute discussion periods. A thirty-minute general discussion period is scheduled after the last presenter in each session. **Please note: Thursday panel sessions have adjusted start/end times.** Panel sessions are scheduled as follows:

Monday, December 3rd	3:00 p.m. - 5:30 p.m.
Tuesday, December 4th	8:30 a.m. - 11:00 a.m. 3:00 p.m. - 5:30 p.m.
Wednesday, December 5th	8:30 a.m. - 11:00 a.m. 3:00 p.m. - 5:30 p.m.
Thursday, December 6th	8:00 a.m. - 10:30 a.m. 12:00 p.m. - 2:30 p.m.

MINI PANELS The title and location of each mini-panel session is indicated in the program. A mini-panel is a moderately formal 75-minute session that includes 1 chair and 3 presenters. Each of the 3 presentations lasts 25 minutes which allows for a 20-minute presentation and 5-minute discussion period. Mini-panel sessions are scheduled as follows:

Monday, December 3rd	8:30 a.m. – 9:45 a.m. 9:45 a.m. – 11:00 a.m.
Tuesday, December 4th	3:00 p.m. – 4:15 p.m. 4:15 p.m. - 5:30 p.m.
Wednesday, December 5th	8:30 a.m. – 9:45 a.m. 9:45 a.m. – 11:00 a.m.
Thursday, December 6th	8:00 a.m. – 9:15 a.m. 9:15 a.m. - 10:30 a.m.

STUDY GROUPS Study groups will be issue oriented and are scheduled for Monday evening. The title and location of each study group is indicated in the program.

Monday, December 5th	7:30 p.m. - 9:00 p.m.
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POSTER SESSIONS

POSTERS Poster presentations are grouped by general topic area each evening and may be repeated as dictated by submissions.

Monday, December 3rd	5:30 p.m. - 7:30 p.m. Poster Session I Located in Great Hall 1-4
Tuesday, December 4th	5:30 p.m. - 7:30 p.m. Poster Session II Located in Great Hall 1-4
Wednesday, December 5th	5:30 p.m. - 7:30 p.m. Poster Session III Located in Great Hall 1-4

Registration

ON-SITE REGISTRATION PERIODS

Date	Time	Location
Saturday, December 1st	9:00 a.m. - 7:00 p.m.	Grand Ballroom Lobby (level 2)
Sunday, December 2nd	7:00 a.m. - 7:00 p.m.	Grand Ballroom Lobby (level 2)
Monday, December 3rd	7:00 a.m. - 6:00 p.m.	Grand Ballroom Lobby (level 2)
Tuesday, December 4th	7:00 a.m. - 6:00 p.m.	Grand Ballroom Lobby (level 2)
Wednesday, December 5th	7:30 a.m. - 5:30 p.m.	Grand Ballroom Lobby (level 2)
Thursday, December 6th	8:00 a.m. - 3:00 p.m.	Grand Ballroom Lobby (level 2)

ON-SITE REGISTRATION FEES

Categories	On-Site Fee
ACNP Associate Members	\$250
ACNP Members & Fellows	\$600
ACNP Emeritus members	\$250
Members of AsCNP, CCNP, CINP, ECNP, JSNP (on list)	\$600
Corporate Representatives	\$600
Non-Member Program Participant	Waived
2012 Travel Awardees	Waived
Trainee*	\$250
Invited Guest	\$850
Past Travel Awardee (2008-2011)	\$250
Optional Journal Subscription for non-ACNP members**	\$150
Accompanying Person***	\$200

*A Trainee is a person in a training position and not a full-time, permanent position. A Trainee can be a M.D., Ph.D., Postdoctoral Fellow, Resident, or Research Fellow. Trainees have all the rights and privileges of Invited Guests.

**Non-ACNP members may purchase a personal online subscription to *Neuropsychopharmacology* at the significantly discounted rate of \$150 (USD), a savings of over 65% off the regular personal subscription rate. The subscription includes full online access from January through December 2013 for all 12 issues printed in 2013 and also *Neuropsychopharmacology Reviews*.

***An accompanying person is a spouse, relative, or significant other who accompanies an attendee. An accompanying person may only attend the social functions of the meeting, which includes the morning refreshments, lunch buffets, and the opening welcome reception. Accompanying persons are also invited to attend the evening poster sessions.

Hotel Facilities

The Westin Diplomat guest rooms, common areas, and transportation services are in compliance with the public accommodation requirements of the Americans with Disabilities Act. These facilities will be accessible to and usable by individuals with disabilities who attend and participate in the Annual Meeting.

The Westin Diplomat is a smoke-free facility.

Services

ACNP Computer Center

The ACNP Computer Center is provided for the convenience of meeting attendees. Computers will be available in the Diplomat 1 & 2 Foyer for the following uses: e-mail, internet access, and annual meeting evaluation.

Sunday through Wednesday	7:30 a.m. - 9:00 p.m.
Thursday	7:30 a.m. - 2:00 p.m.

Speaker Ready Room

A speaker ready room will be available in Diplomat 3. The speaker ready room will be open Sunday - Wednesday, 7:30 a.m. - 6:30 p.m.; Thursday, 7:00 a.m. - 2:30 p.m.

Travel Information Desk

The Travel Information Desk is located in the Grand Ballroom Foyer (level 2). An ACNP staff member will be available to assist participants with lodging, travel, or car rental information at this desk.

Refreshment Breaks

All registered individuals are invited.

Coffee and light pastries for registered individuals will be available Sunday - Wednesday at 7:30 a.m.; Thursday 7:00 a.m. All breaks are in the Grand Ballroom Lobby.

Luncheons

All registered individuals are invited.

Date: Monday, December 3rd

Time: 11:30 a.m. - 1:30 p.m.

Place: Great Hall 1-4

Date: Tuesday, December 4th

Time: 11:00 a.m. - 12:30 p.m.

Place: Great Hall 1-4

Date: Wednesday, December 5th

Time: 12:30 p.m. - 2:00 p.m.

Place: Great Hall 1-4

Date: Thursday, December 6th

Time: 10:30 a.m. - 12:00 p.m.

Place: Grand Ballroom Lobby

Opening Reception

All registered individuals are invited.

Date: Sunday, December 2nd

Time: 7:00 p.m. - 9:00 p.m.

Place: Infinity Pool "T" Area

Special Events

Saturday, December 1, 2012

Travel Award Reception

(by invitation only)

Time: 6:30 p.m. – 8:30 p.m.

Place: Great Hall 5

Sunday, December 2, 2012

Past Presidents' Luncheon

(by invitation only)

Time: 11:30 a.m. – 1:00 p.m.

Place: Room 219-220

Associate Member Reception

(by invitation only)

Time: 6:30 p.m. – 7:30 p.m.

Place: Room 201-202

Monday, December 3, 2012

Women's Luncheon

Time: 11:30 a.m. – 1:00 p.m.

Place: Great Hall 5 & 6

Wednesday, December 5, 2012

Travel Award Luncheon

(by invitation only)

Time: 12:30 p.m. – 2:00 p.m.

Place: Great Hall 5

College Meetings

Saturday, December 1, 2012

Neuropsychopharmacology & Neuropsychopharmacology Reviews

Editors & Deputy Editors

Time: 2:00 p.m. – 4:00 p.m.

Place: Room 208

Monday, December 3, 2012

ACNP Leadership & NIH Directors

Time: 7:00 a.m. – 8:00 a.m.

Place: Room 207

Tuesday, December 4, 2012

ACNP Website Editors

Time: 12:00 p.m. – 1:00 p.m.

Place: Room 209

Corporate Liaison Luncheon

(by invitation only)

Time: 11:00 a.m. - 12:30 p.m.

Place: Diplomat Ballroom 4

Wednesday, December 5, 2012

Annual Business Meeting

(ACNP Members and Associate Members only)

Chair: John Krystal

Time: 11:15 a.m. - 12:30 p.m.

Place: Regency Ballroom 2

***Neuropsychopharmacology* Editorial Board**

Time: 7:30 p.m. – 9:00 p.m.

Place: Diplomat Ballroom 4

Thursday, December 6, 2012

ACNP/AsCNP/CINP/ECNP/ JSNP Officers

Time: 7:00 a.m. – 8:00 a.m.

Place: Room 212

Council Meetings

Council is involved in many activities during the Annual Meeting. The official Council meetings are listed below:

Saturday, December 1, 2012

ACNP Council

Time: 8:00 a.m. - 3:00 p.m.

Place: Room 319-320

Tuesday, December 4, 2012

ACNP Council

Time: 6:00 p.m. - 11:00 p.m.

Place: Room 319-320

Thursday, December 6, 2012

ACNP Council

Time: 9:00 a.m. - 12:00 p.m.

Place: Room 319-320

Committee/Task Force Information

Meetings listed below are as scheduled by the Committee Chairs.

Saturday, December 1, 2012

Membership Committee

Time: 8:00 a.m. - 5:00 p.m.

Place: Diplomat Ballroom 5

Ethics Committee

Time: 4:00 p.m. – 5:30 p.m.

Place: Room 202

Publications Committee

Time: 4:00 p.m. - 5:30 p.m.

Place: Room 208

Sunday, December 2, 2012

Public Information Committee

Time: 7:00 a.m. – 8:30 a.m.

Place: Room 307

Liaison Committee

Time: 11:30 a.m. – 1:00 p.m.

Place: Diplomat Ballroom 4

Program Committee

Time: 11:30 a.m. – 1:00 p.m.

Place: Diplomat Ballroom 5

Tuesday, December 4, 2012

Education & Training Committee

Time: 7:00 a.m. – 8:30 a.m.

Place: Diplomat Ballroom 4

Member Advisory Task Force

Time: 7:00 a.m. – 8:30 a.m.

Place: Room 203

ACNP Sanctioned Meetings

American Journal of Psychiatry Editorial Board Meeting

Date: Tuesday, December 4, 2012
Time: 7:00 a.m. – 8:30 a.m.
Place: Room 220

ASCP Board of Directors Meeting

Date: Wednesday, December 5, 2012
Time: 7:00 a.m. – 8:30 a.m.
Place: Room 201

CDI Booster Sessions

Date: Monday, December 3, 2012
Time: 6:45 a.m. – 8:00 a.m.
Place: Room 220

Date: Wednesday, December 5, 2012
Time: 7:00 a.m. – 8:30 a.m.
Place: Room 220

Consortium on the Genetics of Schizophrenia (COGS)

Date: Monday, December 3, 2012
Time: 11:30 a.m. – 1:30 p.m.
Place: Room 201

CME Institute Executive Director Meeting

Date: Tuesday, December 4, 2012
Time: 7:00 a.m. – 8:30 a.m.
Place: Room 207

FNIH Biomarkers Consortium Neuroscience Steering Committee

Date: Monday, December 3, 2012
Time: 11:30 a.m. – 1:00 p.m.
Place: Room 312-313

iSPOT-D Meeting

Date: Saturday, December 1, 2012
Time: 9:30 a.m. – 12:00 p.m.
Place: Room 307

JAMA Network

Date: Monday, December 3, 2012
Time: 7:00 a.m. – 8:00 a.m.
Place: Diplomat Ballroom 4

SOBP (Society of Biological Psychiatry)

Council Meeting

Date: Friday, November 30, 2012
Time: 11:00 a.m. – 5:00 p.m.
Place: Room 202

Journal Editors' Meeting

Date: Tuesday, December 4, 2012
Time: 7:30 a.m. - 8:30 a.m.
Place: Room 202

Program Committee Meeting

Date: Wednesday, December 5, 2012
Time: 12:30 p.m. – 2:00 p.m.
Place: Diplomat Ballroom 4

U19 Scientific Advisory Board Meeting

Date: Sunday, December 2, 2012
Time: 11:30 a.m. – 1:00 p.m.
Place: Room 205

U19 Committee Meeting: Emory-MSSM-Baylor-SFVA

Date: Tuesday, December 4, 2012
Time: 11:00 a.m. – 12:30 p.m.
Place: Room 220

Future ACNP Annual Meetings

<i>Dates</i>	<i>Hotel</i>	<i>Location</i>
December 8 - 12, 2013	The Westin Diplomat	Hollywood, Florida
December 7 - 11, 2014	Marriott Desert Ridge Resort	Phoenix, Arizona
December 6 - 10, 2015	The Westin Diplomat	Hollywood, Florida
December 4 - 8, 2016	The Westin Diplomat	Hollywood, Florida
December 3 - 7, 2017	JW Marriott Desert Springs Resort	Palm Springs, California

In Memoriam

Edward G. Jones
June 6, 2011

Wylie Vale
January 3, 2012

Arthur Yuwiler
February 17, 2102

Andrew C. Leon
February 18, 2012

Samuel C. Kaim
March 24, 2012

Toni S. Shippenberg
June 25, 2012

John Cymerman Craig
September 26, 2012

Claude de Montigny
October 19, 2012

Sunday At A Glance

7:00 AM – 8:30 AM	ACNP Public Information Committee	Room 307
7:30 AM	Morning Break	Grand Ballroom Lobby
8:30 AM – 11:30 AM	<i>Neuropsychopharmacology Reviews</i> Plenary: “Epigenetics”	Regency Ballroom 1 & 2
11:30 AM – 1:00 PM	Lunch on Own (<i>no group lunch served</i>)	
11:30 AM – 1:00 PM	ACNP Past Presidents’ luncheon	Room 219-220
11:30 AM – 1:00 PM	ACNP Program Committee Meeting	Diplomat Ballroom 5
11:30 AM – 1:00 PM	ACNP Liaison Committee Meeting	Diplomat Ballroom 4
11:30 AM – 1:00 PM	U19 Scientific Advisory Board Meeting	Room 205
1:00 PM – 2:30 PM	NIH Institutes Directors’ Session	Regency Ballroom 1 & 2
2:30 PM – 6:30 PM	Hot Topics	Regency Ballroom 1 & 2
6:30 PM – 7:30 PM	Associate Member Reception (<i>by invitation only</i>)	Room 201-202
7:00 PM – 9:00 PM	Opening Night Reception	Infinity Pool “T” Area

Sunday

PA-Panel MP-Mini Panel PL-Plenary SG-Study Group

Sunday At A Glance

7:00 AM – 8:30 AM	ACNP Public Information Committee	Room 307
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Sunday

PA-Panel MP-Mini Panel PL-Plenary SG-Study Group

Neuropsychopharmacology Reviews Plenary

“Epigenetics”

Co-Chairs: Eric Nestler and Schahram Akbarian

- 8:30 a.m. Epigenetics Mechanisms in Memory Regulations
David Sweatt
- 8:55 a.m. The Role of Histone Deacetylase 3 (HDAC3) in the Acquisition and Extinction of Cocaine-context Associated Memories
Marcelo Wood
- 9:20 a.m. The Impact of MeCP2 Loss- or Gain-of-Function on Synaptic Plasticity
Lisa Monteggia
- 9:45 a.m. microRNA miR-128 Controls Dopamine-mediated Locomotor Behavior and Prevents Fatal Epilepsy-like Disease in Mice
Anne Schaefer
- 10:10 a.m. Epigenetics of Complex Behaviors and Their Inheritance in Mammals
Johannes Bohacek
- 10:35 a.m. Discussion
Eric Nestler and Schahram Akbarian

8:30 a.m – 11:30 a.m.

Neuropsychopharmacology Reviews Plenary
Regency Ballroom 1 & 2

PL

Epigenetics Mechanisms in Memory Regulations

David Sweatt

University of Alabama at Birmingham Medical School

Epigenetic mechanisms typically involve alterations in chromatin structure, which in turn regulate gene expression. “Epigenetics” is functionally equivalent to the mechanisms allowing stable maintenance of gene expression that involve physically “marking” DNA or its associated proteins through post-translational modification. Thus, regulation of chromatin structure and regulation of direct methylation of DNA are the principal mechanisms of epigenetic regulation. This presentation will address the idea that conservation of epigenetic mechanisms for information storage represents a unifying model in biology, with epigenetic mechanisms being utilized for cellular memory at levels from behavioral memory to development to cellular differentiation. Do epigenetic mechanisms operate in behavioral memory formation? We have generated several lines of evidence that support this idea that I will discuss. 1. Contextual fear conditioning triggers alterations in hippocampal DNA methylation and histone post-translational modifications. 2. Inhibitors of DNA methylation block both hippocampal LTP and associative learning in vivo. 3. Remote contextual fear memory is associated with persisting changes in DNA methylation in the Anterior Cingulate Cortex, and DNMT inhibition can reverse established remote memory. 4. Histone acetylation increases in memory formation, and histone deacetylase (HDAC) inhibitors enhance both memory formation and hippocampal long-term potentiation.

David Sweatt obtained his B.S. in Chemistry before attending Vanderbilt University, where he was awarded a Ph.D. for studies of intracellular signaling mechanisms. He then did a post-doctoral Fellowship at the Columbia, working on memory mechanisms in the laboratory of Nobel laureate Eric Kandel. From 1989 to 2006 he was a member of the Neuroscience faculty at Baylor College of Medicine in Houston, Texas. He is currently the Evelyn F. McKnight endowed Chairman of the Department of Neurobiology at UAB Medical School. Dr. Sweatt’s laboratory studies biochemical mechanisms of learning and memory. In addition, his research program also investigates mechanisms of learning and memory disorders, such as mental retardation and aging-related memory

Epigenetics Mechanisms in Memory Regulations (continued)

David Sweatt

dysfunction. Dr. Sweatt has won numerous awards and honors, including an Ellison Medical Foundation Senior Scholar Award, and election as a Fellow of the American Association for the Advancement of Science. This year he won the Ipsen Foundation International Prize in Neural Plasticity.

8:30 a.m – 11:30 a.m.

Neuropsychopharmacology Reviews Plenary

Regency Ballroom 1 & 2

PL

The Role of Histone Deacetylase 3 (HDAC3) in the Acquisition and Extinction of Cocaine-context Associated Memories

Marcelo Wood

University of California, Irvine

How do drugs of abuse, such as cocaine, cause stable changes in neural plasticity that in turn drive long-term changes in behavior? What kind of mechanism can underlie such stable changes in neural plasticity? One prime candidate mechanism is epigenetic mechanisms of chromatin regulation. Chromatin regulation has been shown to generate short-term and long-term molecular memory within an individual cell. They have also been shown to underlie cell fate decisions (or cellular memory). Now, there is accumulating evidence that in the CNS, these same mechanisms may be pivotal for drug-induced changes in gene expression and ultimately long-term behavioral changes. As these mechanisms are also being found to be fundamental for learning and memory, an exciting new possibility is the extinction of drug-seeking behavior by manipulation of epigenetic mechanisms. In particular, we have focused on understanding the role of histone deacetylase 3 (HDAC3) in the acquisition and extinction of cocaine-context associated memories. We have found that HDAC3 is pivotal for both the acquisition and the persistent extinction of drug-seeking behavior.

Dr. Wood received his Ph.D. from the Department of Molecular Biology at Princeton University in molecular cancer biology. He then switched fields to study the neurobiology of learning and memory at the University of Pennsylvania for his postdoctoral fellowship. He is currently an associate professor in the Department of Neurobiology and Behavior at the University of California Irvine. He is also the Director of the Interdepartmental Neuroscience Program. His research program focuses on understanding the molecular mechanisms underlying long-term memory formation and drug-seeking behavior.

The Impact of MeCP2 Loss- or Gain-of-Function on Synaptic Plasticity

Lisa Monteggia

University of Texas, Southwestern Medical Center, Dallas

Methyl-CpG-binding protein 2 (MeCP2) is a transcriptional regulator of gene expression that is an important epigenetic factor in the maintenance and development of the central nervous system. The neurodevelopmental disorders Rett syndrome and MECP2 duplication syndrome arise from loss of function and gain of function alterations in MeCP2 expression, respectively. Several animal models have been developed to recapitulate the symptoms of Rett syndrome and MECP2 duplication syndrome. Cell morphology, neurotransmission, and cellular processes that support learning and memory are compromised as a result of MeCP2 loss- or gain-of-function. Interestingly, loss of MeCP2 function and MeCP2 overexpression trigger diametrically opposite changes in synaptic transmission. These findings indicate that the precise regulation of MeCP2 expression is a key requirement for the maintenance of synaptic and neuronal homeostasis and underscore its importance in central nervous system function. This review highlights the functional role of MeCP2 in the brain as a regulator of synaptic and neuronal plasticity as well as its etiological role in the development of Rett syndrome and MECP2 duplication syndrome.

Dr. Monteggia's research interests focus on the molecular and cellular basis of neural plasticity as it pertains to psychiatric disorders. She utilizes molecular, cellular, behavioral, biochemical and electrophysiological approaches to elucidate how specific genes may contribute to psychiatric disorders, specifically focusing on better understanding Depression and Rett Syndrome/Autism. Dr. Monteggia is Ginny and John Eulich Professor in Autism Spectrum Disorders, and Associate Professor of Psychiatry, at University of Texas Southwestern Medical Center in Dallas. She is currently an associate editor of *Neuropsychopharmacology*, on the editorial board of *Biological Psychiatry*, as well as on several national advisory and review boards. She has received several awards for her research, including the Daniel X. Freedman Award for outstanding basic research achievement from NARSAD, the Rising Star Basic Research Award from the International Mental Health Research Organization, and the Daniel H. Efron Award from ACNP.

8:30 a.m – 11:30 a.m.

Neuropsychopharmacology Reviews Plenary
Regency Ballroom 1 & 2

PL

microRNA miR-128 Controls Dopamine-mediated Locomotor Behavior and Prevents Fatal Epilepsy-like Disease in MiceAnne Schaefer

Friedman Brain Institute, Mount Sinai School of Medicine

MiR-128 is one of the most abundant, postnatal brain-enriched miRNAs and is encoded by two different genes, miR-128-1 and miR-128-2. We found that the majority of miR-128 in the adult brain originates from the miR-128-2 gene. Loss of miR-128-2 in postnatal forebrain neurons causes a fatal epilepsy-like disease, characterized by hyperlocomotion, increased exploratory activity and spontaneous, recurrent seizures. Moreover, deficiency of miR-128-2 specifically in dopamine 1 receptor (Drd1) expressing neurons is sufficient to reproduce the epilepsy-like disease and is associated with an enhanced neuronal responsiveness to Drd1 stimulation. Using cell-type specific mRNA analysis of miR-128-2 deficient Drd1 neurons combined with neuron-specific analysis of Ago2 associated mRNA targets in neurons in the adult brain we identified >30 miR-128 target genes. The nature of these targets and their potential contribution to the development of the epilepsy-like disease in mice will be discussed.

Dr. Schaefer is Assistant Professor of Neuroscience and Psychiatry and named Seaver Fellow at the Friedman Brain Institute at Mount Sinai School of Medicine. She did her graduate studies at the Johannes Gutenberg University, the Charité University Berlin and The Rockefeller University in New York. In 2004 she joined Paul Greengards Laboratory at The Rockefeller University where she completed her postdoctoral studies. She joined the Friedman Brain Institute at Mount Sinai School of Medicine to start her own laboratory in 2011. Her research is focused on understanding how epigenetic mechanisms contribute to maintenance of specialized neuronal functions and their alteration during psychiatric and neurodegenerative diseases.

Epigenetics of Complex Behaviors and Their Inheritance in Mammals

Johannes Bohacek

The University of Zurich

The development and expression of behaviors in mammals are strongly influenced by environmental factors. When favorable and positive, these factors facilitate appropriate responses and allow normal behaviors, but when adverse and negative, they can lead to behavioral alterations. Stressful and traumatic events early in life are particularly negative risk factors that can induce behavioral and psychiatric conditions such as depression, personality disorders and antisocial behaviors. Such disorders can further not only affect the individuals directly exposed to trauma, but they can also be transmitted and similarly expressed in the following generations. The mechanisms underlying the etiology and inheritance of behavioral symptoms induced by early traumatic stress have been proposed to involve epigenetic processes, but remain undefined. This talk will present an experimental model of early traumatic stress in mice and provides initial evidence for the contribution of epigenetic mechanisms to the impact of negative factors on behavior across generations. This model shows that chronic and unpredictable maternal separation combined with maternal stress causes depressive and impulsive behaviors, social withdrawal and cognitive defects in adult mice, and that these symptoms are transmitted to the following offspring across several generations. It further shows that these alterations are associated with persistent changes in DNA methylation in the promoter-associated CpG island of several genes, both in the germline of the stressed animals and in the brain of the offspring. These findings suggest the implication of epigenetic processes in the impact of negative environmental conditions on behavior.

Johannes Bohacek received a diploma in Psychology at the University of Graz, Austria in 2003. He then moved to the United States to complete a Master's degree in Applied Biopsychology at the University of New Orleans, and in 2009 he earned a PhD in Neuroscience from Tulane University. He then joined the laboratory of Isabelle Mansuy as a Postdoc at the Swiss Federal Institute of Technology (ETHZ) and the University Zurich. He has been coordinating a

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Epigenetics of Complex Behaviors and Their Inheritance in Mammals (continued)

Johannes Bohacek

3-year collaboration with Roche to investigate the role of epigenetic factors in the inheritance of disease, and has been funded by a Postdoctoral Fellowship from the ETHZ. His main research interest focuses on the transgenerational effects of early life stress as a risk factor for the inheritance of neuropsychiatric disease.

NIH Institute Director's Session

Chair: John Krystal

Panelists:

Kenneth Warren
NIAAA

Thomas Insel
NIMH

Nora Volkow
NIDA

Neil Buckholtz
NIA

2:30 p.m. – 6:30 p.m.

Hot Topics

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Hot Topics

Co-Chairs: Anissa Abi-Dargham and Randy Blakely

- 2:30 p.m. The Sex Biased Phosphoproteome: A Novel Approach Towards Understanding The Molecular Basis for Sex Differences in Neuropsychiatric Diseases
Rita Valentino
- 2:42 p.m. Alarm Pheromone Processing Areas are Involved in the Intergenerational Social Transfer of Emotional Trauma
Jacek Debiec
- 2:54 p.m. Epigenetics, Neurodevelopment, and Risk for Anxiety and Depression in Model Rats
Sarah M. Clinton
- 3:06 p.m. Astrocyte-specific Ablation in the Mouse Prefrontal Cortex Induces Depressive-like and Anxiety-like Deficits
Mounira Banasr
- 3:18 p.m. Reduced Mitochondrial Energy Production in Major Depressive Disorder: Associations with the Serotonin Transporter and Glutamine Synthetase Genes
Chadi G. Abdallah
- 3:30 p.m. Adolescent Stressors to Epigenetic Modulation in Dopaminergic Neurons Via Glucocorticoids: A Novel Model for Psychotic Depression
Akira Sawa
- 3:42 p.m. Stress Response Systems in Adolescent Girls and Boys with Major Depression: A Multi-modal Approach
Kathryn R. Cullen

Hot Topics (continued)

Co-Chairs: Anissa Abi-Dargham and Randy Blakely

- 3:54 p.m. PTSD is Associated with an Increased Prevalence of Autoimmune Disorders
Thomas Neylan
- 4:06 p.m. The Peripheral Immune System Functionally Contributes to Susceptibility to Repeated Social Defeat Stress
Georgia E. Hodes
- 4:18 p.m. Pain-related Depression of the Mesolimbic Dopamine System in Rats: Expression, Blockade by Analgesics, and Role of Endogenous Kappa Opioids
Steve Negus
- 4:30 p.m. Evidence of an Inflammatory Pathway Leading to Psychosis in Bipolar Disorder
Mikael Landen
- 4:42 p.m. Mapping Brain Metabolic Connectivity in Awake Rats with MicroPET and Optogenetic Stimulation
Panayotis Thanos
- 4:54 p.m. Fine-grained Working Memory Load Manipulation Reveals Absence of Normative Inverted-U Activation in Schizophrenia
Jared X. Van Snellenberg
- 5:06 p.m. BDNF Val66Met Modulates BOLD Response to Affective Instrumental Learning in Humans
Mbemba Jabbi

2:30 p.m. – 6:30 p.m.

Hot Topics

Regency Ballroom 1 & 2

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Hot Topics (continued)

Co-Chairs: Anissa Abi-Dargham and Randy Blakely

- 5:18 p.m. “Erasing” a Cocaine-cue Memory in Mice: Potential Implications for Relapse to Drug Taking
Sheena Josselyn
- 5:30 p.m. Markedly Reduced mGluR5 Receptor Binding in Smokers and Ex-smokers Determined by [11C]ABP688 Positron Emission Tomography
Gregor Hasler
- 5:42 p.m. Differentiating Neural Networks with Interleaved TMS-BOLD Imaging: Insight into Addiction
Colleen A. Hanlon
- 5:54 p.m. Treatment of Depression with Botulinum Toxin A: A Randomized, Double-Blind, Placebo Controlled Trial
Eric Finzi
- 6:06 p.m. A Randomized Controlled Crossover Trial of Ketamine in Obsessive-Compulsive Disorder
Carolyn I. Rodriguez
- 6:18 p.m. Effects of Oxytocin on Social Cognition and Olfaction in Adults with Schizophrenia and Healthy Subjects
Josh Woolley

The Sex Biased Phosphoproteome: A Novel Approach Towards Understanding The Molecular Basis for Sex Differences in Neuropsychiatric Diseases

Tuesday, Poster #138

Rita Valentino, Debra Bangasser, Zach Plona, Hua Ding, Christopher McKennan, Steven Seeholzer
The Children's Hospital of Philadelphia

Background: Stress-related psychiatric disorders (e.g., depression, post-traumatic stress disorder) are nearly two fold more prevalent in females compared to males. Our recent studies implicated sex differences in signaling and trafficking of the receptor for corticotropin-releasing factor (CRF), the molecule that orchestrates the stress response, as a molecular mechanism for these differences. In females the CRF receptor (CRF1) was more highly coupled to its GTP-binding protein (Gs) and it did not associate with b-arrestin 2 following stress as seen in males. These sex differences rendered neurons of female rats more sensitive to CRF and less able to adapt to excess CRF through b-arrestin 2-mediated CRF1 internalization. In addition to promoting receptor internalization, b-arrestin 2 also acts as a scaffold linking receptors to G-protein independent signaling pathways. This suggests that CRF1 signaling is sex biased, such that in females signaling is preferentially through Gs-protein related pathways whereas in males it can involve b-arrestin 2-related, Gs-protein independent pathways. By engaging different signaling cascades stressors can have sex-specific cellular, physiological, behavioral and pathological consequences. Because Gs-protein and b-arrestin 2 signaling regulate phosphorylation dynamics in cells we tested the hypothesis that the excessive CRF that occurs in stress-related psychiatric disorders could result in sex specific phosphoprotein profiles. Keys to understanding sex differences in stress-related psychiatric disorders may lie in the differences between these profiles. This was tested by performing a deep phosphoproteomic analysis of cortex of male and female CRF overexpressing (CRF-OE) mice using stable isotope labeling of whole mouse (SILAM) and high resolution mass spectrometry. **Methods:** Experimental protocols were approved by IACUC of The Children's Hospital of Philadelphia and were in accordance with the NIH *Guide for the Care*

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Hot Topics

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The Sex Biased Phosphoproteome: A Novel Approach Towards Understanding The Molecular Basis for Sex Differences in Neuropsychiatric Diseases

Tuesday, Poster #138 (continued)

Rita Valentino

and Use of Laboratory Animals. Male and female CRF-OE transgenic mice with the mMt-1 promoter driving the CRF gene (including introns) backcrossed onto the C57BL/6 mouse strain were purchased from Jackson Labs. Cortical protein homogenates were obtained from experimental subjects and protein from male and female SILAM brains obtained from mice on a stable isotope labeled amino acid diet was added to experimental samples as an internal standard. After tryptic digest, phosphoproteins were enriched and separated by hydrophylic interaction chromatography in conjunction with immobilized metal affinity chromatography. Samples were subjected to reversed phase LC-MSMS. Raw data were analyzed using MaxQuant 1.2.7.4 and Andromeda search engine.

Results: Over 5300 unique phosphopeptides were identified that were present in both female CRF-OE mice (FOE) and male CRF-OE mice (MOE). Approximately 14% of these differed between groups with 269 being more abundant in FOE and 131 being more abundant in MOE (1%FDR). Kinases were prominent in the FOE group (44 kinases) and were not as well represented in the MOE (10 kinases). Additionally, more phosphatases and phosphodiesterases were represented in the FOE compared to the MOE group. The different types of kinases in the two groups supported the concept that signaling was different between FOE and MOE mice. Calcium/calmodulin kinase (CAMK) subunits and serine/threonine kinases were prominent in the FOE group, whereas no specific kinase was more apparent in the MOE group. Analysis of the overrepresented amino acid motifs in both groups showed some overlap between the groups but also identified motifs that distinguished the groups. Finally, analysis of protein domains using PROSITE revealed that protein kinase domains dominated the FOE group and PDZ and Src domains were most representative in the MOE group. These initial findings support the hypothesis that sex biased CRF receptor signaling translates

The Sex Biased Phosphoproteome: A Novel Approach Towards Understanding The Molecular Basis for Sex Differences in Neuropsychiatric Diseases

Tuesday, Poster #138 (continued)

Rita Valentino

to different profiles of phosphoproteins in brain. Finally, an initial functional pathway analysis strongly implicated the FOE phosphoproteome in calcium signaling with >20 phosphoproteins related to this and also in Alzheimer's disease with key phosphoproteins related to amyloid b (A4) precursor protein binding and processing, including beta secretase and phosphorylation of tau, including tau tubulin kinase and CAMK.

Conclusions: Here we used a proteomic approach to test a hypothesis generated from receptor immunoprecipitation studies. A deep phosphoproteomic analysis comparing cortical tissue of male and female CRF-OE mice to mimic the CRF overactivity of stress-related psychiatric disorders revealed that a substantial proportion of the phosphoproteome differed significantly between the sexes. The different pattern of kinases and protein domains represented in the two groups supported the notion of sex biased CRF signaling. The finding that many phosphoproteins associated with Alzheimer's disease are exclusively present in the FOE phosphoproteome suggests a sex by stress interaction in this disease that may play a role in determining vulnerability.

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Hot Topics

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Alarm Pheromone Processing Areas are Involved in the Intergenerational Social Transfer of Emotional Trauma

Monday, Poster #60

Jacek Debiec, Regina M. Sullivan
University of Michigan, Ann Arbor

Background: A number of studies suggest that psychological trauma can be transmitted to subsequent generations and potentiate the emergence of mental disorders, such as depression, PTSD and specific phobias (Cameron et al., 2005; Yehuda et al., 1998; de Rosnay et al. 2006). We have recently proposed a rat model of a transgenerationally transmitted trauma (Debiec and Sullivan, ACNP 2011). Using 2-DG autoradiographic imaging, we have shown that the lateral nucleus of the amygdala, a key structure underlying associative fear learning, is involved in the transfer of fear responses from mothers to infants (Debiec and Sullivan, ACNP 2011). We have demonstrated that in our paradigm, the social transmission of fear from mothers to their pups is mediated by maternal alarm odor (Debiec and Sullivan, ACNP 2011). Here we ask whether brain structures which are known to process alarm pheromones are also involved in the acquisition of socially transmitted fear.

Methods: Female rats that had undergone olfactory fear conditioning training were re-exposed to their conditioning stimulus (CS) in the presence of the pups (“Paired-CS” ; n=7). Controlled groups included pups exposed to mothers that had been previously conditioned but were not re-exposed to the CS (“Paired-No CS”; n=4) and pups exposed to mothers that had been previously exposed to unpaired presentations of CS odor and electric shock (“Unpaired-CS”; n=6). All pups were injected with ^{14}C S 2-DG prior to their exposure in order to assess the neural changes during acquisition. Following exposure to their mothers, 2-DG reuptake in pups’ brains was assessed.

Results: Statistical analysis revealed that “Unpaired-CS” group exhibited decrease of 2-DG uptake in the granular part of the accessory olfactory bulb (AOB) as compared to two other groups [ANOVA, $F(2, 14) = 6.089$; $p < 0.02$; post hoc Newman-Keuls Multiple Comparison Test $p < 0.05$]. “Paired-CS” group showed significant increase of 2-DG uptake in necklace glomeruli (NG) [ANOVA,

Alarm Pheromone Processing Areas are Involved in the Intergenerational Social Transfer of Emotional Trauma

Monday, Poster #60 (continued)

Jacek Debiec

$F(2, 14) = 8.438; p < 0.004$; post hoc Newman-Keuls Multiple Comparison Test $p < 0.05$].

Conclusions: Our data demonstrate that the acquisition of socially transmitted fear in rat pups involves the AOB and the NG which are both involved in processing of intraspecific chemical alarm signaling (Chamero *et al.*, 2012; Luo, 2008). Interestingly, there was no difference in 2-DG uptake in the AOB between the “Paired-CS” and “Paired-No CS” groups suggesting that the history of trauma (fear conditioning) predicts fear and the AOB activation. In contrast, the 2-DG uptake in the NG was significantly increased as compared to two other groups. This pattern of findings suggests that the NG activity underlies acquisition of socially transmitted fear.

This research was supported by grants NIDCD DC009910 and NIMH MH091451 to RMS and NARSAD Young Investigator Award from the Brain & Behavior Research Foundation to JD.

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Hot Topics

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Epigenetics, Neurodevelopment, and Risk for Anxiety and Depression in Model Rats

Tuesday, Poster #152

Sarah M. Clinton, Rebecca K. Simmons, Matthew E. Glover, Phyllis C. Pugh, Huda Akil
University of Alabama, Birmingham

Background: Individual differences in human temperament powerfully shape ability to cope with stress as well as vulnerability to mental illness. Our ongoing work utilizes rats to model individual differences in temperament to understand molecular and neuroanatomical changes in the developing brain that may put an individual at risk for a depressive/anxiety-like phenotype. The hippocampus emerged as a particularly important node in shaping temperament differences, as we found altered hippocampal volume, cell proliferation, gene expression, and epigenetic (DNA methylation) changes in the developing brain of “depression prone” versus “depression resistant” rats. The current experiments highlight similar changes in the developing amygdala – an area well-known for regulating emotion, in part via interconnections with the hippocampus.

Methods: In rats selectively-bred for differences in locomotor response to novelty, low novelty responders (bLR) exhibit high levels of behavioral inhibition, anxiety- and depression-like behavior, compared to high novelty responders (bHR), which are highly aggressive, impulsive and prone to drug-taking. The bLR/bHR phenotypes are highly predictable across generations and emerge as early as the second week of life. Brains were collected from developing bLR/bHR pups at three time points (postnatal days (P)7, 14, and 21). The amygdala was dissected, and RNA and DNA were extracted for Affymetrix microarray gene expression and Epigentek global DNA methylation assays, respectively.

Results: The microarray experiment revealed dramatic global gene expression differences in the developing amygdala of bLR vs. bHR rats. At P7 and P14, approximately 400 genes were differentially expressed between the strains, and by P21, nearly 700 genes were differentially expressed. At P7, 15% of altered genes were downregulated in bLR vs. bHR, while 85% was upregulated. This pattern dramatically changed at the later timepoints, with approximately 80% of altered genes being downregulated in bLR vs. bHR at P14 and P21, while approximately

Epigenetics, Neurodevelopment, and Risk for Anxiety and Depression in Model Rats

Tuesday, Poster #152 (continued)

Sarah M. Clinton

20% were upregulated. The global DNA methylation assay revealed robustly increased DNA methylation in the amygdala of bLR (vs. bHR) rats specifically at P7. There were no group differences at the other ages (P14 and P21), and we did not see differences at any timepoint for several other brain areas, including the hippocampus, caudate putamen, or septum. Ongoing studies are evaluating methylation status of specific genes in P7 amygdala samples from bLR/bHR rats. **Conclusions:** Our earlier work pointed to marked differences in developing hippocampus of bLR vs. bHR rats, suggesting a possible neurodevelopmental underpinning of their distinct behavioral phenotypes. Here we report remarkable bLR/bHR differences in the developing amygdala. Microarray results revealed substantial gene expression changes at P7 and P14 that expand with age (nearly doubling the number of genes changed at P21 vs. P14). This finding is quite interesting as the pattern differs from our prior microarray study in hippocampus; there we found robust bLR/bHR differences at P7 and P14, with extremely few changes at P21. We also discovered robustly increased global methylation in the P7 amygdala of bLR vs. bHR rats. DNA methylation is typically thought to suppress gene expression, so this dramatic DNA methylation difference may contribute to P14 and P21 gene expression differences where 80% of differing genes were downregulated in bLR vs. bHR rats. Ongoing work aims to identify specific gene targets that are differentially methylated in bLR/bHR rats, and determine whether manipulating methylation in the developing brain can ameliorate some aspects of the bLR/bHR phenotypes. Furthermore, we are also exploring possible differences in hippocampal-amygdala connectivity in developing bLR/bHR rats to identify neural circuit differences that correspond with observed molecular changes. Overall this body of work aims to provide insight into the possible genesis of individual differences in emotionality and related risks for the emergence of emotional disorders (e.g. the anxiety-prone nature of bLRs or drug addiction proclivity of bHRs), and delineate the role of epigenetic processes in these phenomena.

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Hot Topics

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Astrocyte-specific Ablation in the Mouse Prefrontal Cortex Induces Depressive-like and Anxiety-like Deficits

Monday, Poster #18

Mounira Banasr, Meiyu Xu, Gerard Sanacora, Christopher Pittenger, Ronald S. Duman

Yale University School of Medicine

Background: Growing evidence implicates glia in the pathophysiology of depression. Reductions in the number of astrocytes have been reported in postmortem studies examining brain tissue from patients with major depression. Preclinical studies have confirmed some of these changes in both the hippocampus and the prefrontal cortex (PFC) in rodent models of depression based on chronic stress. More specifically we have reported that chronic unpredictable stress reduced number of astrocytes expressing GFAP (glial fibrillary associated protein) and others have demonstrated that the S100Beta-positive cell population was unaffected. We have also demonstrated that rat prefrontal cortex (PFC) glial ablation using local infusion of a gliotoxin induces behavioral deficits similar to chronic stress, including anhedonia, anxiety and helplessness. However, the specific contribution of each subtype of glial cell in the development of depressive-like symptoms remains to be characterized.

Methods: To answer this question, we examined the behavioral consequences of targeted ablation of GFAP-positive cells in the PFC in baseline and stress conditions on anhedonia, anxiety and helplessness. To achieve this goal, we developed an approach adapted from *cre/loxP* system strategy in which GFAP-positive cells of the PFC are altered to express the diphtheria toxin (DT) receptor (DTR), and thereby made sensitive to DT exposure. Adult GFAP-*cre* mice and wild type (WT) littermates were infused with AAV5 virus in the PFC. The viral construct was designed to express DTR only in *cre* expressing cells; more specifically the *loxP* sequences were strategically positioned around the CMV promoter to induce the flipping of the promoter thereby inducing expression of DTR in *cre*-cells. Three weeks after bilateral infusion of the virus (AAV5-fCMV-DTR) in the PFC, animals were injected i.p. daily with saline or DT at 3 different doses (0.1, 5, 20 ug/kg) for 4 days and daily sucrose (1%) consumption over 24h-period

Astrocyte-specific Ablation in the Mouse Prefrontal Cortex Induces Depressive-like and Anxiety-like Deficits

Monday, Poster #18 (continued)

Mounira Banasr

was measured. When an effect on sucrose consumption was observed, we also analyzed the effect of glial ablation in other behavioral tests known to be affected by stress and antidepressant treatment.

Results: Two days after the first injection of DT, *GFAP^{cre}+AAV5-fCMV-DTR* mice injected with 20ug/kg of DT showed a significant decrease in sucrose consumption when compared with *GFAP^{cre}+AAV5-fCMV-DTR* mice injected with saline or the 0.1 ug/kg DT. On day 3, both 5 and 20 ug/kg *GFAP^{cre}+AAV5-fCMV-DTR* mouse groups showed reduced sucrose consumption when compared with the saline or the low DT dose group. WT mice infused with *AAV5-fCMV-DTR* showed no change in sucrose consumption when injected with saline or the different doses of DT. We also measured water consumption on day 5 and found no significant difference in WT or *GFAP^{cre}* mice injected with saline or the various doses of DT. We found that animals *GFAP^{cre}+AAV5-fCMV-DTR* injected with 5 and 20 ug/kg still showed decreased sucrose consumption on day 8, but not day 14 (4 or 10 days after the last injection of DT, respectively). We also examined the consequences of the cell ablation in behaviors measuring the anxiety-like state of the animals. Overall, we found that although the effects were not always significant, the *GFAP^{cre}+AAV5-fCMV-DTR* animals treated with DT tend to exhibit more anxiety-like deficits when compared to the *GFAP^{cre}+AAV5-fCMV-DTR* animals injected with saline. More precisely, we found that *GFAP^{cre}+AAV5-fCMV-DTR* animals treated with the 3 doses of DT showed a significant increase in their latency to drink a milk solution in the novelty induced hypophagia test ($P<0.05$), a trend toward increased latency to feed in the novelty suppressed feeding test ($P=0.15$) and a trend to spend less time in the center in the open field test ($P=0.13$).

Conclusions: Our results demonstrate that selective ablation of GFAP-positive cells in the PFC induces rapid anhedonia- and anxiety-like deficits that persist for at least 8 days but are transitory and not observed at day 14; this reversal could

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Astrocyte-specific Ablation in the Mouse Prefrontal Cortex Induces Depressive-like and Anxiety-like Deficits

Monday, Poster #18 (continued)

Mounira Banasr

be due to glial renewal after cessation of DT infusion, a possibility that we are currently testing. These findings demonstrate that loss of GFAP-positive cells in the PFC is sufficient to cause depressive behavior, supporting the hypothesis that glial loss in depressed patients contributes to depressive symptoms. Moreover, this cell selective ablation approach will allow us to further study the cellular consequences of this astrocyte-specific cortical ablation on the function of the PFC, as well as the contribution of other populations of cells (glial or neuronal) in the development of depressive-like behavior.

Reduced Mitochondrial Energy Production in Major Depressive Disorder: Associations with the Serotonin Transporter and Glutamine Synthetase Genes

Monday, Poster #91

Chadi G. Abdallah, Graeme F. Mason, Henk De Feyter, Madonna Fasula, Ben Kelmendi, Arthur Simen, Lihong Jiang, John H. Krystal, Douglas L. Rothman, Gerard Sanacora
Yale University

Background: Proton magnetic resonance spectroscopy (^1H -MRS) studies have demonstrated altered concentration of aminoacid neurotransmitters in the occipital brain of patients with major depressive disorder (MDD). However, the functional implications of this alteration in total glutamate and GABA levels are not well understood. To elucidate the underlying neuronal mechanisms, we employed ^{13}C magnetic resonance spectroscopy (^{13}C -MRS) to investigate neurotransmitter fluxes and mitochondrial neuroenergetics in MDD subjects.

Methods: 21 medication-free patients with MDD and 14 age- and gender-matched healthy controls had ^1H -MRS and ^{13}C -MRS scans with viable data. A subset of the subjects was genotyped for the serotonin transporter (5-HTTLPR) and glutamine synthetase (GLUL) genes. ^1H -MRS measured total glutamate and GABA concentration in a single voxel placed in the occipital cortex. $[1\text{-}^{13}\text{C}]$ -glucose was infused intravenously during ^{13}C -MRS acquisition, which provided *in vivo* measures of neuronal and astrocytic tricarboxylic acid cycle (V_{TCA_n} and V_{TCA_a}) for mitochondrial energy production, GABA synthesis (V_{GAD}), and glutamate-glutamine cycle (V_{cycle}), which is a measure of glutamate release and uptake.

Results: Patients with MDD had a 26% reduction in mitochondrial energy production of glutamatergic neurons [Mean \pm SEM; MDD $V_{\text{TCA}_n} = 0.36 \pm 0.03$ mM, Healthy $V_{\text{TCA}_n} = 0.49 \pm 0.05$ mM, $t = 2.30$, $n = 35$, $p = 0.028$]. GABA and glutamate concentrations, V_{cycle} , and V_{GAD} did not differ between groups ($p > 0.1$). Among the MDD subjects, carriers of the short allele of 5-HTTLPR (SS or SL) have reduced neuronal V_{TCA} compared to those homozygous for the long allele (LL) [$t = 2.86$, $n = 12$, $p = 0.017$]. In addition, we found a significant association [$p < 0.009$] between astrocytic V_{TCA} and 3 GLUL SNPs (rs12136955; rs12735664; rs4652705).

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Reduced Mitochondrial Energy Production in Major Depressive Disorder: Associations with the Serotonin Transporter and Glutamine Synthetase Genes

Monday, Poster #91 (continued)

Chadi G. Abdallah

Conclusions: The reduction of glutamatergic neuronal energy production (V_{TCAAn}) in the occipital brain of depressed subjects raises two possibilities: (1) reduced activity of glutamatergic neurons in this brain region or (2) impaired mitochondrial function. Although we did not detect a difference in activity (as measured through V_{cycle}), this may be due to the V_{cycle} measurement with $1\text{-}^{13}\text{C}$ glucose being less precise than the V_{TCAAn} measurement. However with the recent demonstration that combined use of ^{13}C glucose and ^{13}C acetate enhances the precision of measuring V_{cycle} several fold, it would be possible to distinguish these possibilities in future studies, as well as further explore the impact of MDD on the astrocytic TCA cycle. Finally, the serotonin transporter and glutamine synthetase genes were associated with mitochondrial energy production in glutamatergic neurons and astrocytes, respectively. Further exploration, in future studies, of these intriguing preliminary findings may provide insight in the mechanisms through which these genes affect cerebral function and psychopathology.

Adolescent Stressors to Epigenetic Modulation in Dopaminergic Neurons Via Glucocorticoids: A Novel Model for Psychotic Depression

Monday, Poster #61

Minae Niwa, Akira Sawa
Johns Hopkins University School of Medicine

Background: Human behavior in adulthood is crucially influenced by various environmental conditions during childhood and adolescence. Nonetheless, individual responses to such environmental factors vary, mainly because of different genetic predispositions among individuals. These gene-environmental interactions may also underlie a variety of neuropsychiatric disorders. Elucidation of the underlying mechanisms and mediators should help development of a means to intervene in such disorders, including prophylactic environmental readjustment.

Methods: A genetic model with isolation stress was examined by behavioral assays and neurochemical assessments. A glucocorticoid receptor antagonist RU38486 (mifepristone) was used. The nuclei of mesocortical and mesolimbic dopaminergic neurons in ventral tegmental area were enriched by labeling with fluorescent retrograde beads and fluorescence-activated cell sorting in a projection-specific manner. Epigenetic modification of the gene for tyrosine hydroxylase was examined by bisulfite sequencing.

Results: We exposed a genetic model (DISC1 mutant mice) to 3-week isolation stress during adolescence (from 5 to 8 weeks of age) and observed behavioral deficits (prepulse inhibition, forced swim test, and locomotor activity) and neurochemical changes associated with dopamine in adulthood. Two distinct dopaminergic projections (mesocortical and mesolimbic) originating from the ventral tegmental area was differentially affected in this model. A mild isolation stress with the genetic risk affected only mesocortical, but not mesolimbic, projection of dopaminergic neurons in which DNA hypermethylation of the tyrosine hydroxylase gene was elicited. The epigenetic alternations were long-lasting, evident in adult animals even if they were maintained in the normal group housing until 20 weeks after the transient adolescent isolation. These molecular, neurochemical, and behavioral deficits in this model were normalized

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Adolescent Stressors to Epigenetic Modulation in Dopaminergic Neurons Via Glucocorticoids: A Novel Model for Psychotic Depression

Monday, Poster #61 (continued)

Akira Sawa

by an administration of a glucocorticoid receptor antagonist RU38486. Given that behavioral deficits may be relevant to endophenotypes of psychotic depression and that RU38486 uniquely benefits this condition, this model of gene-environmental interaction may be a promising tool to study psychotic depression.

Conclusions: This study shows a novel link between adolescent stressors and epigenetic controls in dopaminergic neurons via glucocorticoids, which addresses a central question of neurobiology of how adult behavior patterns are formed by a combination of genetic factors and environmental stressors. Interestingly, the epigenetic modifications by the primary stressor are maintained for long (the isolation stress during adolescence leads to a long-lasting change in adulthood). The present study also offers an innovative mouse model for psychotic depression, a common and debilitating psychiatric disease. The availability of a preclinical model would allow us to study underlying pathological mechanisms, including those in the premorbid and prodromal stages, and explore novel therapeutic strategies. Such a model could provide a good template not only for screening compounds with better efficacy and fewer side effects, but also for prophylactic environmental readjustment, which is crucially important in clinical psychiatry.

Stress Response Systems in Adolescent Girls and Boys with Major Depression: A Multi-modal Approach

Monday, Poster #139

Kathryn R. Cullen, Bonnie Klimes-Dougan, Alaa Hourii, Kelvin O. Lim
University of Minnesota Medical School

Background: The pathophysiology of major depressive disorder (MDD) involves impairment within the neurobiological systems that underlie the response to stress. The neuroendocrine stress response system, encompassed by the Hypothalamic Pituitary Adrenal (HPA), is centrally implicated in MDD. Additionally, fronto-limbic neural circuitry is (a) implicated in MDD, (b) associated with stress response and (c) tightly linked with the HPA system. Core components of this network include the amygdala and the rostral anterior cingulate cortex (rACC). Sex differences have previously been identified in brain development and in stress response. However, the functioning of neurobiological stress systems in adolescents has been understudied. This research is particularly important in adolescence as neurobiological systems are still undergoing development. The goal of the present study was to examine the neurobiological stress systems in adolescent girls and boys with MDD.

Methods: Participants included 54 adolescents aged 12-19, including 39 with MDD (22 unmedicated, 17 medicated) and 15 healthy comparison volunteers. All participants underwent diagnostic evaluation, the Trier Social Stress Test (TSST), and brain imaging (which included a T1 anatomical scan). Statistical analyses were conducted using SPSS. In the TSST, participants were asked to prepare and deliver a short speech to a strange audience. Salivary cortisol measurements were taken at the beginning and immediately following the procedure, and at 15 minutes intervals afterward for a total of five time points. Repeated measures analysis was conducted on the cortisol levels, including group (control, medicated depressed and unmedicated depressed) and sex as fixed effects. Multivariate regression analyses were also conducted on peak cortisol levels and area under the curve measurements to assess for effects of group and sex. Analysis of anatomical imaging data was conducted using the FreeSurfer software to extract brain volumes from key regions of interest. A multivariate regression analysis

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Stress Response Systems in Adolescent Girls and Boys with Major Depression: A Multi-modal Approach

Monday, Poster #139 (continued)

Kathryn R. Cullen

was conducted on key fronto-limbic brain regions that were hypothesized to relate to MDD and the stress system: bilateral amygdala volumes and bilateral rACC volumes, including group and sex as fixed effects, and intracranial volume as a covariate. Finally, we examined correlations between cortisol measurements (peak levels and area under the curve) and brain volumes (amygdala and rostral anterior cingulate.)

Results: For the TSST, repeated measures analysis revealed a significant group by time effect ($F=3.4$, $p=0.002$) and a trend-level sex effect ($F=2.0$, $p=0.1$). The control group exhibited a normative elevation in cortisol followed by return to baseline; the unmedicated group showed elevated baseline cortisol levels, peaked higher and remained elevated at the end of the experiment; and the medicated group showed a marked decrease during the task followed by a return to baseline. Examination of the results separately by sexes showed that while depressed boys tended to show an over-responding pattern, the depressed girls showed an under-responding pattern. Medication seemed to flatten responses for both boys and girls. Multivariate analyses revealed of peak and summary cortisol measures showed significant effects for group ($F=3.7$, $p=0.000$), sex ($F=2.6$, $p=0.03$), and a group by sex interaction ($F=2.8$, $p=0.003$). Whereas unmedicated boys had much higher peak and summary levels than both controls and medicated boys, the female groups were more similar. Finally, although we did not identify significant group effects with respect to brain volumes for our regions of interest, we did find significant correlations between both left and right amygdala volume and peak cortisol during the recovery phase of the experiment (left: $r = -0.4$, $p=0.006$; right: $r = -0.3$, $p = 0.03$) as well as with the summary measure (area under the curve) (left: $r = -0.4$, $p=0.008$, right: $r = -0.3$, $p=0.03$.)

Conclusions: We report results from a multi-method study that examined stress systems in adolescents with MDD. Our findings suggest that the systems that underlie the stress response in adolescents with depression are abnormal,

Stress Response Systems in Adolescent Girls and Boys with Major Depression: A Multi-modal Approach

Monday, Poster #139 (continued)

Kathryn R. Cullen

with unmedicated adolescents showing elevated stress responses and delayed recovery. Current treatment with medication appears to dampen the biological response to social stress. These cross sectional results suggesting the impact of treatment on HPA responding should be followed by longitudinal studies to directly test whether treatment mitigates the stress response in depressed teens. Although this study included fewer boys than girls, tentatively our results suggest that unmedicated depressed boys show an over-responding pattern, whereas for girls the pattern is that of under-responding. The sex effects noted in this study require confirmation with larger and more balanced samples. Finally, although fronto-limbic brain volumes did not differentiate groups, they were inversely related to cortisol measures in the adolescents of this study. Additional research using multi-modal approaches is needed to further delineate the inter-dependent relationships across neurobiological stress systems.

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PTSD is Associated with an Increased Prevalence of Autoimmune Disorders

Wednesday, Poster #137

Aoife O'Donovan, Beth Cohen, Daniel Bertenthal, Mary Margaretten, Karen Seal, Thomas Neylan
University of California, San Francisco

Background: Accumulating evidence links post-traumatic stress disorder (PTSD) with elevated inflammatory activity. However, the clinical significance of this association is unclear. Though inflammation could increase the risk of autoimmune disease, little is known about whether patients with PTSD are at increased risk of developing autoimmune disorders.

Methods: We conducted a retrospective cohort study of 673,277 Iraq and Afghanistan veterans under 55 years old who received VA healthcare from October 1, 2005 to March 31, 2012 with at least one year of follow up. Department of Veterans Affairs administrative data were used to identify ICD-9 codes for mental health and autoimmune disorders and to obtain sociodemographic, military service, and health service utilization information. Generalized Linear Models were used to ascertain the association of PTSD with subsequent autoimmune diagnoses after adjusting for age, race and number of primary care visits.

Results: The sample was 88% male and 49% white with a mean age of 31.3 years (+/- 8.7). PTSD was diagnosed in 206,623 (31%) veterans and mental health disorders other than PTSD were diagnosed in an additional 132,242 (20%) veterans. Compared to veterans with no mental health diagnoses, those diagnosed with PTSD had increased risk for subsequent diagnosis with thyroiditis (Adjusted Relative Risk [ARR] = 1.74; 95% CI, 1.67, 1.82), rheumatoid arthritis, (ARR = 1.92, 95% CI, 1.67, 2.20), inflammatory bowel disease (ARR = 1.32, 95% CI, 1.20, 1.46), multiple sclerosis (ARR = 2.23, 95% CI, 1.88, 2.64), systemic lupus erythematosus (ARR = 1.81, 95% CI, 1.48, 2.23) and any of these disorders alone or in combination (ARR = 1.50, 95% CI, 1.45, 1.56). Moreover, while there was an increased risk for each of these disorders in veterans with mental health disorders other than PTSD, the risk was consistently higher in those diagnosed

PTSD is Associated with an Increased Prevalence of Autoimmune Disorders

Wednesday, Poster #137 (continued)

Thomas Neylan

with PTSD. Women had significantly higher risk for autoimmune disorders overall, but the pattern of results was similar in men and women.

Conclusions: Veterans with PTSD appear to be at increased risk for autoimmune disorders compared to those with no or other mental health diagnoses. Future prospective longitudinal cohort studies are needed to establish causality, measure inflammatory markers in conjunction with PTSD, and evaluate whether successful treatment of PTSD reduces risk of autoimmune disorders.

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Hot Topics

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The Peripheral Immune System Functionally Contributes to Susceptibility to Repeated Social Defeat Stress

Monday, Poster #13

Georgia E. Hodes, Sam A. Golden, Daniel J. Christoffel, Madeline Pfau, Mitra Heshmati, Marylene Leboeuf, Miriam Merad, Scott Russo
Mount Sinai School of Medicine

Background: Subjects with major depression have increased circulating levels of pro-inflammatory cytokines, such as Interleukin-6 (IL-6), which is thought to reflect hyperactivity of their peripheral immune system (Dowlati et al., 2009). We have previously shown similar increases in serum IL-6 levels following repeated social defeat stress (RSDS), a mouse model of mood and anxiety disorders. Mice that are susceptible to RSDS initially have an exaggerated release of IL-6 and exhibit sustained increases of IL-6 levels for at least 1 month following the last defeat. Thus, we predict that there are innate differences in the immune response to stress in susceptible mice that drives depression- and anxiety-like behavioral phenotypes.

Methods: We used RSDS to examine individual differences in response to stress; some animals termed susceptible show a spectrum of depression-like behavior, whereas resilient animals are more similar to controls. To determine whether IL-6 release can be used as a predictive biomarker, we isolated and cultured peripheral blood mononuclear cells (PBMCs) prior to exposure to RSDS, stimulated with the endotoxin lipopolysaccharide (LPS), and then measured IL-6 using enzyme linked immunosorbent assays (ELISA). To determine if peripheral IL-6 was necessary for the development of susceptibility to RSDS, we systemically injected a separate group of animals with an antibody that neutralizes IL-6 in the periphery and tested them for social avoidance, anhedonia (sucrose preference) and anxiety (elevated plus maze). To examine whether the peripheral immune system was sufficient to functionally drive these behavioral adaptations to RSDS, we first ablated the peripheral immune system of naïve mice using irradiation. We then replaced their immune system with bone marrow either from a susceptible mouse following 10 days of RSDS, or a control mouse with a little or no IL-6 response to endotoxin challenge. We then exposed mice to a sub-threshold micro-defeat and tested for depression and anxiety-like behavior.

The Peripheral Immune System Functionally Contributes to Susceptibility to Repeated Social Defeat Stress

Monday, Poster #13 (continued)

Georgia E. Hodes

Results: PBMCs isolated prior to social defeat from mice that later developed a susceptible phenotype had a larger release of IL-6 following LPS stimulation compared to mice that went on to become resilient. Systemic injections of an IL-6 neutralizing antibody in the periphery blocked RSDS-induced social avoidance and anhedonia. Finally, mice that received bone marrow transplants from a susceptible mouse showed greater social avoidance, anhedonia, and anxiety-like behavior following a sub-threshold micro-defeat.

Conclusions: These studies indicate that the peripheral immune system contributes to the development of susceptibility to social defeat stress. We found that a hyperactive peripheral immune response to stress is a risk factor for the development of depression and anxiety-like behavior. We also show a direct functional role of the peripheral immune response to stress in regulating depression and anxiety like behaviors. Together these studies indicate that innate differences in the immune responses to stress may underlie the development of depression like behavior and serve as a novel therapeutic target for treatment.

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Pain-related Depression of the Mesolimbic Dopamine System in Rats: Expression, Blockade by Analgesics, and Role of Endogenous Kappa Opioids

Monday, Poster #52

Steve Negus, Michael Leitl, Matthew L. Banks
Virginia Commonwealth University

Background: Pain is often associated with depression of behavior and mood, and relief from pain-related depression is a common goal of treatment with analgesic drugs. This preclinical study tested the hypothesis that pain-related depression of behavior in rats results from activation of endogenous kappa opioidergic systems and subsequent kappa receptor-mediated inhibition of mesolimbic dopamine neurons. We have reported previously that a common visceral noxious stimulus, intraperitoneal (IP) injection of dilute acid, produces an analgesic-reversible depression of operant responding in an assay of intracranial self-stimulation (ICSS) in rats. The present study compared effects of IP acid and the exogenous kappa agonist U69593 on ICSS and on microdialysis measures of mesolimbic nucleus accumbens dopamine levels in rats. The nonsteroidal anti-inflammatory drug ketoprofen, the mu opioid receptor agonist morphine, and the kappa opioid receptor antagonist norbinaltorphimine were then evaluated for their ability to block acid- and U69593-induced depression of ICSS and nucleus accumbens dopamine. Our hypothesis predicted that both IP acid and U69593 would depress ICSS and nucleus accumbens dopamine levels, and that effects of acid would be blocked by analgesics and by the kappa antagonist.

Methods: Adult male Sprague-Dawley rats were prepared either with intracranial electrodes targeting the medial forebrain bundle (for behavior studies of intracranial self-stimulation) or cannulae targeting the nucleus accumbens (for microdialysis studies of mesolimbic dopamine). Rats in behavioral studies were trained to lever press under a fixed-ratio 1 schedule for electrical brain stimulation, and daily experimental sessions were composed of multiple 10 min components. During each component, the frequency of brain stimulation was systemically varied from 158-56 Hz in ten 0.05 log unit steps, and the primary dependent variable was the total number of stimulations delivered across all frequencies. On test days, ICSS

Pain-related Depression of the Mesolimbic Dopamine System in Rats: Expression, Blockade by Analgesics, and Role of Endogenous Kappa Opioids

Monday, Poster #52 (continued)

Steve Negus

components were conducted before and after experimental treatments, and ICSS data determined after each treatment were expressed as a percent of the baseline data collected before treatment on that day. Rats in neurochemical studies were fitted with microdialysis probes on the day of the experiment, and samples were collected at 6-min intervals before and after experimental treatments. The primary dependent variable was the concentration of dopamine in each sample determined by high performance liquid chromatography coupled to electrochemical detection. Dopamine levels determined after each treatment were expressed as a percent of the baseline data collected before treatment on that day. In both types of studies, rats were treated with vehicle (-30 min), 3.2 mg/kg ketoprofen (-30 min), 3.2 mg/kg morphine (-30 min) or 32 mg/kg norbinaltorphimine (-24 hr) before subsequent treatment with vehicle, 0.56 mg/kg U69593 or dilute lactic acid (1.8 or 5.6% in water). All studies were approved by the Virginia Commonwealth University IACUC and were conducted in accordance with the National Institutes of Health *Guide for the Care and Use of Laboratory Animals*.

Results: The acid noxious stimulus produced a concentration- and time-dependent depression of both ICSS and nucleus accumbens dopamine, and effects of the highest acid concentration (5.6%) in both assays were similar in magnitude to effects of 0.56 mg/kg U69593. Acid-induced depression of ICSS and nucleus accumbens dopamine was blocked by pretreatment with the analgesics ketoprofen and morphine, but not by the kappa antagonist norbinaltorphimine. Conversely, U69593-induced depression of ICSS and dopamine was blocked by norbinaltorphimine but not by ketoprofen; morphine produced intermediate effects. Neither ketoprofen nor norbinaltorphimine altered ICSS or dopamine levels when administered alone without acid. Morphine alone significantly increased basal dopamine, and produced biphasic effects on ICSS manifested as facilitation of low ICSS rates maintained by low brain stimulation frequencies and

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Pain-related Depression of the Mesolimbic Dopamine System in Rats: Expression, Blockade by Analgesics, and Role of Endogenous Kappa Opioids

Monday, Poster #52 (continued)

Steve Negus

reduction in high ICSS rates maintained by high brain stimulation frequencies.

Conclusions: These results support a role for the mesolimbic dopamine system, but not of endogenous kappa opioid systems, in mediating pain-related depression of behavior in rats. Further research to investigate mechanisms of pain-related depression of behavior and mesolimbic dopamine levels may reveal new targets for analgesic drug development.

Evidence of an Inflammatory Pathway Leading to Psychosis in Bipolar Disorder

Tuesday, Poster #135

Mikael Landén, Carl Sellgren, Magdalena Kegel, Carl-Johan Ekman, Patrick Sullivan, Jordan W. Smoller, Pamela Sklar, Göran Engberg, Sophie Erhardt
The Sahlgrenska Academy at Gothenburg University

Background: Family history is the strongest risk factor for bipolar disorder. Yet it has been difficult to identify susceptibility gene variants. An alternative approach to unearth causal genetic mutations is to focus on biomarkers, i.e., measurable key components in biological pathways between genotype and disease. We therefore conducted a genome wide association study (GWAS) of kynurenic acid (KYNA) in cerebrospinal fluid (CSF), based on that elevation of KYNA in brain is a consistently found biochemical aberration in psychotic disorders. We then studied the genetic finding in relation to psychotic symptoms, cognition, and brain gray matter volume. Lastly, we used an *in vitro* model to test if IL-1b is the link between the genetic variant and elevated CSF levels of KYNA.

Methods: CSF was collected from patients with bipolar disorder (N=76) and a genome-wide association study in relation to CSF KYNA was conducted. Patients underwent magnetic resonance imaging (MRI) scans of the brain and a neurocognitive test battery. Human cortical astrocytes were cultured and stimulated with recombinant human IL-1b (10ng/ml). Analysis of KYNA was performed using an isocratic reversed-phase high-performance liquid chromatography (HPLC) system.

Results: One SNP located on chromosome 1 reached genome-wide statistical significance in relation to CSF KYNA (rs10158645, $b=1.05$, $P=3.85 \times 10^{-8}$, $MAF=0.15$). The minor allele of rs10158645 was associated with increased risk of psychotic features ($n=76$, $OR=4.0$, $95\%CI:1.4-12$), increased verbal working memory ($n=108$, $b=1.8$, $P=0.019$) and increased gray matter thickness in corresponding brain regions ($n=138$, $P=0.02$, voxel-level FWE-corrected). The *in vitro* results showed that IL-1b increases KYNA levels in human cortical astrocytes by inducing the rate-limiting enzyme TDO2.

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Evidence of an Inflammatory Pathway Leading to Psychosis in Bipolar Disorder

Tuesday, Poster #135 (continued)

Mikael Landén

Conclusions: The minor allele of rs10158645 has previously been coupled to a decreased expression of sortin nexin 7 (SNX7), which in turn activates caspase-8 that increases IL-1 β . Here we found that IL-1 β stimulates KYNA, known to be increased in psychotic disorders. This raises the possibility that SNX7-induced IL-1 β dependent activation of the kynurenine pathway is a molecular pathway underlying psychosis in bipolar disorder.

Mapping Brain Metabolic Connectivity in Awake Rats with MicroPET and Optogenetic Stimulation

Wednesday, Poster #46

Panayotis Thanos, Lisa Robison, Eric Nestler, Ronald Kim, Mike Michaelides, Mary Kay Lobo, Nora D. Volkow
National Institute on Alcohol Abuse and Alcoholism

Background: Optogenetics allows researchers to map neuronal circuit function in the rodent brain in vivo (Zhang et al., 2010; Lee and Deisseroth, 2012). The combined use of optogenetics and functional magnetic resonance imaging (fMRI) has been used to investigate functional connectivity in the rodent brain (Lee et al., 2010; Lee 2012; Lee and Deisseroth, 2012); however, these are limited by the use of anesthesia, which affects neuronal activity (Qiu et al., 2008; Tsurugizawa et al., 2010). Positron emission tomography (PET) using [¹⁸F] 2-fluoro-2-deoxy-D-glucose (FDG), however, allows researchers to non-invasively measure regional brain glucose metabolism (BGluM), a marker of brain activity, in the awake rodent. The present study used mPET with FDG to measure optogenetic stimulation (OGS) of the nucleus accumbens (NAc). We tested the hypothesis that excitation of the NAc by OGS would increase metabolism in the NAc and in its downstream projection regions. In parallel, we mapped c-Fos expression to corroborate regional activation by OGS.

Methods: Male Sprague Dawley rats (8-10 weeks) were anesthetized and Adeno-associated virus serotype 2 (AAV2)-hsyn-ChR2-EYFP (n=8/group) or AAV2-GFP control virus (n=9/group) was infused into the right NAc core (AP +1.7, ML +1.5, DV -6.5 from bregma) through a 20 gauge cannula. Rats recovered for a minimum of two weeks while waiting for optimal AAV expression. The experiment was conducted in accordance with the Guide for the Care and Use of Laboratory Animals (1996) and approved by the BNL Institutional Animal Care and Use Committee (IACUC). Each rat was scanned twice using FDG-mPET, one week apart (counterbalanced design): once at baseline (optical fiber attached but no stimulation applied) and once following OGS. Rats were placed in a small plexiglass cage to restrict movement and minimize activation from motor behavior. Blue (473 nm) light stimulation, pulsed at 10Hz, was applied

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Mapping Brain Metabolic Connectivity in Awake Rats with MicroPET and Optogenetic Stimulation

Wednesday, Poster #46 (continued)

Panayotis Thanos

through the optical fiber at 30 second intervals for five minutes (light turned off for the baseline scan). Rats were injected intraperitoneally with ~0.5 mCi of FDG (30 minute awake uptake), during which time blue light stimulation was continued, and locomotor activity was measured. After the uptake period, rats were anesthetized and scanned on an R4 mPET tomograph for 30 minutes. Statistical Parametric Mapping (SPM) analysis was performed using paired t-tests for each group (GFP and Chr2) comparing regional brain glucose metabolism between the baseline and the stimulation scans [threshold: $p < 0.005$, $K_e > 100$, $T > 5.7$]. A region of interest (ROI) was manually drawn in the NAc cluster that was significantly activated in the Chr2 group, and activity was measured for the baseline and stimulation condition for the GFP and Chr2 rats. Rats (GFP: $n=4$; Chr2: $n=6$) were again stimulated with blue light for ten minutes, and 90 minutes later, rats were anesthetized, perfused, and brain harvested to assess c-Fos immunofluorescence in the NAc.

Results: Brain metabolic differences between baseline and OGS stimulation of the NAc were determined both for activation (stimulation $>$ baseline) and inhibition (stimulation $<$ baseline). OGS in the Chr2 group resulted in five activated and two inhibited clusters. Activation was seen in the NAc, dorsal hippocampus and stria terminalis; secondary somatosensory cortex and caudate/putamen; globus pallidus, ventral pallidum, and amygdala; and periaqueductal gray. Inhibition was seen in the retrosplenial cortex, anterior cingulate gyrus and secondary motor cortex. The ROI analysis determined that the NAc of each rat in the Chr2 group was activated between the baseline and stimulation scans. Only Chr2 rats showed a significant increase (16% \pm 3) in BGluM in NAc ROI from baseline to stimulation scans ($p < 0.01$; group x intervention [F(1,15)=9.332, $p < 0.01$]). Locomotor measures determined only a significant main effect of time [F(4,56)=5.188, $p=0.001$]; as expected, rats were more active during the habituation sessions compared to the mPET sessions. Analysis of c-Fos

Mapping Brain Metabolic Connectivity in Awake Rats with MicroPET and Optogenetic Stimulation

Wednesday, Poster #46 (continued)

Panayotis Thanos

expression in the NAc following OGS found that c-Fos expression was greater in Chr2 rats compared to GFP rats [$F(1,8)=20.392$; $p<0.01$], and changes in brain glucose metabolism in the NAc (baseline vs. stimulation) and c-Fos expression were significantly correlated ($R=0.77$, $p<0.01$).

Conclusions: OGS of the NAc increased c-Fos expression and BGlUM in the region of stimulation, and these measures were correlated. This is consistent with fMRI results reporting BOLD increases in the area of stimulation (Lee and Deisseroth, 2012). We also observed increased metabolism in regions connected to the NAc including the basal ganglia (caudate, putamen, globus pallidus, and ventral pallidum) and limbic regions (amygdala, hippocampus). Interestingly, we showed decreased metabolic activity in the retrosplenial cortex (posterior cingulate gyrus) and anterior cingulate gyrus, which are regions that form part of the default mode network (DMN), which in conjunction with brain imaging findings in humans (Tomasi et al., 2009; Dang et al., 2012), suggests that activation of the NAc may facilitate DMN inhibition. These results demonstrate the feasibility of using mPET with FDG in conjunction with OGS to map connectivity in the awake rat brain.

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Fine-grained Working Memory Load Manipulation Reveals Absence of Normative Inverted-U Activation in Schizophrenia

Wednesday, Poster #123

Jared X. Van Snellenberg, Ragy R. Girgis, Christina Read, Judy L. Thompson, Jochen Weber, Tor D. Wager, Mark Slifstein, Jeffrey A. Lieberman, Anissa Abi-Dargham, Edward E. Smith
Columbia University College of Physicians & Surgeons

Background: Patients with schizophrenia exhibit serious and clinically relevant deficits in working memory (WM). However, investigations of WM in patients with schizophrenia using functional Magnetic Resonance Imaging (fMRI) have largely failed to reveal a consistent abnormality in brain activation in patients. One hypothesis is that patients exhibit a disordered relationship between the extent of activation in dorsolateral prefrontal cortex (DLPFC) and WM load, for example a left-shift in an inverted-U relationship (Callicott et al., 2003; Manoach, 2002, 2003), such that patients exhibit greater activation relative to healthy individuals at low WM loads and less activation at high loads. To test this hypothesis, we employed a version of the self-ordered working memory task (SOT) for use with fMRI that provides a finer-grained variation in WM load than existing tasks.

Methods: Thirteen patients with schizophrenia and eighteen control participants matched on age, gender, and parental socio-economic status performed the SOT during the acquisition of Blood-Oxygen Level Dependent fMRI images from a Philips 1.5 Tesla Intera scanner (2 s TR, 40 slices of a 64 x 64 plane, 3 mm isotropic voxels). In each trial of the SOT participants are presented with eight line drawings of 3D objects in an array. On each step of the trial the object positions are pseudo-randomly rearranged, and participants must select any object that they have not previously selected, thereby producing a gradual increase in WM load over the eight steps of each trial. Whole-brain fMRI activation data during correctly performed steps was analyzed in a two-way between-groups repeated measures ANOVA with factors Diagnosis (two levels) and Step (eight levels), using the Greenhouse-Geisser correction for non-sphericity. Regions showing a main effect of Step in either group were further analyzed in a random-effects polynomial regression to determine the shape of the change in activation over steps.

Fine-grained Working Memory Load Manipulation Reveals Absence of Normative Inverted-U Activation in Schizophrenia

Wednesday, Poster #123 (continued)

Jared X. Van Snellenberg

Results: Patients and controls exhibited above chance accuracy and monotonic declines in performance from steps two through eight, with patients also performing significantly worse than controls at these steps. Healthy controls exhibited a significant ($p < 0.05$, FDR corrected) negative quadratic polynomial (inverted-U) response to increasing WM load in the SOT in bilateral DLPFC, posterior parietal cortex (PPC), lateral occipital cortex, fusiform gyrus, and left putamen. Patients with schizophrenia exhibited no significant main effect of step in any brain region, even at a relaxed statistical threshold ($p > 0.25$, FDR corrected). Significant between-group differences in the pattern of activation was observed at a relaxed threshold ($p < 0.25$, FDR corrected) in bilateral DLPFC, right PPC, and right cuneus and fusiform gyrus.

Conclusions: The present findings support the hypothesis of an inverted-U relationship between DLPFC activation and WM load in healthy individuals, as proposed by Callicott et al. (2003) and Manoach (2002, 2003), and this relationship was also observed in several other brain regions known to be involved in WM. However, there was no evidence to suggest a left-shift in this inverted-U in patients with schizophrenia; rather, the normative inverted-U relationship was absent in patients. While specification of the functional significance of this inverted-U relationship remains somewhat speculative, the fact that healthy individuals maintained high levels of performance at later steps, and yet showed decreasing activation during correct performance in brain regions known to subservise WM, suggests that healthy individuals may have exhibited a flexible shift in strategy (e.g. to a familiarity-based long-term memory strategy) as their WM capacity was exceeded. Critically, patients with schizophrenia failed to show this shift at high WM load, raising the possibility that they either have a fundamental impairment that makes such a strategy shift a non-optimal approach to performing the task, or that they were exhibiting perseveration on the earlier-adopted strategy. This study is arguably the most comprehensive investigation of the impact of variation

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Fine-grained Working Memory Load Manipulation Reveals Absence of Normative Inverted-U Activation in Schizophrenia

Wednesday, Poster #123 (continued)

Jared X. Van Snellenberg

in WM load on brain activation in patients with schizophrenia and matched controls carried out to date, and reveals several new directions for research into the functional impairment underlying WM deficits in patients with schizophrenia.

BDNF Val66Met Modulates BOLD Response to Affective Instrumental Learning in Humans

Wednesday, Poster #205

Mbemba Jabbi, Brett Cropp, Tiffany Nash, Philip Kohn, Raghav Mattay, Shane Kippenhan, Bhaskar S. Kolachana, Daniel R. Weinberger, Karen F. Berman
National Institute of Mental Health

Background: Preclinical models implicate the BDNF Val66Met polymorphism in impaired fear extinction and anxiety phenotypes (Chen et al., 2006; Lipsky and Marini 2007), but the role of this genotype in human adaptive learning requiring both avoidance of adverse circumstances as well as attainment of rewarding experiences remains largely unknown. Here, we assessed this SNP's influence on the amygdala and hippocampus, two key regions involved in emotional regulation and learning (Ledoux 2000; Farinelli et al., 2006; Herry et al., 2008) during higher-order reinforcement learning whereby videos of fearful and happy expressions predicted choice-related monetary loss and gain respectively.

Methods: Thirty-three healthy participants (12 met carriers, 21 val homozygotes) underwent fMRI (at 3T; 16 channel head coil) while passively viewing dynamic happy, fearful, and neutral facial expressions. In addition, 61 participants including the 33 passive viewing cohort (21 met carriers, 40 val homozygote) underwent reinforcement learning during fMRI (3T, 16 channel head coil), while they 1) watched a cue video of emotional or neutral expression; 2) made a choice between two non-face pictures simultaneously presented, with one of the pictures portraying emotional content concordant with the preceding video; and 3) saw an outcome cue delineating monetary reward if the concordant picture was correctly chosen, or loss if the non-concordant picture was chosen. Preprocessing (8mm smoothing), first-level analysis with SPM5, and random-effects ANOVAs were performed to assess BOLD response to passive viewing and during establishment of cue-outcome association. We tested for regional specificity of Val66Met influence on BOLD response to higher-order emotional cues in amygdala and hippocampus, given their intimate inter-connectivity and collective mediation of learning and LTP and their role in regulation of emotions (Ledoux 2000; Dolan 2002; Farinelli et al., 2006; Herry et al., 2008). To this aim, we extracted

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BDNF Val66Met Modulates BOLD Response to Affective Instrumental Learning in Humans

Wednesday, Poster #205 (continued)

Mbemba Jabbi

percentage BOLD signal change from manually segmented whole amygdala and hippocampal regions of interest (ROI) for each emotional viewing condition separately for each of the 33 individuals who participated in passive viewing, and while they underwent affective reinforcement learning.

Results: Using a 2 *by* 2 *by* 3 repeated measures ANOVA (task [‘passive viewing vs. higher-order emotional conditioning’] *by* region [‘whole amygdala and hippocampus’] *by* valence) with Val66Met genotype as the between-subjects factor, we found a task *by* region *by* BDNF interaction ($F_{2,30} = 3.71$, $p = 0.032$). Whereas the hippocampal response was not affected by genotype, there was a decreased BOLD response to reinforced emotional cues in the amygdala of met carriers. To further assess valence-specific BDNF influence on neural coding of predictive emotional cues during reinforcement learning, we extracted BOLD signals measured during viewing of loss and gain predictive fear and happy cues from left and right amygdala and hippocampal ROIs in all 61 reinforcement learning participants. Using a 2 *by* 2 *by* 3 repeated measures ANOVA (region *by* hemisphere ‘left vs. right’ *by* valence) with BDNF Val66Met genotype as the between subjects factor, we found an interaction between region and hemisphere at $F_{2,58} = 18.21$, $p = 10^{-4}$, driven by a marked reduction in left amygdala BOLD signals. Importantly, no main effects of BDNF genotype and no interaction between genotype and valence was found on neural coding of predictive emotional cues, supporting a Val66Met influence on these regions that is not fear specific. The observed BOLD response pattern was in line with previous research (Soliman et al., 2010; Andero et al., 2012), in that met carriers showed a consistent overall decrease in BOLD response to reinforced emotional cues. To assess the behavioral utility of the Val66Met genotype, we used a 3 *by* 2 (valence *by* conditioning) repeated-measures ANOVA on choice-related performance scores, with BDNF as between-subjects factor. We found main effects of genotype ($F_{1,59} = 4.392$, $p = 0.040$) and valence ($F_{2,58} = 100.59$, $p < 10^{-5}$), with choice accuracy during

BDNF Val66Met Modulates BOLD Response to Affective Instrumental Learning in Humans

Wednesday, Poster #205 (continued)

Mbemba Jabbi

affectively-relevant instrumental choice behavior surpassing chance-level for the aversive and rewarding conditions only, and more so for the met carriers.

Conclusions: Here, we demonstrated that met carriers performed better by gaining more money while avoiding losses in the subsequent choice behavior. This behavioral pattern was associated with an overall decrease in amygdala BOLD response to facial cues, and this neural response pattern was shown to be specific to emotional cues carrying loss/gain predictive value. Together, these findings suggest an adaptive utility of the pronounced decrement in amygdala BOLD response found in the met carriers. By demonstrating that the BDNF Val66Met polymorphism mediates flexible adaptive behavior in both reward and aversive learning, these data may demonstrate a neurogenetic mechanism underlying emotionally meaningful adaptive behavior, and thereby provide a possible framework for understanding the neurogenetic correlates of mood and anxiety disorders.

2:30 p.m. – 6:30 p.m.

Hot Topics

Regency Ballroom 1 & 2

PL

“Erasing” a Cocaine-cue Memory in Mice: Potential Implications for Relapse to Drug Taking

Monday, Poster # 81

Sheena Josselyn, Hwa-Lin (Liz) Hsiang, Michel van den Oever, Chen Yan, Asim Rashid, Paul Frankland
University of Toronto

Background: One significant obstacle for the treatment of drug addiction is the high incidence of relapse to drug-taking following months, or even years, of abstinence. Exposure to stimuli that were previously associated with prior drug use can awaken powerful memories that may trigger drug craving and provoke a relapse. Therefore, understanding how animals learn and remember the association between a cue and a drug of abuse (such as cocaine) is a crucial step to develop more effective treatment strategies for preventing and treating relapse in humans. Here we examined the neural mechanisms that mediate how cues become associated with the rewarding properties of cocaine to determine if disrupting expression of this cue-reward memory can help prevent relapse.

Methods: CREB (cAMP/Ca²⁺ responsive element binding protein) is a transcription factor that has a well-documented role in neuronal plasticity and long-term memory formation. Previously we found that increasing levels of the transcription factor CREB in a subset of lateral amygdala (LA) neurons in mice enhanced the formation of a fear memory and that selectively ablating these neurons post-training essentially “erased” the fear memory (Han et al., *Science*, 2007, 2009). We took advantage of this approach to investigate whether LA neurons are also critically involved in a cocaine-cue associated memory. To assess cocaine-cue memory, we used the conditioned place preference (CPP) paradigm. In this task, an otherwise neutral environment is paired with cocaine administration. A second neutral environment is paired with saline administration. Drug-free mice are then given the opportunity to spend time in each of these environments. Mice that have learned and remember the association between the particular environment and cocaine spend more time in this drug-paired environment.

Results: To increase CREB function in a subset of LA neurons, we microinjected replication-defective herpes simplex virus (HSV) vectors encoding CREB or GFP (control) into the LA of mice. Increasing CREB in a small subset of

“Erasing” a Cocaine-cue Memory in Mice: Potential Implications for Relapse to Drug Taking

Monday, Poster # 81 (continued)

Sheena Josselyn

LA neurons during (but not after) conditioning (pairing cocaine with a neutral environment) enhanced cocaine-CPP memory. To determine if these LA neurons with increased CREB function comprised a crucial component of the “cocaine memory-trace”, we used inducible diphtheria toxin receptor (iDTR) transgenic mice to selectively ablate just these neurons after conditioning. Deletion of LA neurons with increased CREB function (but not a similar proportion of random neurons) blocked expression of a previously acquired cocaine-CPP memory. That is, we were able to disrupt expression of a cocaine CPP by simply ablating a small portion of LA neurons that overexpressed CREB during conditioning. In contrast to extinction training, the disruption of CPP produced by ablating neurons overexpressing CREB was resistant to reinstatement following a low priming dose of cocaine. These findings suggest that a critical component of the cocaine-CPP memory is essentially erased. Next, rather than (irreversibly) ablating these neurons overexpressing CREB, we took advantage of the DREADD (designer receptors exclusively activated by designer drug) system to temporarily inactivate neurons overexpressing CREB. hM4Di is an engineered receptor that is coupled to Gi protein; binding of hM4Di by clozapine-N-oxide (CNO), an otherwise pharmacologically inert compound, promotes neuronal inhibition. We microinjected viral vectors expressing both CREB and hM4Di and found that “silencing” neurons overexpressing CREB before CPP testing similarly inhibited the expression of cocaine CPP memory.

Conclusions: Our results indicate that, similar to a conditioned fear memory, a small population of LA neurons is critically involved in a cocaine-associated memory. Not only do the results of these studies inform us as to the biology underlying the development and expression of cue-cocaine associations, but, in the future, these findings could serve as a foundation for the development of new pharmacotherapies aimed at treating or even preventing drug relapse.

2:30 p.m. – 6:30 p.m.

Hot Topics

Regency Ballroom 1 & 2

PL

Markedly Reduced mGluR5 Receptor Binding in Smokers and Ex-smokers Determined by [¹¹C]ABP688 Positron Emission Tomography

Tuesday, Poster #73

Gregor Hasler, Funda Akkus, Simon M. Ametamey, Valerie Treyer, Cyrill Burger, Anass Johayem, Daniel Umbricht, Baltazar Gomez Mancilla, Judit Sovago, Alfred Buck
Psychiatric University Hospital, Bern, Switzerland

Background: Nicotine addiction is a major public health problem, resulting in primary glutamatergic dysfunction. We measured the glutamate receptor binding in the human brain and provided direct evidence for the abnormal glutamate system in smokers. Since antagonism of the metabotropic glutamate receptor 5 (mGluR5) reduced nicotine self-administration in rats and mice, mGluR5 is suggested to be involved in nicotine addiction.

Methods: We used positron emission tomography (PET) with the radiolabeled mGluR5 antagonist 3-(6-methyl-pyridin-2-ylethynyl)-cyclohex-2-enone-O-11C-methyl-oxime ([¹¹C]ABP688) (15), which binds with high selectivity to an allosteric site, to measure mGluR5 availability in 14 healthy subjects (non-smokers), 14 smokers, and 10 ex-smokers. The mean duration of nicotine abstinence in ex-smokers was 18.2 weeks (standard deviation, 14.2).

Results: We found a marked global reduction (20.6%; $p < 0.0001$) in the mGluR5 distribution volume ratio (DVR) in the gray matter in smokers. The most prominent reductions were found in the bilateral medial orbitofrontal cortex. Compared to non-smokers, ex-smokers had global reductions in the average gray matter mGluR5 DVR (12.4%; $p < 0.005$). In contrast, the differences in mGluR5 DVR in any brain region between smokers and ex-smokers did not reach statistical significance after Bonferroni correction. In smokers, age was positively correlated with mGluR5 DVR in most regions of interest, and the strongest correlations were found in the putamen. Clinical variables reflecting current nicotine consumption, dependence, and abstinence were not correlated with mGluR5 DVR.

Conclusions: These findings suggest that the reduced mGluR5 may not reflect a simple consequence of nicotine consumption but may represent a precondition

Markedly Reduced mGluR5 Receptor Binding in Smokers and Ex-smokers Determined by [11C]ABP688 Positron Emission Tomography

Tuesday, Poster #73 (continued)

Gregor Hasler

of nicotine dependence and/or a trait-like pathogenetic or compensatory change associated with nicotine addiction. This study encourages the development and testing of drugs against addiction that directly target the glutamatergic system.

2:30 p.m. – 6:30 p.m.

Hot Topics

Regency Ballroom 1 & 2

PL

Differentiating Neural Networks with Interleaved TMS-BOLD Imaging: Insight into Addiction

Wednesday, Poster #159

Colleen A. Hanlon, Melanie Canterbury, Joseph Taylor, Truman Brown, Mark S. George
Medical University of South Carolina

Background: Interleaved transcranial magnetic stimulation (TMS) and BOLD imaging in the MR environment provides us with a unique opportunity to probe neural circuitry. The purpose of the current study was to determine if, through the use of an optimized interleaved TMS-BOLD sequence in two cortical targets, we could differentially activate lateral and medial prefrontal cortex neural circuits.

Methods: Interleaved TMS/BOLD imaging data was acquired for a cohort of 15 healthy individuals and 15 cocaine users who received TMS in 2 runs with the coil positioned over the: 1) dorsolateral prefrontal cortex (DLPFC, EEG: F3), and 2) orbitofrontal/medial prefrontal cortex (OFC/MPFC, EEG: FP1)(Magstim MR-compatible coil). The TMS pulse started 100ms before the onset of the next TR (100% motor threshold). BOLD data was analyzed using standard parametric techniques. Additionally 5 participants were scanned twice to evaluate test-retest reliability.

Results: Among healthy controls, DLPFC TMS was associated with a significant elevation of BOLD signal in multiple dorsal cortical areas. In contrast, OFC/MPFC TMS was associated with a significant elevation of BOLD signal in multiple ventral medial cortical regions as well as limbic subcortical areas. The cocaine users demonstrated a similar pattern of activity, but had selective dysregulation in the OFC/MPFC network.

Conclusions: These novel data demonstrate that it is possible to differentially activate known cortical-subcortical networks through an optimized TMS/BOLD sequence over the DLPFC and the OFC/MPFC. Test-retest reliability is high in healthy controls and among cocaine users there is a selective deficit in OFC/MPFC circuit function. These data have important implications for both basic neuroscience research and in patient populations that may have pathology differentially affecting mesolimbic versus mesocortical circuitry.

Treatment of Depression with Botulinum Toxin A: A Randomized, Double-Blind, Placebo Controlled Trial

Wednesday, Poster #5

Eric Finzi, Norman Rosenthal
Chevy Chase Cosmetic Center

Background: In spite of advances in our understanding and treatment of major depressive disorder(MDD) , many patients fail to achieve remission . Recently, it has been proposed that inhibition of frowning could be used as a treatment for MDD(Finzi et al., 2006). Preliminary studies have suggested that botulinum toxin treatment of frown muscles may help depression (Finzi et al. 2006, Wollmer et al., 2012). The corrugator (frown) muscle plays an essential role in the facial expressions of anger and sadness. Charles Darwin first suggested that muscle contractions involved in the formation of facial expressions contribute to emotional states and mood; William James elaborated on this concept, which has been confirmed experimentally, and is now known as the facial feedback hypothesis. Darwin also recognized that severely depressed individuals show corrugator muscle overactivity, which may result in the “omega sign.” Botulinum toxin (BT) reversibly inhibits muscle contraction. When injected into the glabellar region, BT reversibly inhibits frowning for about three months. We have conducted a randomized, double-blind, placebo controlled trial of BT injection into the glabellar region as a treatment for MDD.

Methods: The study was IRB approved, and informed consent was given by all subjects. Male or female outpatients aged 18 to 65 years, with MDD, as diagnosed by the Structured Clinical Interview for Axis I DSM-IV Disorders (SCID), were eligible. Subjects were required to have a Montgomery-Asberg (MADRS) score ≥ 26 and a Clinical Global Impression – Severity (CGI) score ≥ 4 at screening Eligible subjects were randomly assigned at screening to receive either onabotulinumtoxinA(OBA) (Botox Cosmetic, Allergan) or placebo(PLB) (0.9%NaCl) injections in the glabellar region(Finzi et al.,2006). Women received 29 U of OBA and men, 40 U. All patients were assessed at randomization and after 3 and 6 weeks with the MADRS, Beck Depression Inventory II (BDI) and CGI. The primary outcome measure was response to treatment, as defined as a

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Hot Topics

Regency Ballroom 1 & 2

PL

Treatment of Depression with Botulinum Toxin A: A Randomized, Double-Blind, Placebo Controlled Trial

Wednesday, Poster #5 (continued)

Eric Finzi

$\geq 50\%$ decrease in MADRS score. Remission was defined as a MADRS score of 10 or lower along with a $\geq 50\%$ decrease in score. Secondary outcomes were response to treatment in scores on BDI and CGI. Subjects at rest and maximal frowns, were assessed photographically at the beginning and end of the study.

Results: 121 subjects were screened, of whom 84 subjects were randomized: 41 to OBA and 43 to placebo. Eight patients were excluded (4 patients in the OBA group for withdrawal of consent, and two in each group for protocol violations.) One OBA subject was lost to follow-up after injection. 33 subjects in the OBA group and 41 in the placebo group completed all three visits. The two groups did not differ significantly on any of the demographic or clinical baseline variables. 91% of the OBA, and 80% of the PLB subjects suffered from recurrent depression. The average number of antidepressants tried during subject lifetimes, were 2.2 for OBA, and 1.8 for PLB, and the mean duration of the current depressive episode was 27.9 months. As for the primary end point, MADRS scores at the six week visit versus baseline, there was a significant improvement in the OBA group compared to the PLB group; there was a 47.0% reduction in MADRS scores for OBA subjects, versus a 20.6% reduction for PLB (student's t test, $p < 0.0004$). The OBA group showed a significant clinical improvement in depression, compared to the PLB group, over time, as measured by MADRS score, (ANOVA, $f=9.7$, $p < 0.0028$, two-tailed); BDI-II score, (ANOVA, $f=5.7$, $p < 0.019$, two-tailed.); and CGI score (ANOVA, $f=15.3$, $p < 0.0002$, two-tailed.). The response rate for MADRS was 51.5 % vs. 14.6 %; $p < 0.0009$ Fisher's exact test. The remission rate, as judged by MADRS, was significantly higher in the OBA group, 27.3%, than in the PLB group, 7.3%, $p < 0.027$, Fisher's exact test. A decrease in the maximal ability to frown at 6 weeks (among all subjects) was correlated with MADRS response; $p < 0.01$; Spearman coefficient. In the OBA group, there was a trend towards greater response ($\geq 50\%$ decrease in MADRS score) with increasing baseline frown (N.S., $p < 0.07$).

Treatment of Depression with Botulinum Toxin A: A Randomized, Double-Blind, Placebo Controlled Trial

Wednesday, Poster #5 (continued)

Eric Finzi

Conclusions: This is the first randomized, double-blind and placebo -controlled clinical trial to show that a single treatment of the glabellar region with OBA induces a strong and sustained alleviation of symptoms in a broadly defined group of people with MDD. The results are consistent with those of our earlier pilot study (Finzi et al.) and the prior smaller controlled study of BT in patients with refractory depression. Our study is also the first to show that subjects treated with OBA went into remission at a significantly higher rate than placebo subjects. The mechanism of action of OBA in helping depression is unknown, but our results support the facial feedback hypothesis and suggest that it can be utilized therapeutically. The results also support the concept of *emotional proprioception* (Finzi, 2013) whereby the brain continuously monitors the relative valence of salient facial expressions, which may be an important influence on mood.

2:30 p.m. – 6:30 p.m.

Hot Topics

Regency Ballroom 1 & 2

PL

A Randomized Controlled Crossover Trial of Ketamine in Obsessive-Compulsive Disorder

Wednesday, Poster #7

Carolyn I. Rodriguez, Lawrence S. Kegeles, Amanda Levinson, Sue Marcus,
Helen Blair Simpson
Columbia University

Background: Obsessive-compulsive disorder (OCD) is a leading cause of illness-related disability (1). First-line OCD pharmacological treatments lead to limited symptom relief and typically have a lag time of 6-10 weeks before clinically meaningful improvement(2). Identifying more effective pharmacological treatments with faster onset of action would be a major advance. Medications thought to modulate the glutamate system are a promising new class of pharmacological agents for the treatment of OCD (3-8). Ketamine, a non-competitive N-methyl-D-aspartate (NMDA) receptor antagonist, modulates glutamate and has been shown to have rapid anti-depressant effects in multiple studies (9-15). A recent case study of a unmedicated individual with OCD without comorbid depression who was given ketamine (0.5mg/kg IV over 40 minutes) showed rapid anti-obsessional effects that persisted from 1 to 7 days post-infusion, long after the drug had cleared (16). A subsequent open trial of ketamine in ten individuals showed modest but significant improvement in OCD symptoms over days 1 to 3 following ketamine infusion compared to baseline; the majority of individuals with OCD in this study were taking multiple medications and had moderate to severe current comorbid depression (17). We investigated the effects of ketamine on individuals with OCD who were not currently on medications and did not have moderate to severe comorbid depression.

Methods: In a randomized, double-blind, placebo-controlled, crossover design, unmedicated adults (N=10) with OCD received two intravenous infusions: one of saline and one of ketamine (0.5mg/kg) over 40 minutes. These infusions were spaced at least 1 week apart; the order of each pair of infusions was randomized. To be eligible, participants were required to have at least moderate to severe OCD (Yale-Brown Obsessive-Compulsive Scale [YBOCS] score > 16) with no or mild depression (Hamilton Depression Rating Scale [HDRS-17] < 25), and endorse

A Randomized Controlled Crossover Trial of Ketamine in Obsessive-Compulsive Disorder

Wednesday, Poster #7 (continued)

Carolyn I. Rodriguez

near-constant intrusive obsessions (>8 hours per day) (18, 19). To assess rapid changes in obsessions, the OCD visual analogue scale (OCD-VAS) was used at baseline, at 26, 90, 110, and 230 minutes and daily for 7 days post-infusion (16). To assess both obsessive and compulsive symptoms, the YBOCS scale, designed to be used to assess OCD symptoms at 1 week intervals, was used at baseline and 7 days post-infusion. To monitor depressive symptoms, the HDRS-17 was used at baseline and 1 and 3 days post-infusion. Response rate of obsessions was defined as a minimum of 35% improvement in obsessions (as measured by the OCD-VAS), and response rate for OCD symptoms was defined as a minimum of 35% reduction in OCD symptoms (as measured by the YBOCS).

Results: All ten participants completed the study. At baseline, participants had moderate to severe OCD symptoms (mean YBOCS 27.1+/-3.4 SD, range: 22-34). On average, there was a significant rapid decrease in obsessions (as measured by OCD-VAS) which decayed over time and then reached a plateau. Responder rate (n=10) of obsessions (as measured by OCD-VAS) at post-infusion time points were as follows: 90% at 3 hours, 80% at 1 day, 60% at 2 days, 50% at 3 days, and 50% until day 7. Responder rate (n=10) for OCD symptoms (as measured by YBOCS) was 50% at day 7. Responder rate for OCD symptoms among the subset of patients (n=5) who got the ketamine infusion first (and thus the effects of ketamine could be evaluated at both day 7 and day 14), was 40% at day 14. At baseline, participants had minimal depressive symptoms (mean HDRS 4.2+/-5.6, range: 0-16). The average depressive symptoms of the 10 patients did decrease somewhat after the ketamine infusion (4.2 +/- 5.6 to 1.8 +/- 1.9, $F(2,17) = 3.38$, $p = 0.058$).

Conclusions: These data suggest that ketamine can rapidly relieve symptoms of OCD, and this effect can persist for at least one week in 50% of OCD patients with constant intrusive thoughts. A subset of individuals had relief for up to two weeks. Future research is needed to better understand the mechanism of ketamine's rapid

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Hot Topics

Regency Ballroom 1 & 2

PL

A Randomized Controlled Crossover Trial of Ketamine in Obsessive-Compulsive Disorder

Wednesday, Poster #7 (continued)

Carolyn I. Rodriguez

anti-obsessional effect and persistent reduction in OCD symptoms, long after the drug has cleared. These insights will help inform the development of new treatment strategies for individuals suffering with OCD.

Effects of Oxytocin on Social Cognition and Olfaction in Adults with Schizophrenia and Healthy Subjects

Monday, Poster #30

Josh Woolley, Brandon Chuang, Olivia Lam, Kate Rankin, Daniel H. Mathalon, Sophia Vinogradov
University of California, San Francisco

Background: Patients with schizophrenia (SCHZ) have multiple social cognitive deficits, including difficulty in recognizing facial emotion, interpreting paralinguistic cues (e.g. sarcasm) and understanding other's mental states (i.e., theory of mind). Patients also have impaired olfaction, which is associated with worse negative symptoms and decreased social motivation. Social cognitive and olfactory deficits are correlated with worse functional outcome and quality of life and currently there are few available treatments for these deficits in SCHZ. The neuropeptide oxytocin (OT) has multiple prosocial effects when administered intranasally in humans and offers a potential remedy for these social deficits. OT has been implicated in bonding and has shown promise in enhancing social cognition in SCHZ. Further, OT signaling has been implicated in socially relevant olfaction in animals. Therefore, we investigated the effects of intranasal oxytocin on social cognition and olfaction in patients with SCHZ and healthy subjects (HS).

Methods: We administered OT (40IU) and placebo (PL) intranasally to 22 male adult patients with SCHZ and 20 HS of similar age and educational level in a randomized, double-blind, cross-over, within-subject study, with the two days of testing separated by one week. We measured performance on The Awareness of Social Inference Test (TASIT), which uses short video clips of actors to assess subjects' ability to comprehend counterfactual statements from paralinguistic cues signaling white lies (*White Lie items*), sarcasm (*Sarcasm Items*), and to make judgments about the actors' thoughts (*Theory of Mind Items*). Olfactory thresholds were measured for lyral, clove, and anise oils using a modified Munich Olfaction Test. Subjects identified the bottle with the testing compound from amongst two mineral oil containing distracter bottles in an upward step procedure

2:30 p.m. – 6:30 p.m.

Hot Topics

Regency Ballroom 1 & 2

PL

Effects of Oxytocin on Social Cognition and Olfaction in Adults with Schizophrenia and Healthy Subjects

Monday, Poster #30 (continued)

Josh Woolley

with increasing geometric dilutions ($12.5 \times 10^{-6}\%$ to $20\% \text{ m}^3/\text{m}^3$). Paired t-tests were used for all comparisons and data are expressed as Mean \pm (S.E).

Results: OT administration to SCHZ patients (Age: 44.0 (10.0), Education: 13.5 (2.2)) improved overall performance on TASIT ($74\% \pm 2\%$ vs. $70\% \pm 2\%$, $p=0.02$), on *White Lie Items* ($77\% \pm 2\%$ vs. $71\% \pm 2\%$, $p=0.02$), *Sarcasm Items* ($70\% \pm 3\%$ vs. $65\% \pm 3\%$, $p=0.05$) and *Theory of Mind Items* ($77\% \pm 2\%$ vs. $72\% \pm 3\%$, $p=0.05$). In HS (Age: 36.0 (13.1), Education: 15.3 (1.9)), OT administration-induced changes on these scales did not reach significance. In SCHZ patients, OT led to enhanced detection of Lyral (but not Anise or Clove) at lower concentrations ($3 \times 10^{-5}\% \pm 1 \times 10^{-7}\% \text{ m}^3/\text{m}^3$ vs. $2 \times 10^{-4}\% \pm 1 \times 10^{-7}\% \text{ m}^3/\text{m}^3$, $p=0.03$). In HS, OT effects on olfactory thresholds did not reach significance. For TASIT, we divided subjects on a median split based on performance on the placebo day. The group of patients who scored poorly on the placebo day had greater OT-induced improvements on multiple measures (*Overall Items*: (OT-PL) $9\% \pm 2\%$ vs. $-1\% \pm 2\%$ $p=0.001$; *Sarcasm Items*: $16\% \pm 3\%$ vs. $-4\% \pm 2\%$ $p=0.0001$; *Theory of Mind Items*: $12\% \pm 3\%$ vs. $-3\% \pm 2\%$ $p=0.0001$). However, in HS this relationship did not reach significance (*Overall Items*: $4\% \pm 4\%$ vs. $-2\% \pm 1\%$ $p=0.2$; *Sarcasm Items*: $7\% \pm 5\%$ vs. $-6\% \pm 3\%$ $p=0.06$; *Theory of Mind Items*: $5\% \pm 5\%$ vs. $-2\% \pm 2\%$ $p=0.21$).

Conclusions: Our findings indicate that OT significantly improves SCHZ patients' ability to 1) interpret paralinguistic cues (e.g., white lies and sarcasm), and understand other's mental states (i.e., theory of mind), and 2) detect lyral at lower concentrations. The OT-induced improvement in social cognition is clinically significant because deficits in these domains are strong predictors of functional outcome in SCHZ and are currently difficult to treat. The OT-induced improvement in detection of lyral demonstrates that OT may be the first pharmacological agent to remediate the olfactory deficits in SCHZ. Furthermore, the selectivity of this effect for lyral fits with previous data indicating that

Effects of Oxytocin on Social Cognition and Olfaction in Adults with Schizophrenia and Healthy Subjects

Monday, Poster #30 (continued)

Josh Woolley

patients with SCHZ are selectively impaired at detecting lyral possibly due to cAMP signaling dysfunction and that OT can increase cAMP signaling *in vitro*. Finally, it appears that OT has differential effects depending on an individual's baseline ability in that subjects who score poorly on the placebo day have large significant improvements on performance when administered OT. The underlying mechanisms for this differential effect remain unknown, however, OT may be improving basic, early sensory processing, such as gaze to the eye region, which helps individuals with poor baseline social cognition. In sum, our data provide support for using OT as a pharmacological agent to remediate multiple social deficits in SCHZ. Larger studies focused on patients with SCHZ who have significant baseline deficits in social cognition are needed to confirm and extend our findings.

Monday At A Glance

6:45 AM – 8:00 AM	CDI Booster Session	Room 220
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7:00 AM – 8:00 AM	ACNP Leadership & Institute Directors Meeting	Room 207
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7:00 AM – 8:00 AM	JAMA Network Meeting	Diplomat Ballroom 4
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7:30 AM	Morning Break	Diplomat Ballroom Lobby
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8:00 AM – 11:30 AM	President's Plenary: Taking Stock of the Connectome	Grand Ballroom
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11:30 AM – 1:00 PM	FNIH Biomarkers Consortium Neuroscience Steering Committee	Room 312-313
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11:30 PM – 1:00 PM	Women's Luncheon	Great Hall 5 & 6
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11:30 AM – 1:30 PM	Lunch Buffet	Great Hall 1-4
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11:30 AM – 1:30 PM	Consortium on the Genetics of Schizophrenia (COGS)	Room 201
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1:30 PM – 3:00 PM	Distinguished Lecture – Jeffrey Friedman Leptin and the Biologic Basis of Obesity	Grand Ballroom
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Mini-Panel Sessions

3:00 PM – 4:15 PM	Renaissance in Opioid Biology: From Preclinical Concepts to Clinical Practice	Diplomat Ballroom 1 & 2
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4:15 PM – 5:30 PM	Interaction of Ontogeny and Environment in Adolescent Substance Abuse	Diplomat Ballroom 1 & 2
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PA-Panel MP-Mini Panel PL-Plenary SG-Study Group

Monday

Monday At A Glance (continued)

Panel Sessions

3:00 PM – 5:30 PM	Neuroplasticity Deficits in Neuropsychiatric Illness: New Targets for Cognitive Enhancement	Regency Ballroom 2
3:00 PM – 5:30 PM	Common Neural Mechanisms across Dimensions of Pediatric Psychopathology	Atlantic Ballroom 1
3:00 PM – 5:30 PM	Unraveling the Genetic Architecture of Mental Illness with Whole Genome Sequence Data	Regency Ballroom 3
3:00 PM – 5:30 PM	De-risking the Pathway of Treatment Development for Autism Spectrum Disorders	Regency Ballroom 1
3:00 PM – 5:30 PM	Links between Activity, Sleep and Mental Function: Translational Models	Atlantic Ballroom 3
3:00 PM – 5:30 PM	Glial Regulation of Synaptic Pathology: Novel Mechanisms of Neuropsychiatric Disease and Avenues for Repair	Atlantic Ballroom 2

5:30 PM – 7:30 PM	Poster Session I with Reception	Great Hall 1-4
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Study Groups

7:30 PM – 9:00 PM	'If We Thought our Field was in Trouble Before...' Is Ethical Mental Health Care Possible in the Second Decade of the 21st Century?	Atlantic Ballroom 1
7:30 PM – 9:00 PM	Practical, Societal, Ethical, and Legal Challenges for Modern Brain and Biobanking: Experiences from America and Europe	Regency Ballroom 1
7:30 PM – 9:00 PM	The Role of Corticotropin-Releasing Factor (CRF) in the Pathophysiology of Mood and Anxiety Disorders: A Tribute to Wylie Vale	Regency Ballroom 2
7:30 PM – 9:00 PM	NIMH Research Domain Criteria Project: How will the Criteria Work for Studies of Diagnosis and New Drug Development?	Atlantic Ballroom 2

PA-Panel MP-Mini Panel PL-Plenary SG-Study Group

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

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

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

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

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

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).

Connectomics, Psychiatry and Drug Development

Edward Bullmore

The University of Cambridge/GlaxoSmithKline (GSK)

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

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

Connectomics, Psychiatry and Drug Development

PL

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.

Making Sense of the Functional Connectomes

Bruce Rosen

Harvard University

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 NCRR 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

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

PL

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

Huda Akil

University of Michigan

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

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

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.

Leptin and the Biologic Basis of Obesity

Jeffrey Friedman (continued)

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

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*

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*

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

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

SG

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

Tuesday Morning At A Glance

7:00 AM – 8:30 AM	ACNP Education & Training Committee Meeting	Diplomat Ballroom 4
7:00 AM – 8:30 AM	ACNP Membership Advisory Task Force Meeting	Room 203
7:00 AM – 8:30 AM	American Journal of Psychiatry Editorial Board Meeting	Room 220
7:00 AM – 8:30 AM	CME Institute Executive Director's Meeting	Room 207
7:00 AM – 8:30 AM	SOBP Biological Psychiatry Journal Editors' Meeting	Room 202

7:30 AM	Morning Break	Grand Ballroom Lobby
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Panel Sessions

8:30 AM – 11:00 AM	Neuroscience and the Future of Psychiatric Diagnosis: Updates on Development of the Fifth Edition of Diagnostic and Statistical Manual of Mental Disorders	Regency Ballroom 2
8:30 AM – 11:00 AM	Developmental Programming of the Brain: Implications for Shared Mechanisms Across Neuropsychiatric Disorders	Regency Ballroom 3
8:30 AM – 11:00 AM	One Size Doesn't Fit All: Molecular Mechanisms Underlying Diverse Estradiol Signaling in the Brain	Regency Ballroom 1
8:30 AM – 11:00 AM	Optimizing Cognitive Interventions for Schizophrenia: Predictive Biomarkers and Pharmacologic Enhancement	Diplomat Ballroom 1 & 2
8:30 AM – 11:00 AM	The Developmental Trajectory of Cannabis Effects on Neurobiological Functioning	Atlantic Ballroom 2
8:30 AM – 11:00 AM	Multi-level Classification of Schizophrenia and Bipolar Disorder: New Evidence and Controversies	Atlantic Ballroom 3
8:30 AM – 11:00 AM	Immune Modulation of Neurodevelopment in Schizophrenia and Autism	Atlantic Ballroom 1

11:00 AM – 12:30 PM	Lunch Buffet	Great Hall 1-4
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11:00 AM – 12:30 PM	U19 Committee Meeting: Emory-MSSM-Baylor-SFVA	Room 220
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11:15 AM – 1:30 PM	Data Blitz Session	Regency Ballroom 2
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PA-Panel MP-Mini Panel PL-Plenary SG-Study Group

Tuesday Afternoon At A Glance

12:00 PM – 1:00 PM	ACNP Website Editors Meeting	Room 209
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1:30 PM – 3:00 PM	Associate Member Session: “Ask the Experts: Peer Review”	Regency Ballroom 1
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Mini-Panel Sessions

3:00 PM – 4:15 PM	Rescuing Novel Mechanisms: Minimizing Placebo Response and Optimizing Signal Detection in Proof of Concept Trials	Diplomat Ballroom 1 & 2
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4:15 PM – 5:30 PM	Exploring Therapeutic Use of Psilocybin; A Classic Hallucinogen	Diplomat Ballroom 1 & 2
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Panel Sessions

3:00 PM – 5:30 PM	New Perspectives on the Role of Glutamatergic Neurotransmission in Alcoholism and Drug Addiction	Regency Ballroom 1
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3:00 PM – 5:30 PM	High Anxiety: Endocannabinoid Regulation of the Stress Response and Emotional Behavior	Atlantic Ballroom 2
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3:00 PM – 5:30 PM	Longitudinal Neuroimaging of Emerging Substance Use: Brain Indicators of Early Risk and Effects of Use	Regency Ballroom 3
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3:00 PM – 5:30 PM	Neuropeptide Receptor Ligands in Psychiatric Diseases: New Hopes after Multiple Failures	Regency Ballroom 2
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3:00 PM – 5:30 PM	Are We at a Turning Point in Psychiatric Genetics?	Atlantic Ballroom 1
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3:00 PM – 5:30 PM	The Ups and Downs of AKT Signaling: A Nexus of Risk for Psychiatric Disorders	Atlantic Ballroom 3
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5:30 PM – 7:30 PM	Poster Session II with Reception	Great Hall 1-4
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6:00 PM – 11:00 PM	ACNP Committee Chairs Waiting Room	Room 318
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6:00 PM – 11:00 PM	ACNP Council Meeting	Room 319-320
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PA-Panel MP-Mini Panel PL-Plenary SG-Study Group

**Neuroscience and the Future of Psychiatric Diagnosis:
Updates on Development of the Fifth Edition of Diagnostic
and Statistical Manual of Mental Disorders**

Chair: David J. Kupfer

- 8:30 a.m. Autism Spectrum Disorder in DSM5
Edwin H. Cook
- 9:00 a.m. DSM-5 Schizophrenia Spectrum: Major Changes, Controversies,
and Linkage with the NIMH Research Domain Criteria
William Carpenter
- 9:30 a.m. Anxiety Disorders, Obsessive-compulsive and Related Disorders,
Trauma- and Stressor-Related Disorders, and Dissociative
Disorders: Changes for DSM-5
Katharine A. Phillips
- 10:00 a.m. A Potential Biomarker for Addiction
Charles P. O'Brien
- 10:30 a.m. Discussant: *Darrel A. Regier*

PA

8:30 a.m. – 11:00 a.m.

Panel

Regency Ballroom 3

Developmental Programming of the Brain: Implications for Shared Mechanisms Across Neuropsychiatric Disorders

Chair: Jill M. Goldstein

Co-Chair: Paul J. Harrison

- 8:30 a.m. Genome-wide Analysis Identifies Loci with Shared Effects on Five Major Psychiatric Disorders
Jordan W. Smoller
- 9:00 a.m. Shared Fetal Programming of Sex Differences in Stress Response Circuitry and Endocrine Deficits in Schizophrenia and Depression: Shared Mechanisms but Different Disorders
Jill M. Goldstein
- 9:30 a.m. Long-term, Sex-specific Effects of Developmental Exposure to Excess Glucocorticoids on Gene Expression in the Hypothalamus
Robert J. Handa
- 10:00 a.m. Trans-generational Effects of Endocrine Disrupting Compounds on Brain and Behavior
Emilie Rissman
- 10:30 a.m. Discussant: *Paul J. Harrison*

PA

One Size Doesn't Fit All: Molecular Mechanisms Underlying Diverse Estradiol Signaling in the Brain

Chair: David Rubinow

- 8:30 a.m. Roles of ER β in the CNS
Jan-Ake Gustafsson
- 9:00 a.m. Development of Ligands for Estrogen Receptor Beta and the Genomic vs. Non-Genomic Pathway: Appreciating and Exploiting the Many Dimensions of Activity and Selectivity
John Katzenellenbogen
- 9:30 a.m. Acute Estrogen Modulation of Synapses in the Hippocampus
Catherine Woolley
- 10:00 a.m. Serotonin Transporter Function: Interaction among Ovarian Steroids and Antidepressants
Alan Frazer
- 10:30 a.m. Discussant: *Tracy Bale*

8:30 a.m. – 11:00 a.m.

Panel

Diplomat Ballroom 1 & 2

Optimizing Cognitive Interventions for Schizophrenia: Predictive Biomarkers and Pharmacologic Enhancement

Chair: Neal R. Swerdlow

- 8:30 a.m. Predictors of Cognitive Improvement after “Neuroplasticity-based” Computerized Cognitive Training in Schizophrenia
Sophia Vinogradov
- 9:00 a.m. Neuroanatomical Predictors of Response to Cognitive Remediation
Matcheri Keshavan
- 9:30 a.m. Combining a Cognitive Enhancer and Cognitive Training: Proof of Principle and Potential Complexities in Real-life
Shitij Kapur
- 10:00 a.m. Memory Consolidation Deficits in Schizophrenia and the Combination of D-cycloserine with Cognitive Remediation
Donald Goff
- 10:30 a.m. Discussant: *Deanna Barch*

PA

The Developmental Trajectory of Cannabis Effects on Neurobiological Functioning

Chair: Barbara J. Mason

Co-Chair: Yasmin Hurd

- 8:30 a.m. Cannabis and the Adolescent Brain: Differentiating Vulnerability from Pathology
Dan I. Lubman
- 9:00 a.m. Trajectory of Adolescent THC Exposure on Mesocorticolimbic Molecular, Epigenetic and Structural Modifications: Transgenerational Effects
Yasmin Hurd
- 9:30 a.m. Δ 9-tetrahydrocannabinol Impairs Reversal Learning and Visuospatial Associative Memory in Rhesus Macaques
Michael Taffe
- 10:00 a.m. Effects of Cannabis Abuse on the Functional Brain Architecture for Visual Learning and Recognition Memory: Psychopharmacological and Developmental Evidence before and after Sustained Abstinence
Frank Haist
- 10:30 a.m. Discussant: *Deborah Yurgelun-Todd*

8:30 a.m. – 11:00 a.m.

Panel

Atlantic Ballroom 3

Multi-level Classification of Schizophrenia and Bipolar Disorder: New Evidence and Controversies

Chair: Michael Davidson

- 8:30 a.m. Cognitive Heterogeneity in Bipolar Disorder: Implications for Overlap with Schizophrenia
Katherine E. Burdick
- 9:00 a.m. Cognitive and Functional Deficits in Schizophrenia and Bipolar Disorder vary by Psychosis Presence and History
Christopher R. Bowie
- 9:30 a.m. MRI can be used to Differentiate Schizophrenia Patients from Those with Bipolar Disorder: Clinical and Theoretical Implications
Rene Kahn
- 10:00 a.m. Developmental Trajectories in Schizophrenia and Bipolar Disorder: Evidence for Distinct Etiologies
Michael Davidson
- 10:30 a.m. Discussant: *Avi Reichenberg*

PA

Immune Modulation of Neurodevelopment in Schizophrenia and Autism

Chair: Alan Brown

Co-Chair: Karoly Mirnics

- 8:30 a.m. Novel Roles for Immune Molecules in Early Postnatal Cortical Development: Implications for Schizophrenia and Autism Spectrum Disorders
Kimberley McAllister
- 9:00 a.m. Neuroimmune Changes in a Mouse Model of the Maternal Infection Risk Factor for Schizophrenia and Autism
Paul H. Patterson
- 9:30 a.m. Elevated Maternal C-Reactive Protein and Autism in a National Birth Cohort
Alan Brown
- 10:00 a.m. Neuroimmune Changes in the Brain of Subjects with Schizophrenia or Autism
Karoly Mirnics
- 10:30 a.m. Discussant: *John H. Gilmore*

11:15 a.m. - 1:30 p.m.
Data Blitz Session
Regency Ballroom 2

Data Blitz Session

Chair: William Carlezon

This session is comprised of rigorously timed 5 minute presentations by 17 young investigators.

- 11:15 a.m. Early Exposure to Antidepressants does not Recapitulate Constitutive Serotonin Transporter Deficiency
Anne M. Andrews
- 11:22 a.m. Activity-dependent Phosphorylation of MeCP2 T308 Regulates Interaction with NCoR Co-repressor Complex
Daniel H. Ebert
- 11:29 a.m. A Potential Mechanism of Behavioral Alteration by Genome Diversification: The Role of Neural MILI/piRNA Complexes on De Novo L1 Retrotransposition
Daisy Lin
- PL 11:36 a.m. Top-down Control of Raphe Circuits in Affective Resilience: Key Role of Raphe GABA Interneurons
Olivier Berton
- 11:43 a.m. Compared to What? Reappraising the Early Brain Overgrowth Hypothesis in Autism
Armin Raznahan
- 11:50 a.m. Allele-specific DNA deMethylation in FKBP5: A Molecular Mediator of Gene x Environment Interactions with Childhood Trauma
Tortsen Klengal
- 11:57 a.m. Differential Control of Learning and Anxiety along the Dorsal-ventral Axis of the Dentate Gyrus
Mazen A. Kheirbek

- 12:04 p.m. Broader Autism Phenotype: Relationships between Maternal/
Paternal BAP, Parental SSRI Treatment, WB 5-HT and Child's
Autism Symptoms
Suma Jacob
- 12:11 p.m. The Functional Significance of Antipsychotic-related Cortical
Thinning in First Episode Schizophrenia
Tyler A. Lesh
- 12:18 p.m. Imaging the Sensitivity of [123I]5-IA-85380 to Increases in
Acetylcholine at the Beta2-nicotinic Acetylcholine Receptors in
Human Subjects
Irina Esterlis
- 12:25 p.m. Self-regulation of Amygdala Activity with Real-time fMRI
Neurofeedback in Patients with Depression
KyMBERLY Young
- 12:32 p.m. Cannabinoid Facilitation of Extinction Recall via Increased
Recruitment of Prefrontal-hippocampal Circuitry in Healthy
Humans
Christine A. Rabinak
- 12:39 p.m. A Brief Monetary Progressive Ratio Task Predicts Clinical
Amotivation and Ventral Striatum Activation in Schizophrenia
Daniel Wolf
- 12:46 p.m. The Neurosteroids Allopregnanolone and DHEA Enhance
Emotion Regulation Neurocircuits and Modulate Memory for
Emotional Stimuli
Rebecca K. Sripada
- 12:53 p.m. Neuronal Signatures of Self-control in Anterior Cingulate Cortex
Benjamin Hayden

11:15 a.m. - 1:30 p.m.

Data Blitz Session

Regency Ballroom 2

- 1:00 p.m. Impaired Reward Responsiveness during Nicotine withdrawal
in Rats and Humans Assessed in a Translational Behavioral
Procedure
Andre Der-Avakian
- 1:07 p.m. Long Acting Injectable vs. Oral Antipsychotics in Schizophrenia:
A Systematic Review and Meta-analysis of Mirror-image Studies
Taishiro Kishimoto

PL

Early Exposure to Antidepressants does not Recapitulate Constitutive Serotonin Transporter Deficiency

Monday, Poster #64

Stefanie Altieri, Hongyan Yang, Hannah O'Brien, Julie G. Hensler, Anne M. Andrews
University of California, Los Angeles

Background: Administration of serotonin transporter inhibiting antidepressants (SSRIs) during a critical early postnatal period in rodents is postulated to reproduce changes in behavior arising from constitutive reductions in serotonin transporter (SERT) expression. Both animal models have medical significance related to neonatal exposure to SSRIs and differential SERT expression associated with human *Sert* gene polymorphisms, respectively.

Methods: We investigated the effects of postnatal administration of escitalopram (SCIT) or fluoxetine *vs* constitutive SERT deficiency in mice on emotion-related behaviors, presynaptic 5-HT_{1A} receptor expression and function, and extracellular serotonin levels in adolescence and late into adulthood. SSRIs were administered daily during postnatal days 5-21. Behavior was assessed in the elevated plus maze, open field, forced swim test, and sucrose preference test. Thermic responses to the 5-HT_{1A} receptor agonist 8-OH-DPAT were investigated. *In vivo* microdialysis and zero-net-flux analysis were used to evaluate changes in extracellular serotonin levels.

Results: Increased anxiety-related behavior, which is highly characteristic of SERT-deficient mice and humans with low-expression *Sert* alleles, was notably absent or potentiated in mice postnatally exposed to SSRIs. Furthermore, whereas 5HT_{1A}-mediated decreases in body temperature were attenuated in SERT-deficient mice, SSRI-treated mice showed pronounced hypothermia after 8OHDPAT. Moreover, postnatal treatment with SCIT resulted in 5-HT_{1A} autoreceptor *hypersensitivity*. Extracellular serotonin levels have been shown to be elevated in mice with constitutive reductions in SERT, however, mice exposed to SSRIs during early postnatal development showed reduced extracellular serotonin levels as adults.

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Early Exposure to Antidepressants does not Recapitulate Constitutive Serotonin Transporter Deficiency

Monday, Poster #64 (continued)

Anne M. Andrews

Conclusions: Transient *vs* constitutive SERT deficiency produces opposing and long-lasting changes in regulation of extracellular serotonin and 5-HT_{1A} autoreceptor function evident as early as adolescence. Persistent changes in presynaptic serotonergic circuitry are hypothesized to contribute to differential emotional phenotypes associated with these two models. Ongoing work is aimed at investigating mechanisms of differential serotonin and 5HT_{1A} regulation. These findings have important implications for antidepressant use during pregnancy and neonatal life in humans, which corresponds to the early postnatal period in rodents. They further clarify genetic influences associated with differential SERT expression regarding effects on behavior and underlying neurotransmission.

PL

Activity-dependent Phosphorylation of MeCP2 T308 Regulates Interaction with NCoR Co-repressor Complex

Monday, Poster #77

Daniel H. Ebert, Michael E. Greenberg

Massachusetts General Hospital and Harvard Medical School

Background: Rett syndrome (RTT) is a neurodevelopment disorder with features of autism that is caused by mutations in *MeCP2*. In addition, less severe mutations in *MeCP2* can lead to a wider spectrum of neuropsychiatric disorders, including autism and psychotic spectrum disorders. MeCP2 is a nuclear protein that binds DNA at methylated cytosines and represses transcription. In neurons, MeCP2 is expressed at high levels, stoichiometrically equivalent to core histones and is bound broadly across the genome. The molecular mechanisms of how loss of MeCP2 leads to RTT are not well understood. Neuronal activity triggers the phosphorylation of MeCP2 at S421. While S421A knock-in mice have defects in synapse development and behavior, the mutation had no detected effect on transcription. In addition, the proximal molecular impact of phosphorylation at S421 on MeCP2 is not known. Mass spectrometry studies have revealed many additional sites of phosphorylation in MeCP2; however, no other phosphorylation site has reproducibly been shown to be induced by neuronal activity. We hypothesized that multiple post-translational modifications of MeCP2, bound broadly across the genome, dynamically regulate its activity, modifying transcription and chromatin.

Methods: To identify novel sites of activity-dependent phosphorylation, we used phosphotryptic mapping of MeCP2 derived from ³²P-orthophosphate-labeled primary cortical neurons that had been left untreated or membrane-depolarized. To identify the sites of phosphorylation that correspond to the phospho-peptide spots that appeared with membrane-depolarization, we generated phosphotryptic maps of MeCP2, wildtype or with missense mutations at putative sites of phosphorylation, which had been phosphorylated using *in vitro* kinase assays. We generated phospho-site specific antibodies to each site of phosphorylation. We used these antibodies in Western blotting to determine if various stimuli in neuronal cell culture, or *in vivo*, induce the phosphorylation at each site in MeCP2.

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Activity-dependent Phosphorylation of MeCP2 T308 Regulates Interaction with NCoR Co-repressor Complex

Monday, Poster #77 (continued)

Daniel H. Ebert

We used synthetic peptides in pull-down assays and co-immunoprecipitation assays to determine if phosphorylation of T308 altered MeCP2's ability to bind co-factors. We used organotypic hippocampal cultures biolistically transfected with MeCP2 variants to determine the role of phosphorylation of MeCP2 T308 in regulating dendritic arborization.

Results: Using phosphotryptic mapping, we found multiple sites of activity-induced phosphorylation of MeCP2. Phosphorylation of these sites are differentially induced by neuronal activity, brain-derived neurotrophic factor, or agents that elevate the intracellular level of cAMP, suggesting that MeCP2 functions as an epigenetic regulator of gene expression that integrates diverse signals from the environment. By Western blotting with the phospho-site specific antibodies to MeCP2 pT308, we find that the phosphorylation of MeCP2 T308 is induced by neuronal activity upon calcium influx into neurons via L-type calcium channels and NMDA receptors. We find that the common RTT missense mutations at R306, by disrupting the basophilic kinase recognition motif, prevent phosphorylation of MeCP2 T308. Phosphorylation of MeCP2 T308 abrogates an interaction of MeCP2 with NCoR co-repressor complex and impairs the ability of MeCP2 to provide transcription repression. We find that phosphorylation of MeCP2 T308 regulates dendritic arborization, a phenotype altered in RTT.

Conclusions: These findings indicate that neuronal activity induces phosphorylation of MeCP2 at T308 and that phosphorylation at this site disrupts an interaction with NCoR co-repressor complex and regulates MeCP2's ability to mediate transcription repression. The NCoR co-repressor complex contains HDAC3, a histone deacetylase. The regulated interaction between MeCP2 and NCoR may modulate histone acetylation in response to neuronal activity to control transcription in neurons. The phosphorylation of T308 has not been observed previously in mass spectrometry studies from many different laboratories, indicating the utility of phosphotryptic mapping in identifying sites

Activity-dependent Phosphorylation of MeCP2 T308 Regulates Interaction with NCoR Co-repressor Complex

Monday, Poster #77 (continued)

Daniel H. Ebert

of activity-dependent phosphorylation. The common RTT missense mutations at R306 both disrupt an interaction with NCoR and prevent experience triggered phosphorylation of MeCP2 at T308. The loss of this regulated interaction between MeCP2 and the NCoR co-repressor complex may underlie critical aspects of RTT. Investigation of activity-dependent phosphorylation of MeCP2 may help identify targets for novel therapeutics for RTT and the broader spectrum of neuropsychiatric disorders caused by mutations in *MeCP2*.

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Data Blitz Session
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A Potential Mechanism of Behavioral Alteration by Genome Diversification: The Role of Neural MILI/piRNA Complexes on De Novo L1 Retrotransposition

Monday, Poster #114

Daisy Lin, Jay A. Gingrich
Columbia University/New York State Psychiatric Institute

Background: Mobile retrotransposable elements, such as the long interspersed element (LINE) L1, can insert copies of themselves throughout the genome and influence the expression of nearby genes. This is one of the mediators of genome diversification. Recent findings show that L1 element insertion can occur in dividing neurons, resulting in somatic cell diversification and possibly neuropsychiatric disorders such as Rett syndrome and schizophrenia. Our group is interested in developmental pathways leading to neuropsychiatric disorders. As such, while examining mechanisms of paternal influence on offspring behavioral outcomes, we discovered a possible role of the MILI/piRNA system as mechanism of neural L1 suppression. The mouse MILI gene is a member of the well-conserved PIWI family of proteins that are expressed across phylogenetically diverse species. Canonically, the MILI/piRNA complex has been shown to suppress L1 activity in the male germline, but a role for regulating L1 expression in dividing neurons has not been established. We hypothesized that there is a MILI/piRNA system in the brain and it has a direct impact on the behavior of the mouse by the regulation of L1 activity.

Methods: The expression of MILI in the mouse brain was detected by: real-time PCR, western blot hybridization and *in situ* hybridization (data taken from Allen Brain Atlas). Brain samples from wildtype (WT) and MILI^{+/-} mice of C57BL/6 background were used to compare MILI expression change between the two sets of samples. In order to understand the impact of neural MILI on neuronal L1 expression, semi-quantitative reverse transcription PCR probing the expression of a retrotransposition competent L1 transcript from the L1 A subfamily was performed. To examine whether there are changes in the behavior of these two sets of mice, our breeding approach was to mate C57BL/6 MILI^{+/+} and +/- sires (but not -/- sires, since they are sterile) with WT C57BL/6 females. We then

A Potential Mechanism of Behavioral Alteration by Genome Diversification: The Role of Neural MILI/piRNA Complexes on De Novo L1 Retrotransposition

Monday, Poster #114 (continued)

Daisy Lin

examined several different behavioral dimensions using an array of behavioral tests such as: the open field, light-dark choice test, social interaction (SI), elevated plus maze and forced swim test (FST).

Results: Contrary to what is generally thought to be a male mouse germline specific gene, here we show that, MILI, is also expressed in the mouse brain. Both MILI mRNA and protein expression are significantly reduced in the brains of MILI^{+/-} compared to MILI^{+/+} mice. We further observed that L1 expression is significantly increased in the brains of MILI^{+/-} offspring compared to MILI^{+/+} offspring of the same fathers. We observed that MILI^{+/-} offspring had depression and SI related behavioral changes compared to MILI^{+/+} offspring (demonstrated by increased floating duration during FST and increased time spent with social target during SI test, respectively). It could be argued that this change in MILI^{+/-} offspring is due to inheritance of genetic diversity that arose in the germline of MILI^{+/-} fathers, in this case we would observe changes in all offspring of MILI^{+/-} fathers compared to offspring of MILI^{+/+} fathers. However, changes in behavior were only observed in MILI^{+/-} offspring, but not MILI^{+/+} offspring of the same MILI^{+/-} fathers. Based on the data, we now know that changes in the behavior of MILI^{+/-} offspring is due to a direct effect of the MILI gene and independent of paternal germline MILI expression.

Conclusions: In the course of examining the effect of reduced paternal MILI expression on offspring behavior, we unexpectedly observed an independent effect of inheriting a null MILI allele on offspring behavior. This observation surprised us because MILI activity (if indeed testes specific) should not affect the brain. Here, we show that MILI is expressed in the brain. We wondered whether the MILI/piRNA complex might also function in dividing neurons. If so, the MILI haploinsufficient mice might exhibit behavioral differences due to an increase in *de novo* L1 insertions. Indeed, we observed increased L1 expression in the

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A Potential Mechanism of Behavioral Alteration by Genome Diversification: The Role of Neural MILI/piRNA Complexes on De Novo L1 Retrotransposition

Monday, Poster #114 (continued)

Daisy Lin

brains of MILI^{+/-} mice. Together, these observations will allow future work to directly assess the presence of piRNA species in neurons and determine whether MILI/piRNA complexes control *de novo* L1 insertion in neurons. Somatic L1 insertions in neural populations has been proposed as a mechanism for generating behavioral diversity as well as a potential etiology for neuropsychiatric disorders. A better understanding of the role of the MILI/piRNA system in L1-mediated neuronal genome diversification may lead to new insights into the origins of certain neuropsychiatric disorders as well as new preventative strategies.

PL

Top-down Control of Raphe Circuits in Affective Resilience: Key Role of Raphe GABA Interneurons

Monday, Poster #115

Collin Challis, Julie Espallergues, Sheryl Beck, [Olivier Berton](#)
University of Pennsylvania

Background: Imaging studies have revealed dysfunctions in the ventromedial prefrontal cortex (vmPFC) of patients with major depressive disorder (MDD). Deep brain stimulation (DBS) targeting the vmPFC is therapeutic in patients suffering from treatment resistant depression and produces antidepressant-like responses in rodents. Lesion studies in rodents suggest that serotonin (5-HT) partly mediates this antidepressant-like therapeutic activity of vmPFC DBS. The top-down control exerted by vmPFC over raphe circuits has also been implicated in stress coping and the expression of affective resilience. Underlying circuit mechanisms are poorly understood. The median and dorsal Raphe Nuclei (RN) receive strong excitatory inputs from the vmPFC. Although the RN contains a large proportion of 5-HT neurons, previous reports suggest that excitatory mPFC inputs preferentially synapse onto GABAergic interneurons. Given their position as primary postsynaptic target for vmPFC inputs in the RN, we hypothesized that Raphe GABA interneurons may be pivot to regulate vmPFC-RN connectivity during stress and the expression of affective resilience

Methods: We used combined genetic, electrophysiological and behavioral approaches to dissect the function of RN GABA interneurons in the social defeat (SD) mouse model of depression. Reporter mice with fluorescently-tagged GABA (GAD-Tomato) or 5-HT neurons (Pet1-tomato) were exposed to 10 days of social defeat and were segregated into resilient and vulnerable subpopulations based on social avoidance tests. SD-induced cFos was mapped using immunohistochemistry. Whole cell recordings of genetically identified neurons were conducted to characterize SD-induced changes in intrinsic properties of RN neurons and their synaptic inputs. mPFC-RN connectivity was assessed morphologically using viral-mediated track tracing and functionally using optogenetic stimulation and cFos mapping. To examine the role of RN excitatory inputs, Chr2 was targeted to vmPFC pyramidal neurons using an AAV vector under the control CamK2a

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Top-down Control of Raphe Circuits in Affective Resilience: Key Role of Raphe GABA Interneurons

Monday, Poster #115 (continued)

Olivier Berton

promoter. Axon terminals of transduced neurons were photostimulated locally in the DR. To examine the role of local 5-HT and GABA neurons in the expression of vulnerable and resilient behavioral phenotypes, the proton pump Arch was targeted to GABA or 5-HT neurons thereby allowing selective photosilencing of each cell populations. Photostimulation and photosilencing were conducted in freely moving mice, either during the sensory phase of social defeat training or during social interaction tests.

Results: We found GABA neurons to be preferentially activated over 5HT neurons after repeated experiences of SD. cFos induction by SD was significantly greater in stress-resilient than vulnerable mice and was topographically distributed in the RN. A strikingly similar pattern of activation was observed following direct photostimulation of vmPFC terminals in the RN. Using AAV-mediated anterograde tracing we observed a topographical overlap between the distribution of cFos expressing GABA neurons in the RN and the distribution of mPFC terminals, a result suggesting that vmPFC inputs drive GABAergic activation in RN during defeat stress and the degrees of this mPFC-driven GABAergic activation predicts subsequent social avoidance. In contrast to cFOS data, whole-cell electrophysiology of genetically identified GABA neurons revealed a diminished glutamatergic input in resilient mice. These results suggest that repeated phasic activation of RN GABA neurons during SD in resilient mice may trigger neuroadaptations that result in a tonic reduction of glutamatergic synaptic inputs. Lastly, we found that photosilencing DRN GABA neurons during the sensory contact period that follows physical defeat but not social interaction test, promoted the expression of a resilient phenotype and fully prevented social avoidance.

Conclusions: These results highlight a key role for DRN GABAergic neurons in expression of resilience to social defeat and stress induced neuroplasticity of mPFC raphe circuits.

Compared to What? Reappraising the Early Brain Overgrowth Hypothesis in Autism

Monday, Poster #125

Armin Raznahan, Rhoshel Lenroot, Audrey Thurm, Marta Gozzi, Sarah Spence, Susan Swedo, Jay Giedd
National Institute of Mental Health

Background: The presence of a robust association between Autism Spectrum Disorder (ASD) and early brain overgrowth (EBO) is widely accepted, prominent in lay understanding of the condition, and continues to influence the cutting edge of ASD research. The bulk of published data regarding early brain growth in ASD comes from studies of head circumference (HC), an excellent proxy for brain size in early childhood. Two developments since last systematic review of EBO studies in ASD urge reappraisal of the evidence base behind the influential notion in ASD research however: (i) multiple independent studies outside the field of ASD research have found that large contemporary samples of typically developing children appear to show EBO relative to HC reference norms (HCRNs) that have been used by several seminal EBO reports in ASD, and (ii) multiple new longitudinal tests of the EBO hypothesis have been published which build on earlier work by using larger sample sizes and contrasting HC growth in ASD with locally recruited controls (LRCs) as well as HCRNs.

Methods: We systematically review all published HC tests of the EBO hypothesis in ASD (34 studies encompassing ~ 3k ASD and 60k LRC participants), and analyze new data from a cohort of 57 preschool-aged male Caucasian children (35 ASD, 22 LRCs) with ~ 330 longitudinal HC measures between birth and age 18 months. Our study (i) distinguishes cross-sectional analyses of mean HC in ASD within a given age-range from longitudinal analyses that can measure brain size change, and (ii) assesses the dependence of evidence for EBO on the type of control data with which HC data in ASD are compared. We supplement traditional sources of HCRNs in ASD research [such as the Center for Disease Control (CDC)] with recently published “Primary Care Norms” (PCNs): the largest (~500k HC measures between birth and 18 months) contemporary set of US-based HCRNs.

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Compared to What? Reappraising the Early Brain Overgrowth Hypothesis in Autism

Monday, Poster #125 (continued)

Armin Raznahan

Results: Systematic Review: The majority ($> 65\%$) of HC studies in ASD are cross-sectional. Of the 11 existent longitudinal HC studies, 10 have been published since the EBO hypothesis was last subjected to systematic review. 85% of all HC studies in ASD make use of HCRNs. Cross-sectional studies that do not find evidence for brain enlargement in ASD tend to include a comparison with LRCs rather than solely relying on HCRNs ($p=0.0006$), and tend to use smaller age-ranges for calculating mean HC ($p=0.03$). All published comparisons of HCRN-defined macrocephaly rate in ASD vs. LRC groups have been negative. Elevated macrocephaly rates in ASD relative to HCRN-defined null of 3% vary 6-fold, with older HCRNs tending to identify higher rates ($p=0.04$). Over 85% of all longitudinal HC tests of the EBO hypothesis have compared ASD data to CDC HCRNs and identified rapid HC centile increases in ASD between birth and ~age 12 months. In contrast, most studies comparing HC growth between ASD participants and LRCs do not find evidence for EBO in ASD during the first year of life. By transforming existing HC reports into a common CDC reference frame we confirm the well-replicated pattern of EBO in ASD relative to CDC HCRNs, but show that the timing of this EBO is almost perfectly recapitulated by (i) 2012 PCN HCRNs which incorporate $> 500k$ HC measures from typically developing children in the US, *and* (ii) a weighted mean summary of HC growth for all LRCs included in ASD research. New Data Analysis: We did not find any cross-sectional differences in raw HC between ASD and LRCs at any pediatric surveillance time-point in the first 24 months of life, or group differences in raw HC change between time-points. Both ASD and LRC groups showed abnormally accelerated HC growth relative to CDC HCRNs, but had a stable mean PCN HC centile that remained within normal ranges between birth and 24 months.

Conclusions: By combining systematic review with analysis of new data and comparisons across multiple HCRNs we find several lines of evidence that oppose the hypothesis of EBO in ASD as currently formulated. Specifically

Compared to What? Reappraising the Early Brain Overgrowth Hypothesis in Autism

Monday, Poster #125 (continued)

Armin Raznahan

(i) EBO in ASD relative to CDC appears to reflect a mis-match between CDC norms and contemporary patterns of HC growth that is shared by large samples of healthy children, (ii) HCRN-defined macrocephaly rates in ASD have usually been indistinguishable from those in contemporaneously ascertained LRCs, and (iii) macrocephaly rate reports that lack parallel LRC comparison appear to vary as a function of how old the HCRNs used define macrocephaly were. Existing data potentially provide partial support for a subtle divergence of HC growth between a sub-group of children with ASD and LRCs during the second year life that (i) results in ~5mm group difference in mean HC at 24 months, and (ii) may index body size and SES related confounds.

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Data Blitz Session
Regency Ballroom 2

Allele-specific DNA deMethylation in FKBP5: A Molecular Mediator of Gene x Environment Interactions with Childhood Trauma

Tuesday, Poster #6

Torsten Klengel, Divya Mehta, Christoph Anacker, Jens C. Pruessner, Carmine M. Pariante, Thaddeus W.W. Pace, Kristina B. Mercer, Helen S. Mayberg, Bekh Bradley, Charles Nemeroff, Florian Holsboer, Christine M. Heim, Kerry J. Ressler, Theo Rein, Elisabeth Binder
Max-Planck-Institute of Psychiatry

Background: For most psychiatric diseases neither a genetic disposition nor environmental factors on its own are sufficient to elicit a specific disorder. Rather, genetic variation and environmental exposure interact to shape the development and function of the human brain and ultimately moderate the risk to suffer from psychiatric disorders. Here, we delineate an epigenetic mechanism for the gene x environment (GxE) interaction of the gene with childhood abuse on the development of post-traumatic stress disorder (PTSD) in adulthood.

Methods: Data from this study were collected as part of the Grady Trauma Project and replication was performed in data from the Conte Center Study for the Psychobiology of Early-Life Trauma (Emory University, Atlanta, GA, USA). Individuals were assessed using different measures for PTSD and childhood abuse. For genotyping and pyrosequencing, DNA was extracted from peripheral blood. Methylation analysis was performed by pyrosequencing of bisulfite treated genomic DNA. The functional impact of differential methylation was analyzed using a CpG free luciferase reporter construct and an GR sensitivity assay. In addition, we used a multipotent hippocampal progenitor cell line to assess the methylation status of FKBP5 in human neuronal cells in response to dexamethasone stimulation.

Results: FKBP5 rs1360780 interact with child abuse exposure (CTQ) on the development of current PTSD symptoms (mPSS) in adulthood ($F_{1963,2} = 4.40, P = 0.012$). The risk to suffer from lifetime PTSD (CAPS) is significantly increased by exposure to early trauma in *FKBP5* risk allele carriers ($X^2 = 28.6, df = 2, P < 0.001$), but not in carriers of the protective genotype ($X^2 = 2.02, df = 2, P = 0.36$).

Allele-specific DNA deMethylation in FKBP5: A Molecular Mediator of Gene x Environment Interactions with Childhood Trauma

Tuesday, Poster #6 (continued)

Torsten Klengel

Pyrosequencing of bisulfite treated DNA of highly traumatized individuals and controls revealed a significant demethylation of CpGs around glucocorticoid responsive elements (GREs) of *FKBP5* in abused individuals. We found a significant interaction of *FKBP5* genotype and childhood abuse on DNA methylation level in 3 CpGs in intron 7 ($F_{73,1} = 31.01$, $P_{\text{corr}} < 0.001$). When correlating level of child abuse using the Childhood Trauma Questionnaire (CTQ) with the methylation of intron 7, significant differences in the correlation coefficients were observed between the risk allele carriers and carriers of the protective allele ($R = -0.646$, $P < 0.001$ and $R = 0.414$, $P = 0.078$, Fisher Z-score of -4.23 , $P\text{-value} = 7.0 \times 10^{-5}$). This emphasizes the effects of early trauma severity on *FKBP5* demethylation in risk allele carriers, but not in carriers of the protective allele. Replication in an independent cohort from the Conte Center Study confirm these findings. Employing a CpG-free reporter construct, we demonstrate that changes in DNA methylation in intron 7 alter glucocorticoid responsiveness of *FKBP5* *in vitro*. An *ex-vivo* GR sensitivity assay demonstrate that intron 7 DNA methylation alters the ultra-short feedback loop between GR and *FKBP5* and thus GR sensitivity with reduced methylation in intron 7 associated with higher induction of FKBP5 by GR, representing an enhancement of the ultra-short feedback loop leading to increased GR resistance. In a multipotent human hippocampal progenitor cell line we show that *FKBP5* demethylation is initiated by GR-activation with dexamethasone which led to a highly significant DNA demethylation in CpGs in intron 7 similar to the CpGs in intron 7 affected by early trauma in *FKBP5* risk allele carriers (average of 17.1% demethylation in these 3 CGs, $P < 0.001$). We currently extend these results comparing DNA methylation changes in dexamethasone treated hippocampal progenitor cells with trauma exposed individuals on Illumina's 450k methylation bead chip. Preliminary data suggest a strong allele-dependent overlap between methylation in neuronal cells and childhood abused individuals.

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Allele-specific DNA deMethylation in FKBP5: A Molecular Mediator of Gene x Environment Interactions with Childhood Trauma

Tuesday, Poster #6 (continued)

Torsten Klengel

Conclusions: *FKBP5*, an important regulator of the stress hormone system, increase the risk of developing PTSD by allele-specific, childhood trauma-dependent demethylation of CpGs in functional GREs of *FKBP5*. For the first time, we delineate a molecular mechanism by which environmental impact in early life is encoded in epigenetic modifications and moderated by genetic predisposition influencing the development of psychiatric symptoms in later life. Our findings might be of particular relevance for the developing organism since the effects on DNA methylation seemed to be restricted to exposure to childhood trauma and were not influenced by traumatic experiences in adulthood, suggesting a possible sensitive period in early development for these epigenetic effects.

PL

Differential Control of Learning and Anxiety along the Dorso-ventral Axis of the Dentate Gyrus

Tuesday, Poster #22

Mazen A. Kheirbek, Liam J. Drew, Daniel Costantini, Nesha Burghardt, Lindsay Tannenholz, Susanne E. Ahmari, Hongkui Zeng, Andre Fenton, Rene Hen
Columbia University

Background: The hippocampus, in addition to its role in learning and memory, is increasingly implicated in the pathophysiology of anxiety disorders. The hippocampus shows marked variation along its dorso-ventral axis in terms of both afferent and efferent connectivity, yet it is unclear if this heterogeneity mediates its differential contributions to memory processing and to anxiety-like behavior, whether the three primary subregions of the hippocampus (dentate gyrus, CA3 and CA1) perform the same operations along the dorso-ventral axis, or if real-time activity changes in the hippocampal circuitry can acutely affect emotional state. Granule cells (GCs) of the dentate gyrus subregion of the hippocampus are implicated in affective processing, as they are especially susceptible to damage by elevated stress hormone levels, and adult neurogenesis, a unique feature of the DG, modulates emotional states and is required for some of the behavioral effects of antidepressants. To test the specific contribution of DG GCs to emotional behavior we examined the effects of acutely increasing or decreasing activity in DG GCs in tests of cognition and mood.

Methods: We used optogenetic techniques to modulate activity in DG GCs in real-time. To target ops expression selectively to GCs we used a POMC-Cre line crossed to conditional eNpHR3.0-EYFP and ChR2-tdTomato lines. Mice were implanted with fiber optics targeted to either the dorsal or the ventral DG, and tested for behavioral effects of light induced inhibition or excitation. To test the role of DG GCs in learning and anxiety-like behavior, mice were tested in contextual fear conditioning, active place avoidance, elevated plus maze and open field test.

Results: Dependent on their position along the dorso-ventral axis of the hippocampus, GCs control specific features of anxiety-related behavior and

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Differential Control of Learning and Anxiety along the Dorso-ventral Axis of the Dentate Gyrus

Tuesday, Poster #22 (continued)

Mazen A. Kheirbek

contextual learning. In mice expressing eNpHR3.0 in the DG, inhibition GCs in the dorsal, but not ventral, DG blocks the encoding, but not the retrieval, of contextual fear memories and the rapid and flexible encoding of spatial information in active place avoidance, while having no effect on anxiety-related behaviors. In contrast, elevating the activity of GCs in the dorsal DG with ChR2 resulted in a dramatic increase in exploratory behavior, while elevating activity in the ventral DG powerfully suppressed innate anxiety.

Conclusions: By using optogenetic techniques that allow neural activity in DG GCs to be acutely, reversibly and bidirectionally manipulated, this study supports the hypothesis that the dorsal and ventral poles of the hippocampus are functionally distinct and demonstrates that hippocampal activity not only has a mnemonic function but can also strongly influence anxiety-related behaviors. Specifically, dorsal GCs were shown to contribute to spatial and contextual learning, where they were required for rapid encoding of contextual information, but not for memory retrieval. Surprisingly, elevating dorsal DG activity also induced a dramatic increase in exploratory behavior in novel environments. The ventral DG was not required for contextual fear learning, but was found to exert a major influence on innate anxiety-like behavior. Recent studies employing deep brain stimulation to ameliorate symptoms of treatment resistant depression highlight the effectiveness of circuit based approaches for the treatment of psychiatric illness. Our results provide the first evidence that increasing activity in the ventral DG can reduce innate anxiety without affecting learning. The clear dissociation between the contributions of the dorsal and ventral poles of the DG to cognitive function and anxiety offers a rationale for pursuing strategies that target the ventral DG to treat anxiety with minimal cognitive side effects.

Broader Autism Phenotype: Relationships between Maternal/paternal BAP, Parental SSRI Treatment, WB 5-HT and Child's Autism Symptoms

Tuesday, Poster #53

Tal Levin-Decanini, Nell Maltman, Guter Stephen, Edwin H. Cook, Suma Jacob
University of Illinois, Chicago

Background: Subtle expression of related traits in relatives of persons with autism spectrum disorders (ASD), known as the broader autism phenotype (BAP) has been demonstrated previously. Elevated whole blood serotonin (WB 5-HT) is the most long-standing and best-replicated biological findings in ASD. Previous research has shown a relationship between whole blood serotonin levels and parental ratings of depression but those studies did not include measurement of BAP. The present study focused on the relationship between parental BAP and sex, selective serotonin reuptake inhibitor (SSRI) treatment, and the child's autism symptoms.

Methods: Subjects included 197 children with ASD, and 357 of their parents (n=357). Of these parents, 25 were taking SSRIs. Proband symptoms were measured using the ADOS, ADI-R, CRI, and RBS-R domain scores. Parental BAP was measured by the Broader Autism Phenotype Questionnaire (BAPQ). In addition to total BAPQ, Aloofness, Rigidity, Pragmatic Language subscores, nine clinical expert raters identified items that measure autism-related "insistence on sameness" (IS) in order to examine those characteristics in the parent measure that may be more closely related to proband characteristics and WB 5-HT. The BAP-IS subscale was obtained by summing responses across six statements in the Rigid subscale of the BAPQ. MANCOVAs were performed to explore relationships between sex and SSRI medication use on the subscales of parent BAPQ and proband symptom scores.

Results: There were significantly different average BAPQ scores across sex and medication groups, although the effect size was modest (Wilks' $\Lambda=0.881$, $F(12,709) = 2.897$, $p = 0.001$; $\eta_p^2 = 0.041$). Sex and medication had significant effects on the Total score ($F(3,271) = 5.103$, $p = 0.002$; $\eta_p^2 = 0.058$) and on Aloof ($F(3,271) = 6.015$, $p = 0.001$; $\eta_p^2 = 0.062$), Rigid ($F(3,271) = 5.212$, $p = 0.001$; $\eta_p^2 = 0.055$)

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Broader Autism Phenotype: Relationships between Maternal/ paternal BAP, Parental SSRI Treatment, WB 5-HT and Child's Autism Symptoms

Tuesday, Poster #53 (continued)

Suma Jacob

and the subset of items related to IS ($F(3,271) = 5.103, p = 0.002; \eta_p^2 = 0.053$). Post-hoc analyses showed mothers others taking SSRIs also had higher Total ($p=0.021$) and Rigid subscale scores ($p = 0.026$) than those mothers not on SSRI medications. Fathers not taking SSRIs had higher Aloof and Rigid scores compared to mothers not taking SSRIs.

Conclusions: The results of this study showed that among parents not on SSRI medication, fathers score significantly higher on multiple measures of the BAPQ than mothers, namely BAP Total, Aloof and IS-related items. We did not find a relationship between parental Rigid scores and child Insistence on Sameness scores. However, we did confirm correlation of WB 5-HT between parents and children and found higher Aloof and Rigid scores in fathers compared to mothers. The increase in BAPQ scores in our subsample of mothers being treated with SSRIs was unexpected and requires careful interpretation and further study.

PL

The Functional Significance of Antipsychotic-related Cortical Thinning in First Episode Schizophrenia

Tuesday, Poster #60

Tyler A. Lesh, Costin Tanase, Tara Niendam, Jong Yoon, J Daniel Ragland, Michael Minzenberg, Marjorie Solomon, Cameron Carter
University of California, Davis

Background: Findings of structural and functional brain abnormalities are consistently replicated in magnetic resonance imaging (MRI) studies of patients with schizophrenia. Studies using voxel-based morphometry and measurements of cortical thickness identify gray matter reductions and cortical thinning in prefrontal and temporal structures, as well as increased volume in the basal ganglia. Schizophrenia patients typically show altered activation of these same regions, particularly reduced activity in dorsolateral prefrontal cortex, during fMRI tasks tapping the fronto-parietal cognitive control circuit (e.g., AX-CPT, N-back). However, the degree to which antipsychotic medications are associated with changes in brain structure, function, and behavioral performance within the illness are poorly understood. We sought to examine these effects in first episode schizophrenia patients, who were evaluated within one year of illness onset, utilizing cortical thickness measurements and fMRI. Cortical thickness measurements were derived from surface-based registration methods where homologous regions are matched, as opposed to relying upon spatial smoothing of VBM analyses, potentially offering increased sensitivity to subtle cytoarchitectural changes. The AX-CPT was used as a measure of functional fronto-parietal recruitment. When compared to healthy controls, we hypothesized that patients with schizophrenia would show thinner cortex and reduced activation of dorsolateral prefrontal cortex, as well as lower performance reflecting impaired cognitive control. Additionally, we anticipated that patients receiving antipsychotic medication compared to those who were unmedicated would show more extensive prefrontal cortical thinning in the context of improved functional activity and better behavioral performance. **Methods:** Medicated (n=24) and unmedicated (n=21) first episode schizophrenia patients as well as healthy control participants (n=28) were identified from referrals to the UC Davis Early Detection and Preventative Treatment clinic

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The Functional Significance of Antipsychotic-related Cortical Thinning in First Episode Schizophrenia

Tuesday, Poster #60 (continued)

Tyler A. Lesh

using the Structured Clinical Interview for DSM-IV. Images were obtained on a 1.5-Tesla General Electric scanner and processed using Freesurfer 4.1 (structural analysis) and SPM8 (fMRI analysis). Statistical analyses of structural data were conducted using a vertex-wide threshold of $p < .01$ followed by a cluster-wise correction for multiple comparisons (5000 Monte Carlo simulations, $p < 0.05$). Statistical analysis of AX-CPT fMRI data focused on CueB versus CueA trials and both whole-brain cluster-corrected and DLPFC region of interest analyses were performed.

Results: Analyses of structural data revealed significant cortical thinning in medicated patients with schizophrenia relative to healthy controls in dorsolateral prefrontal and orbitofrontal cortices. Unmedicated schizophrenia patients demonstrated no significant cortical thickness differences from healthy controls after cluster-wise correction. A comparison of medicated and unmedicated patients revealed significant cortical thinning in medicated patients only in the DLPFC. With regard to brain activation during the AX-CPT, both patient groups showed reduced activity in frontal and parietal regions compared to healthy controls. However, medicated schizophrenia patients also demonstrated higher DLPFC activation compared to unmedicated patients. A similar pattern emerged in behavioral performance, in which medicated patients showed higher performance (as indexed by d' -context scores) than unmedicated patients.

Conclusions: These findings highlight the complex relationship between antipsychotic treatment and the structural, functional, and behavioral deficits repeatedly identified in schizophrenia. Although treatment with antipsychotic medications was associated with prefrontal cortical thinning, treatment was also related to better cognitive control and increased prefrontal functional activity that was comparable to healthy controls. This study also highlights the critical importance of multi-modal analyses in understanding the complex nature of structural and functional neurophysiological changes in the disorder.

Imaging the Sensitivity of [¹²³I]5-IA-85380 to Increases in Acetylcholine at the Beta2-nicotinic Acetylcholine Receptors in Human Subjects

Tuesday, Poster #74

Irina Esterlis, Jonas Hannestad, Frederic Bois, Andrew Sewell, Rachel Tyndale, John Seibyl, Marina Picciotto, John H. Krystal, Marc Laruelle, Richard Carson, Kelly Cosgrove
Yale University

Background: Introduction: Acetylcholine is one of the major neurotransmitters in the brain and has been implicated in psychiatric and medical illnesses. Our evaluation of the nicotinic acetylcholine system (β_2^* -nAChR) in vivo in individuals with current and remitted major depressive disorder demonstrated significantly lower receptor availability associated with current depression (and less so with remitted depression) compared to controls. We followed up with a postmortem study to quantify β_2 -nAChR density under the conditions where endogenous acetylcholine is washed out, and observed no difference in β_2 -nAChR density between individuals with and without major depressive disorder. An interpretation of these results is β_2^* -nAChRs might be lower in depression, and/or that increased acetylcholine concentration in the vicinity of β_2^* -nAChR might reduce the availability of these receptors to the binding of the radioligand. Previously, Fujita and colleagues showed that the high affinity β_2^* -nAChR radioligand [¹²³I]5-IA-85380 ([¹²³I]5-IA) may be sensitive to extracellular increases in acetylcholine in baboons¹; however, such an examination in humans has lagged. Given that acetylcholine is one of the major neurotransmitters in the brain and has been implicated in the psychiatric and medical illnesses, we developed a paradigm to interrogate the cholinergic system in vivo via use of [¹²³I]5-IA single photon emission computed tomography (SPECT) imaging and physostigmine, a centrally-acting acetylcholinesterase inhibitor.

Methods: Six healthy subjects (3 men, 3 women; 31±4.1 yrs) participated in one [¹²³I]5-IA SPECT study and one magnetic resonance imaging (MRI) scan. MRI was used to guide placement of regions of interest for SPECT scans. [¹²³I]5-IA was administered as a bolus plus constant infusion (B/I 7.0h); total injected dose

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Imaging the Sensitivity of [¹²³I]5-IA-85380 to Increases in Acetylcholine at the Beta2-nicotinic Acetylcholine Receptors in Human Subjects

Tuesday, Poster #74 (continued)

Irina Esterlis

was 390.2 ± 13.2 MBq. After three 30-min baseline scans at 6-8h post infusion, physostigmine (1-1.5mg) was administered IV over 60 min, and nine additional 30-min scans were collected during the next 6h. The outcome measure was V_s/f_p (specific volume of distribution), calculated as V_T/f_p (estimated receptor availability) minus V_{ND}/f_p (nondisplaceable binding; previously estimated in a smoking to satiety paradigm ²).

Results: We observed a peak average decrease in V_s/f_p of $25 \pm 15\%$ in cortical regions ($t=3.2, p=0.03$), $15 \pm 11\%$ in thalamus ($t=2.8, p=0.05$), $16 \pm 14\%$ in striatum ($t=2.6, p=0.06$), and $35 \pm 34\%$ in cerebellum ($t=2.8, p=0.05$). This effect reflected a combination of a significant decrease in tissue concentration of 5-IA (7-16% region specific, $p<0.05$) and a significant increase in plasma parent concentration (8%, $p<0.05$). There were no significant changes in subjects' self-reported mood symptoms after physostigmine challenge.

Conclusions: We developed a paradigm to interrogate the cholinergic system *in vivo* in human subjects and observed a significant decrease in specific binding of [¹²³I]5-IA following physostigmine challenge, consistent with an increase in endogenous extracellular ACh levels. This confirms a previous study in baboons (Fujita et al. 2003). This imaging tool may have enormous potential to facilitate the development of innovative medicines aimed at modulating the cholinergic system. This study is inherently innovative in the use of neuroreceptor imaging techniques to interrogate the ACh system *in vivo* in human subjects. 1. Fujita M, Al-Tikriti M, Tamagnan G, et al. Influence of acetylcholine levels on the binding of a SPECT nicotinic acetylcholine receptor ligand [¹²³I]5-I-A-85380. *Synapse*. 2003;48:116-122. 2. Esterlis I, Cosgrove K, Batis J, et al. Quantification of smoking induced occupancy of β_2 -nicotinic acetylcholine receptors: estimation of nondisplaceable binding. *Journal of Nuclear Medicine*. 2010;51:1226-1233.

Self-regulation of Amygdala Activity with Real-time fMRI Neurofeedback in Patients with Depression

Tuesday, Poster #91

Kymerly Young, Raquel Phillips, Vadim Zotev, Wayne C. Drevets, Jerzy Bodurka
Laureate Institute for Brain Research

Background: Up to two-thirds of patients with major depressive disorder (MDD) who seek standard pharmacological and/or psychological interventions will not respond, while only one-half who do will achieve sustained remission. Cognitive-behavioral therapy (CBT), the most commonly implemented psychological treatment for MDD, is most effective for mildly to moderately depressed patients. In severely ill patients, CBT is often ineffective, and treatments available for these severely ill non-responders (such as electroconvulsive therapy, vagus nerve stimulation, and deep brain stimulation) are invasive, expensive, and pose significant risks. Therefore, there is a need to develop novel and non-invasive treatments for MDD. MDD is associated with the deregulation of brain emotional circuitry, with significant changes in amygdala activity. Research has shown that the hemodynamic response of the amygdala is exaggerated to negative stimuli in MDD, with further evidence that amygdala responses to *positive* stimuli are *attenuated* in MDD, and that this later response normalizes with remission. The availability of real-time functional magnetic resonance imaging (rtfMRI) and recent advances in rtfMRI neurofeedback (rtfMRI-nf) permit, for the first time, direct targeting of this region. The current study aims to determine whether individuals with MDD are able to use rtfMRI-nf to enhance the hemodynamic response of the amygdala to positive stimuli, and whether this ability will correspond to alterations in mood.

Methods: Unmedicated participants with a current diagnosis of MDD participated in the current study (n=19). Twelve received active rtfMRI-nf with the left amygdala (LA) as the target region of interest (ROI), and 7 received sham feedback in which the target ROI was the left horizontal segment of intraparietal sulcus (HIPS), a region putatively not involved in emotional regulation. In each of four 8min runs, alternating 40s blocks of Rest, Count, and Happy were

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Self-regulation of Amygdala Activity with Real-time fMRI Neurofeedback in Patients with Depression

Tuesday, Poster #91 (continued)

Kymerly Young

presented. During Rest blocks participants were instructed to clear their minds and focus on the screen. During Count blocks participants were instructed to count backwards from 300 by the number provided. During Happy blocks, the cue “Happy” and two colored bars (red, blue) were displayed on the screen. The red bar represented the actual BOLD neurofeedback signal from the target ROI, which was updated every 2s by changing the height of the bar. Subjects were instructed to retrieve and contemplate positive autobiographical memories while also attempting to increase the level of the red bar to that of the fixed target level displayed by the blue bar. The target blue bar level was increased from run to run. A final 8min Transfer run was presented in which no feedback was provided. Additionally, an 8min rest run was included at the beginning and end of the fMRI session. All imaging was conducted on a GE Discovery MR750 3T MRI scanner with an 8-channel receive-only brain coil. Single shot gradient-recalled EPI with sensitivity encoding (SENSE) was used for fMRI with FOV/slice=240/2.9mm, TR/TE=2000/30ms, SENSE=2, 96×96 matrix, flip=90°, 34 axial slices. A T1-weighted MPRAGE sequence was used for anatomical reference and to define ROIs. Neurofeedback was implemented using a custom real-time fMRI system utilizing AFNI real-time features and a custom GUI software. For each subject, three spherical ROIs (7 mm radius in Talairach space) were centered, respectively, at the left and right amygdala and the HIPS region. The fMRI data analysis was based on GLM and performed in AFNI.

Results: Four of the MDD patients in the active rtfMRI-nf group were unable to learn to successfully regulate their amygdala (defined as LA BOLD response no different from 0 during the transfer run) and were therefore excluded from the group analysis. These patients were significantly younger and had increased fatigue ratings compared to those patients in the active group who successfully regulated their LA. The remaining 8 participants in the active rtfMRI-nf group significantly increased their LA response (BOLD response for Happy-Rest

Self-regulation of Amygdala Activity with Real-time fMRI Neurofeedback in Patients with Depression

Tuesday, Poster #91 (continued)

Kymerly Young

condition > 0) and maintained this elevated activity during the transfer run in which no neurofeedback was provided ($p=0.03$). In the sham neurofeedback group, the BOLD response within the LA did not significantly increase from 0 in any of the training or transfer runs ($p_s>0.10$). The difference between the active and sham groups in LA activity was significant for the last training run and the transfer run ($p_s<0.05$). BOLD activity did not significantly change within the right amygdala or HIPS for either the active or sham groups ($p_s>0.11$). State measures of happiness significantly increased, while state measures of depression significantly decreased in the group receiving active rtfMRI-nf ($p_s<0.05$), but did not change significantly in the group that received sham feedback ($p_s>0.15$).

Conclusions: Our results show that by using rtfMRI-nf from the LA during recall of positive autobiographical memories, a subset of individuals with MDD can learn to self-regulate their amygdala BOLD responses. We also found an association between the ability to regulate the LA and reductions in depression ratings, as well as improvements in happiness ratings. These preliminary results suggest applications for rtfMRI-nf training and positive autobiographical memory recall in the treatment of MDD.

PL

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Cannabinoid Facilitation of Extinction Recall via Increased Recruitment of Prefrontal-hippocampal Circuitry in Healthy Humans

Tuesday, Poster #164

Christine A. Rabinak, Mike Angstadt, Chandra Sripada, Mohammed R. Milad, Israel Liberzon, K. Luan Phan
University of Michigan

Background: Enhancing extinction learning may optimize gains achieved by exposure therapy for anxiety disorders (e.g., maintenance of effects, hastened pace of improvement, greater generalization outside therapeutic context). Emerging evidence from animal studies suggest that enhancing cannabinoid system within the ventromedial prefrontal cortex (vmPFC) and hippocampus (HPC), brain structures critical to fear extinction, enhances fear extinction and its retention. However, the role of cannabinoids on the retention of extinction memory and its effect on the underlying neural circuits in humans remains unknown.

Methods: We conducted an fMRI study using a randomized, double-blind, placebo-controlled, between-subjects design, coupled with a standard Pavlovian fear extinction paradigm and simultaneous skin conductance response (SCR) recording with an acute pharmacological challenge with oral dronabinol (synthetic Δ^9 -tetrahydrocannabinol; THC, n = 15) or placebo (PBO, n = 15) 2 hours prior to extinction learning in healthy adult volunteers to assess the effects of THC on vmPFC and HPC activation when tested for recall and maintenance of extinction learning at 24 hours and 1 week after training, respectively.

Results: Compared to subjects who received PBO, those who received THC showed increased vmPFC activation and functional coupling with the HPC, as well as low SCR to a previously extinguished CS when extinction memory recall was tested, suggesting that THC prevented the recovery of fear via increased recruitment of the vmPFC and HPC.

Conclusions: These results advance the neurobiology of extinction learning and prompt development of novel pharmacological modulators of the cannabinoid system to maximize the potency of exposure therapy for anxiety disorders.

A Brief Monetary Progressive Ratio Task Predicts Clinical Amotivation and Ventral Striatum Activation in Schizophrenia

Wednesday, Poster #47

Jacob Kantrowitz, Natalie Katchmar, Theodore Satterthwaite, Lillie Vandekar, Ruben Gur, Raquel Gur, Daniel Wolf
University of Pennsylvania

Background: Motivational deficits play a central role in disability due to negative symptoms of schizophrenia, which constitute a major unmet therapeutic need in psychiatry. Despite this importance, amotivation in schizophrenia has been understudied and its pathophysiology remains largely unknown. Negative symptoms of schizophrenia have previously been linked to hypofunction in ventral striatum (VS), a crucial component of the mesolimbic dopamine motivation circuitry. However, further work is needed to determine whether specific negative symptoms such as amotivation drive this relationship. This effort can be facilitated by new interview-based assessments like the Clinical Assessment Interview for Negative Symptoms (CAINS), which distinguishes amotivation from related negative symptoms such as anhedonia and asociality by emphasizing both subjective experience and objective behaviors. In addition, improved reliability, validity and translatability to animal models will require applying neurobehavioral measures of amotivation in the laboratory. Here we report initial validation of a brief, computerized progressive ratio task (PRT) that quantifies effort exerted in pursuit of monetary reward. We show that motivation assessed dimensionally with this PRT predicts both clinical amotivation on the CAINS and VS fMRI responses to monetary reward.

Methods: 41 patients with schizophrenia (SCH, stable/medicated) and 37 group-matched controls (CTR) performed a brief computerized PRT to earn money. The PRT required repetition of easy but attention-requiring trials (choosing which of 2 numbers was larger). Within each of three runs, an increasing number of repetitions was required to obtain the monetary reward. Across the three runs, the amount of reward progressively decreased (50 cents, 25 cents, 10 cents). A run ended when the subject chose not to attempt or complete the required number of repetitions. This “breakpoint” was used to quantify motivation, and was defined here as the

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A Brief Monetary Progressive Ratio Task Predicts Clinical Amotivation and Ventral Striatum Activation in Schizophrenia

Wednesday, Poster #47 (continued)

Daniel Wolf

ratio of effort (maximum number of performed trials) to monetary value, averaged across runs. Prior to the PRT, subjects performed BOLD fMRI at 3T including a monetary guessing paradigm that robustly activates VS. VS activation measures (win>lose) were extracted and correlated with PRT breakpoints. Psychopathology was evaluated with self-report and interview scales; the CAINS provided the primary measure of clinical amotivation.

Results: Total PRT duration averaged 16 min (+/-14), without group differences. PRT breakpoints ranged widely in both groups, from ~0.1 trials per cent (tpc) up to ~10 tpc. As expected, average PRT motivation was reduced in patients [SCH 2.3 tpc (+/-2.8), CTR 4.3 tpc (+/-3.7), 1-tail p=0.03]. In SCH, the predicted inverse correlation of PRT with CAINS amotivation was significant ($r = -0.40$, 1-tail p=0.005). The same relationship was found in CTR ($r=-0.29$, 1-tail p=0.04). When the relationship of PRT breakpoint to both diagnosis and CAINS amotivation were tested in a multiple regression, the effect of CAINS was significant (2 tail p=0.002) but not diagnosis (p=0.75), indicating that the group difference in PRT was attributable to individual differences in motivation as assessed with the CAINS, rather than to a simple categorical effect of diagnosis. Correlations between PRT and other negative symptom domains were also negative, but less robust. Potential confounds including socioeconomic status, cognition, reaction time, smoking, depression, and positive symptoms did not explain the relationship between PRT breakpoint and CAINS amotivation. In SCH, lower PRT motivation also predicted reduced VS activation to monetary reward ($r=0.36$, 2-tail p=0.03).

Conclusions: We report one of the first applications of PRT in schizophrenia, and provide initial evidence of its construct validity in relationship to clinical motivation and an fMRI measure of motivation circuit function. It is striking that a brief laboratory measure of motivation shows even these moderate correlations with a clinical measure that is inevitably impacted by various life circumstances operative outside, but not necessarily inside, the laboratory. The

A Brief Monetary Progressive Ratio Task Predicts Clinical Amotivation and Ventral Striatum Activation in Schizophrenia

Wednesday, Poster #47 (continued)

Daniel Wolf

brief computerized PRT described here has advantages over clinical measures of motivation, including translatability to non-human models, greater objectivity, and potentially greater specificity. The observed correlation with VS activation supports further use of the PRT in studies aiming to identify neural circuit, molecular-genetic, and psychiatric symptom correlates of motivation, and for assessing and predicting response to novel therapeutic interventions.

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The Neurosteroids Allopregnanolone and DHEA Enhance Emotion Regulation Neurocircuits and Modulate Memory for Emotional Stimuli

Wednesday, Poster #76

Rebecca K. Sripada, Christine E. Marx, Anthony P. King, Sarah N. Garfinkel, James L. Abelson, Israel Liberzon
 University of Michigan

Background: Allopregnanolone (ALLO) and dehydroepiandrosterone (DHEA) are endogenously-produced neurosteroids with neuroprotective, anxiolytic, antidepressant, and antigluocorticoid effects. Dysregulated release of these neurosteroids has been extensively linked to mood and anxiety disorders. Both neurosteroids are endogenously released in response to stress, and reduce negative affect when administered exogenously. Though these antidepressant and anxiolytic effects have been well established, no research to date has examined the neural pathways involved. In particular, brain imaging has not been used to link neurosteroid effects to emotion regulation neurocircuitry.

Methods: To investigate the brain basis of ALLO and DHEA's impact on emotional response and regulation, subjects were administered 400mg of pregnenolone (N=16), 400mg of DHEA (N=14), or placebo (N=15) and underwent 3T fMRI while performing the Shifted-Attention Emotional Appraisal Task (SEAT), a test of emotional processing and regulation. FMRI data were analyzed in SPM8 random-effects models ($p < 0.05$, FWE-corrected for whole brain analyses, small-volume-corrected [SVC] for ROIs).

Results: Compared to placebo, ALLO and DHEA both reduced activity in the amygdala ([27,-1,-17]; $F(1,29)=9.97$; [27,-10,-14]; $F(1,27)=6.9$, $p < .05$, SVC). ALLO decreased activity in the insula ([42,8,4]; $F(1,29)=10.97$, $p < .05$, SVC), whereas DHEA decreased activity in the hippocampus ([-33,-28,-11]; $F(1,29)=22.07$, $p < .05$, SVC) and enhanced connectivity between the amygdala and hippocampus ([30,-13,-11]; $z=3.40$, $p < .05$, SVC). DHEA enhanced activity in the rostral anterior cingulate cortex ([3,41,-2]; $F(1,29)=13.3$, $p < .05$, SVC), whereas ALLO increased activity in the dorsal medial prefrontal cortex ([3,56,37]; $F(2,232)=6.41$, $p < .05$, SVC) and enhanced connectivity between the amygdala

The Neurosteroids Allopregnanolone and DHEA Enhance Emotion Regulation Neurocircuits and Modulate Memory for Emotional Stimuli

Wednesday, Poster #76 (continued)

Rebecca K. Sripada

and dorsal medial prefrontal cortex ($[-30,-1,-23]$; $t=4.8$, $p<.001$), an effect that was associated with reduced self-report anxiety ($r=-.52$, $p=.046$). DHEA reduced memory accuracy for emotional stimuli (conjunctive d' ; $t(27)=2.31$, $p=.029$), and reduced activity in regions associated with conjunctive memory encoding.

Conclusions: These results demonstrate that ALLO and DHEA reduce activity in regions associated with generation of negative emotion and enhance activity in regions linked to regulatory processes. Considering that activity in these regions is altered in mood and anxiety disorders, our results provide initial neuroimaging evidence that these neurosteroids may be useful as pharmacological interventions for these conditions and invite further investigation into the brain basis of neurosteroid emotion regulatory effects.

PL

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Neuronal Signatures of Self-control in Anterior Cingulate Cortex

Wednesday, Poster #105

Benjamin Hayden, Tommy Q. Blanchard
University of Rochester

Background: Addiction, obsessive compulsive disorder (OCD), and Tourette Syndrome (TS) are diseases in which the core symptoms include deficits in self-control. Recent studies have begun to identify the key brain areas that govern our ability to resist temptation, but the circuit-level mechanisms of self-control remains poorly understood. Of the core elements of self-control, delay of gratification, or persistent commitment to the choice of a delayed option, has been identified as especially important. Inspired by the recent development of delay-of-gratification tasks for rhesus macaques, we have developed corresponding tasks that are usable with single unit recordings. We focused on the dorsal anterior cingulate cortex (dACC). The dACC has been linked overcoming impulsive behaviors, to self-control, and to executive control more broadly. Lesions to dACC produce frank deficits in self-control and variations in structure and function in dACC predict susceptibility to addiction, OCD, and TS.

Methods: On each trial of our task, one of several possible options (colored rectangles) appears at the top of a computer monitor and quickly glides down the screen. Monkeys can accept or reject this option by fixating on it. Once an option is accepted, the monkey must maintain gaze on it for several seconds in order to obtain a reward. Failures to maintain gaze for the duration of the shrinking period (several seconds) are considered persistence failures and lead to no reward. Options vary in their benefit (reward amount offered) and cost (delay until reward). We recorded firing rate activity of 125 single neurons in the dACC while two macaques performed this task.

Results: We found that monkeys' chose approximately optimally and that their choices reflected a balance between costs and benefits for each option. Neuronal activity was tonically enhanced throughout the hold period, suggesting that it contributes to active resistance to temptation. Consistent with this idea, we found that variations in dACC activity predicted accept or reject decisions for individual stimuli, and in approximately one quarter of neurons, firing rate during the second

Neuronal Signatures of Self-control in Anterior Cingulate Cortex

Wednesday, Poster #105 (continued)

Benjamin Hayden

before the stimulus appeared predicted the monkeys decision. When monkeys made a decision and stuck with it past 750 ms, they proceeded to successfully maintain gaze on it for about 90-95% of trials. On the remaining trials they failed to maintain gaze and let the option disappear. These trials are demonstrably suboptimal because, due to the structure of the task, it was always better to reject any option immediately. We therefore classify these trials as self-control failures. In approximately 20% of dACC neurons, we found a slight but significant suppression in neuronal activity during the half second before the failed self-control gaze shifts. These reductions in activity are therefore predictive of self-control failures.

Conclusions: Our results indicate that dACC plays a direct role in controlling delay-of-gratification decisions in a macaque self-control task. Specifically, they suggest that dACC provides a proactive control signal that facilitates persistent commitment to an abstemious decision. These results therefore constitute the first putative self-control signal observed at the single neuron level in macaques. Past studies have generally emphasized the importance of dACC for monitoring and for reactive control; the present results demonstrate its key role in proactive control as well. It is likely that dACC is only one of several brain areas important for self-control. The dorsolateral prefrontal cortex and ventromedial prefrontal cortex has been hypothesized to play distinct roles in self-control decisions as well. Our results suggest that dACC may be a high-level controller that tunes activity in these other areas during self-control decisions. More broadly, these results offer a potential explanation for the observed diminution in self-control that accompanies addiction, OCD, and other diseases associated with aberrant structure and function of the dACC. Finally, these results suggest that dACC may be a good target for future therapies, such as deep brain stimulation, that aim to improve self-control in severe psychiatric conditions.

PL

11:15 a.m. - 1:30 p.m.
Data Blitz Session
Regency Ballroom 2

Impaired Reward Responsiveness during Nicotine Withdrawal in Rats and Humans Assessed in a Translational Behavioral Procedure

Wednesday, Poster #111

Andre Der-Avakian, Michele L. Pergadia, Manoranjan S. D'Souza, Pamela AF. Madden, Andrew C. Heath, Saul Shiffman, Diego A. Pizzagalli, Athina Markou
University of California, San Diego

Background: Nicotine withdrawal produces negative affective symptoms similar to those seen in depression. It is hypothesized that chronic drug exposure and withdrawal promotes the abnormal processing of rewards, which may contribute to addictive behaviors and negative affect during withdrawal that in turn may lead to relapse. Clinical evidence suggests that depressed subjects display abnormal processing of positively reinforcing stimuli (i.e., anhedonia, or decreased interest in rewards) when assessed using the Response Bias Probabilistic Reward Task [Pizzagalli et al. (2008) *J Psychiatr Res* 43, 76-87]. Briefly, this task involves exposure to two different visual stimuli that are difficult to discriminate, each requiring a different response to lead to reinforcement. Correct responses to one stimulus are rewarded three times more frequently (i.e., rich) compared to correct responses to the other stimulus (i.e., lean). Non-depressed human subjects modulate their behavior during testing as a function of prior reinforcement by gradually developing a biased response toward the rich stimulus. In contrast, depressed subjects fail to develop this response bias for the more frequently rewarded stimulus. The result is a quantitative task that objectively measures deficits in reward processing in depressed individuals. The goal of the present study was to determine whether nicotine withdrawal is associated with impaired reward responsiveness similarly in rats and humans using the Response Bias Probabilistic Reward Task originally developed in humans and recently adapted for rats. We hypothesized that nicotine withdrawal would be associated with similarly decreased reward responsiveness in rats chronically exposed to nicotine and heavy smoking human subjects.

Methods: Rats: Male Wistar rats were food restricted and trained in operant boxes to press a lever to receive a food pellet as a reward. Rats were then presented with

Impaired Reward Responsiveness during Nicotine Withdrawal in Rats and Humans Assessed in a Translational Behavioral Procedure

Wednesday, Poster #111 (continued)

Andre Der-Avakian

either a short or long tone (identical in all parameters other than duration) and trained to discriminate the tones by pressing one of two levers associated with each tone duration. Once these associations were learned, defined as more than 70% accuracy, the difference between the two tone durations was made more ambiguous during a 100-trial test session split into three blocks. Correct responses on the lever associated with either the short or the long tone (counterbalanced) were reinforced three times more frequently (i.e., rich) than correct responses on the other lever (i.e., lean). After this baseline test, rats were surgically prepared with subcutaneous osmotic minipumps delivering either 6.32 mg/kg/day nicotine (base) or saline vehicle for 28 days. After 28 days, minipumps were removed and rats were tested again 24 hr later during withdrawal. Humans: Participants classified as heavy smokers were presented on a computer screen with one of two mouths varying slightly in length on a schematic face, and instructed to discriminate the mouths by pressing one of two keys on a keyboard associated with each mouth length. During a 300-trial session split into three blocks, correct responses for either the long or short mouth (counterbalanced) resulted in presentation of a monetary reward (5 cents) three times more frequently than correct responses for the other mouth. At the end of the session, participants were given the amount of money won. In one session, participants were smoking at their usual rate prior to testing. In another session, participants were instructed to be 24 hr smoke-free prior to testing (i.e., withdrawal), which was biologically verified. The smoking and abstinence sessions were randomly counterbalanced across subjects and were approximately one-week apart.

Results: Rats: Saline-treated rats developed a response bias towards the rich stimulus, which was comparable to the response bias previously quantified in non-depressed human subjects. In contrast, response bias was significantly decreased in rats exposed to nicotine withdrawal ($p < 0.05$). Humans: In heavy smokers,

11:15 a.m. - 1:30 p.m.
Data Blitz Session
Regency Ballroom 2

Impaired Reward Responsiveness during Nicotine Withdrawal in Rats and Humans Assessed in a Translational Behavioral Procedure

Wednesday, Poster #111 (continued)

Andre Der-Avakian

response bias was significantly decreased within subjects during 24 hr abstinence relative to the smoking session ($p < 0.05$). Collectively, control rats and heavy smokers when smoking (relative to their abstinence day) responded more toward the rich stimulus than the lean stimulus. However, rats and humans experiencing withdrawal from nicotine responded similarly with less overall responsiveness toward the rich stimuli despite the fact that correct responses for the rich stimuli were reinforced more frequently.

Conclusions: The results indicate that withdrawal from nicotine significantly impairs reward responsiveness in both rats and humans as assessed using the Response Bias Probabilistic Reward Task. This impairment of reward responsiveness is reflected by an inability to alter behavioral responding as a function of prior reinforcement experience. Being able to identify reward processing impairments that are analogous across species will facilitate translational research investigating the behavioral and neurobiological mechanisms that underlie nicotine reward and withdrawal.

PL

Long Acting Injectable vs. Oral Antipsychotics in Schizophrenia: A Systematic Review and Meta-analysis of Mirror-image Studies

Wednesday, Poster #175

Taishiro Kishimoto, Masahiro Nitta, Michael Borenstein, John Kane, Christoph U. Correll
The Zucker Hillside Hospital, Glen Oaks, New York

Background: As psychopathology and social functioning can worsen with repeated psychotic episodes in patients with schizophrenia, relapse prevention is critical. High non-adherence rates in this population can limit the efficacy of pharmacotherapy, therefore, the use of long-acting injectable antipsychotics (LAIs) is considered to be an important treatment option. However, new, large, randomized controlled trials (RCTs) showed no significant benefit of LAIs over oral antipsychotics (OAPs) (e.g., Rosenheck et al. 2011; Schooler et al. 2011). Moreover our latest meta-analysis of RCTs showed no superiority of LAIs over OAPs (Kishimoto et al. in submission: Studies=21, $n=4,950$, $RR=0.93$, 95%CI: 0.80-1.08, $p=0.35$). However, clinical trials might over-represent patients with better treatment adherence and lower illness severity. In addition, patients in clinical trials are likely to receive more and different types of attention than those in routine care, such as measures of adherence, reminders to attend clinical/research assessment sessions, etc. Therefore, the standard RCT might not be the best strategy to examine the efficacy of LAIs, and this possibility needs to be examined carefully. Mirror image studies, which compare the periods pre- and post-LAI introduction within subjects might be a more informative design to examine the effect of LAIs in the targeted population, even though mirror image studies have their own limitations.

Methods: A systematic review/meta-analysis was conducted of mirror image studies following patients at least 12 months (at least 6 months each on OAP and LAI). Co-primary outcomes were hospitalization rate and number of hospitalizations. Pooled risk ratio or rate ratio together with their 95% confidence intervals (CIs) were calculated, using random-effects model. Number-needed-to-treat (NNT) was calculated where appropriate. With regard to the heterogeneity, τ^2 , I^2 , Q , p values were reported.

11:15 a.m. - 1:30 p.m.
 Data Blitz Session
 Regency Ballroom 2

Long Acting Injectable vs. Oral Antipsychotics in Schizophrenia: A Systematic Review and Meta-analysis of Mirror-image Studies

Wednesday, Poster #175 (continued)

Taishiro Kishimoto

Results: We identified a total of 26 studies across 22 countries including 5,940 participants. LAIs showed strong superiority over OAPs in preventing a next hospitalization (18 studies, $n=2722$, risk ratio=0.44, 95%CI: 0.38-0.52, $p<0.001$, $NNT=3$; heterogeneity: $\tau^2=0.078$, $I^2=79\%$, $Q=81.4$, $df=17$, $p<0.001$). LAIs also showed strong superiority over OAPs in decreasing the number of hospitalizations (19 studies, 7034 person years, rate ratio=0.40, 95%CI: 0.31-0.51, $p<0.0001$; heterogeneity: $\tau^2=0.266$, $I^2=93.8\%$, $Q=288.2$, $df=18$, $p<0.001$). Although substantial heterogeneity was seen, all studies except one each consistently showed significant superiority regarding the rate and number of hospitalizations favoring of LAIs. This strong superiority remained across all of the following subgroups: first-generation antipsychotic-LAIs, second-generation antipsychotic-LAIs, studies published before 2000 and studies published after 2000, studies applying intention-to-treat analysis and those reporting observed cases. The extent to which publication bias might have contributed to these findings will be further discussed.

Conclusions: Result from mirror image studies in patients eligible for clinical use of LAIs showed strong superiority of LAIs compared to OAPs in preventing hospitalization. The results are in contrast with the meta-analysis of RCTs, which showed non-superiority of LAIs. However, given the possible biases in mirror image studies; i.e., expectation bias, time effect, etc., a cautious interpretation is required. Nevertheless, the population in mirror image studies better reflects the population receiving LAIs in clinical practice. Future RCTs may benefit from including patients at high-risk for relapse and those more closely reflecting routine clinical care.

References: [1] Rosenheck RA, Krystal JH, Lew R, Barnett PG, Fiore L, Valley D, Thwin SS, Vertrees JE, Liang MH; CSP555 Research Group., 2011. Long-acting risperidone and oral antipsychotics in unstable schizophrenia. *N Engl J Med.* 64(9):842-51. [2] Schooler NR, Buckley PF, Mintz J, et al. PROACTIVE: Initial

Long Acting Injectable vs. Oral Antipsychotics in Schizophrenia: A Systematic Review and Meta-analysis of Mirror-image Studies

Wednesday, Poster #175 (continued)

Taishiro Kishimoto

results of an RCT comparing long-acting injectable risperidone to 2nd generation oral antipsychotics. American College of Neuropsychopharmacology 50th annual meeting. Kona, Hawaii, USA; 2011 [3] Kishimoto T, Robenzadeh A, Leucht C, Leucht S, Watanabe K, Mimura M, Borenstein M, Kane JM, Correll CU. Long-acting injectable vs. oral antipsychotics for relapse prevention in schizophrenia: A meta-analysis of randomized trials.

1:30 p.m. - 3:00 p.m.

Associate Member Session

Regency Ballroom 1

Special Session for Associate Members

“Ask the Experts: Peer Review”

Panelists:

Bill Carlezon
Marlene Freeman
Bob Friedman

John Krystal
Lisa Monteggia
Nora Volkow

Peer review is a critical centerpiece of the academic process, determining which articles get published and which grants are funded. Despite its importance, there is little formal training in how to become a useful peer reviewer. This workshop will provide a panel discussion on how to perform an optimal peer review for journal articles and grant proposals. The panel will consist of editors of top psychiatric journals and NIH staff. The discussion will be moderated by members of the ACNP Member Advisory Task Force and will begin with a small number of structured questions followed by audience participation. Topics to be discussed will include, but not be limited to: Why should I review (I'm so busy!)? What are some tips for writing a good review? Are there any common mistakes/pitfalls to avoid? How long should a review be – should I mention every problem I see or just the major issues? What are the boundaries determining relevant conflict of interest? What should I do if I know something about the study (that affects my judgment) that isn't transparent in the proposal/article? How does one become an Editorial Board Member of a journal? Through this session, it is expected that participants will improve their understanding of how to perform a useful peer review, and will also gain a better understanding of the peer review process. It is expected that the latter understanding will enhance writing of articles and grants and responding to reviews.

PL

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

Rescuing Novel Mechanisms: Minimizing Placebo Response and Optimizing Signal Detection in Proof of Concept Trials

Chair: Michael Thase

Co-Chair: William Z. Potter

- 3:00 p.m. Decline in Signal Detection: Background and Proposed Strategies
Michael Thase
- 3:25 p.m. Missing Data, Placebo Response, and Positive Controls: How They Influence Signal Detection
Craig H. Mallinckrodt
- 3:50 p.m. First, Do No ... Help!?: The Problems with Therapeutic Alliance and Expectation Bias in Clinical Trials
Michael Detke

MP

4:15 p.m. – 5:30 p.m.

Mini Panel

Diplomat Ballroom 1 & 2

Exploring Therapeutic Use of Psilocybin, A Classic Hallucinogen

Chair: Roland Griffiths

- 4:15 p.m. Experimental Studies of Psilocybin in Healthy Volunteers:
Persisting Attribution of Positive Changes in Attitudes, Mood
and Behavior
Roland Griffiths
- 4:40 p.m. Psilocybin Treatment for Anxiety in Patients with Advanced-
Stage Cancer
Charles Grob
- 5:05 p.m. Effects of Psilocybin in the Treatment of Addictions: A Review
and Preliminary Results from Two Ongoing Trials
Michael P. Bogenschutz

MP

New Perspectives on the Role of Glutamatergic Neurotransmission in Alcoholism and Drug Addiction

Chair: Mary-Anne Enoch

- 3:00 p.m. Translational Support for the Glutamate Hypothesis of Addiction
Derik Hermann
- 3:30 p.m. The Effects of Chronic, Heavy Alcohol and Cocaine Use
on Glutamatergic Gene Expression in Postmortem Human
Hippocampus
Mary-Anne Enoch
- 4:00 p.m. Adaptations of Glutamatergic Transmission in Extended
Amygdala in Stress and Reward
Danny Winder
- 4:30 p.m. A Functional Grm2 Stop Codon Increases Alcohol Preference in
Alcohol Preferring (P) Rats
David Goldman
- 5:00 p.m. Discussant: *Gary Aston-Jones*

3:00 p.m. – 5:30 p.m.

Panel

Atlantic Ballroom 2

High Anxiety: Endocannabinoid Regulation of the Stress Response and Emotional Behavior

Chair: Alexander Neumeister

- 3:00 p.m. PET Reveals Abnormal CB1 Receptor Binding in PTSD
Alexander Neumeister
- 3:30 p.m. Reduced Plasma Endocannabinoid Levels in PTSD
Rachel Yehuda
- 4:00 p.m. The Endocannabinoid System as a Therapeutic Target for Stress-related Disorders
Daniele Piomelli
- 4:30 p.m. Stress-induced Regulation of Endocannabinoid Signaling in the Amygdala: Mechanisms and Functional Implications
Matt Hill
- 5:00 p.m. Discussant: *Ken Mackie*



Longitudinal Neuroimaging of Emerging Substance Use: Brain Indicators of Early Risk and Effects of Use

Chair: Mary Heitzeg
Co-Chair: Godfrey D. Pearlson

- 3:00 p.m. Individual Differences in Control and Reward and Their Relationship to Substance Use Risk: The IMAGEN Study
Hugh Garavan
- 3:30 p.m. Longitudinal fMRI Studies of Impulse Control and Incentive Responding: Effects of Risk and Alcohol Use
Mary Heitzeg
- 4:00 p.m. Effects of Alcohol use Initiation on Brain Structure and Behavioral Functions in Adolescents
Monica Luciana
- 4:30 p.m. Longitudinal Studies of Alcohol Effects on Academic Grades and MRI Hippocampal Volumes in the BARCS College Sample
Godfrey D. Pearlson
- 5:00 p.m. Discussant: *Edith Sullivan*

3:00 p.m. – 5:30 p.m.

Panel

Regency Ballroom 2

Neuropeptide Receptor Ligands in Psychiatric Diseases: New Hopes after Multiple Failures

Chair: Stephen Stahl

Co-Chair: Guy Griebel

- 3:00 p.m. Neuropeptides to Treat Affective Disorders: Did Animal Model
Fail to be Predictive, or did Clinical Research Fail to Detect
Effects?
Catherine Belzung
- 3:30 p.m. Neuropeptides and Major Depression/Depression-like Behavior:
Focus on Substance P and Galanin
Tomas Hokfelt
- 4:00 p.m. Hypocretin/orexin, Sleep and Narcolepsy: Immune and
Pharmacological Implications
Emmanuel Mignot
- 4:30 p.m. Identifying the Right Patient for Neuropeptide Receptor Ligands
- Biomarkers for Central CRH Overexpression
Marcus Ising
- 5:00 p.m. Discussant: *Thomas Steckler*

PA

Are We at a Turning Point in Psychiatric Genetics?

Chair: Kalpana Merchant
Co-Chair: David A. Collier

- 3:00 p.m. How Common and Rare Variants are Beginning to Provide Insights into Biological Mechanisms Underlying Psychiatric Disorders
David A. Collier
- 3:30 p.m. Dissecting Complexity in Neuropsychiatric Genetics with Network Inference
Neelroop N. Parikshak
- 4:00 p.m. Modelling Schizophrenia Using Induced Pluripotent Stem Cells
Kristen Brennand
- 4:30 p.m. Optogenetics and Psychiatric Disease: Focus on Social Behaviors
Karl Deisseroth
- 5:00 p.m. Discussant: *Kalpana Merchant*

3:00 p.m. – 5:30 p.m.

Panel

Atlantic Ballroom 3

**The Ups and Downs of AKT Signaling:
A Nexus of Risk for Psychiatric Disorders**

Chair: Daniel R. Weinberger

Co-Chair: Thomas F. Franke

- 3:00 p.m. Dissecting the Role of the AKT/PKB Family in
Neurodevelopment and Schizophrenia
Amanda Law
- 3:30 p.m. Integrated Approaches to Understand the Actions of GPCRs:
The β -arrestin-dependent D2R Signaling Axis
Marc G. Caron
- 4:00 p.m. DISC1 Regulation of Neural Development through
AKT-mTOR-CYFIP1 Signaling
Guo-li Ming
- 4:30 p.m. Studying AKT1 Signaling in Human Brain
Daniel R. Weinberger
- 5:00 p.m. Discussant: *Thomas F. Franke*

PA

Wednesday Morning At A Glance

7:00 AM – 8:30 AM	CDI Booster Session	Room 220
7:00 AM – 8:30 AM	ASCP Board of Director's Meeting	Room 201

7:30 AM	Morning Break	Grand Ballroom Lobby
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Mini-Panel Sessions

8:30 AM – 9:45 AM	Behavioral Paradigms to Improve Signal Detection in Trials of Cognition Enhancing Drugs	Diplomat Ballroom 1 & 2
9:45 AM – 11:00 AM	Pathology Driven Biomarker Development for Major Depressive Disorder: Bridging Central to Peripheral Markers	Diplomat Ballroom 1 & 2

Panel Sessions

8:30 AM – 11:00 AM	Molecular and Cellular Mechanisms Underlying Resilience in Mood and Other Social-psychological Stress-related Disorders: New Avenue for Novel Therapeutics?	Regency Ballroom 2
8:30 AM – 11:00 AM	Reward/Motivation Deficits in Attention Deficit Hyperactivity Disorder (ADHD) and the Effects of Medication	Regency Ballroom 1
8:30 AM – 11:00 AM	Opioid and Cannabinoid Mechanisms in Alcohol Addiction: Recent Evidence from Functional Brain Imaging	Regency Ballroom 3
8:30 AM – 11:00 AM	Beta-amyloid Neuropathology in Cognitively Normal Individuals: Preclinical Alzheimer's Disease or Cognitive Resilience?	Atlantic Ballroom 2
8:30 AM – 11:00 AM	Inhibition of Phosphodiesterases to Treat Psychiatric Disorders: Advances through Innovation in Preclinical Models and Feedback from the Clinic	Atlantic Ballroom 3
8:30 AM – 11:00 AM	Dendritic Spine Plasticity in Depression and Addiction	Atlantic Ballroom 1

11:15 AM – 12:30 PM	ACNP Business Meeting <i>(ACNP Fellows, Members, and Associate Members Only)</i>	Regency Ballroom 2
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PA-Panel MP-Mini Panel PL-Plenary SG-Study Group

Wednesday Afternoon At A Glance

12:30 PM – 2:00 PM	Lunch Buffet	Great Hall 1-4
12:30 PM – 2:00 PM	SOBP Program Committee Meeting	Diplomat Ballroom 4
12:30 PM – 2:00 PM	Travel Awardee Luncheon (<i>by Invitation Only</i>)	Great Hall 5

Panel Sessions

3:00 PM – 5:30 PM	Circadian Rhythms and Mood Disorders: Clock Genes and New Treatment Implications	Regency Ballroom 1
3:00 PM – 5:30 PM	Neuronal Circuit Regulation of Ventral Tegmental Area Neurons	Atlantic Ballroom 1
3:00 PM – 5:30 PM	Applying Translational Research and Imaging to Treatment Strategies in Alcoholism	Regency Ballroom 3
3:00 PM – 5:30 PM	Lesson from Animal Studies of Genetic Risk Factors for Psychiatric Disorders of Neurodevelopmental origin: How can we Move Forward with our Research for Novel Treatment Interventions?	Atlantic Ballroom 2
3:00 PM – 5:30 PM	Glucocorticoid Receptors as Pharmacologic Targets in Psychiatry	Atlantic Ballroom 3
3:00 AM – 5:30 PM	Anxiety Disorders: New Evidence for Structural and Functional Connectivity Abnormalities	Regency Ballroom 2
3:00 PM – 5:30 PM	Army STARRS Suicide Research: From Bench to Battlefield	Diplomat Ballroom 1 & 2

5:30 PM – 7:30 PM	Poster Session III with Reception	Great Hall 1-4
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7:30 PM – 9:00 PM	<i>Neuropsychopharmacology</i> Editorial Board	Diplomat Ballroom 4
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PA-Panel MP-Mini Panel PL-Plenary SG-Study Group

8:30 a.m. – 9:45 a.m.
Mini Panel
Diplomat Ballroom 1 & 2

Behavioral Paradigms to Improve Signal Detection in Trials of Cognition Enhancing Drugs

Chair: Richard Keefe
Co-Chair: Stephen R. Marder

- 8:30 a.m. The Phosphodiesterase 4 Inhibitor, HT-0712, Facilitates
Cognitive Rehabilitation Following Traumatic Brain Injury
Tim Tully
- 8:55 a.m. Cognitive Remediation with D-cycloserine Added to Cue
Exposure Therapy
A. Eden Evins
- 9:20 a.m. The Effects of Modafinil and Cognitive Training on Cognitive
Performance
Avi Reichenberg

MP

9:45 a.m. – 11:00 a.m.

Mini Panel

Diplomat Ballroom 1 & 2

Pathology Driven Biomarker Development for Major Depressive Disorder: Bridging Central to Peripheral Markers

Chair: Jeffrey Meyer

9:45 a.m. Targeting Monoamine Oxidase A as a Biomarker for Major Depressive Disorder

Jeffrey Meyer

10:10 a.m. Imaging the 18 kDa Translocator Protein (TSPO) in Vivo

Eugenii A. Rabiner

10:35 a.m. Protein Kinase A: Biomarker for Major Depression

Yogesh Dwivedi

MP

Molecular and Cellular Mechanisms Underlying Resilience in Mood and Other Social-psychological Stress-related Disorders: New Avenue for Novel Therapeutics?

Chair: Husseini K. Manji

- 8:30 a.m. The Lasting Legacy of Early Social Stress on the Epigenome
Dietmar Spengler
- 9:00 a.m. Blockade of the Inflammasome in Brain Produces Antidepressant Effects and Resilience to Stress
Ronald S. Duman
- 9:30 a.m. Plausible Roles of Bcl-2 Family Proteins in Cellular and Behavioral Resilience to Mood Disorders
Guang Chen
- 10:00 a.m. Mechanisms Underlying the Resilience to Severe Social Stress and the Role of Ventral Tegmental Area (VTA)
Ming-Hu Han
- 10:30 a.m. Discussant: *Steven E. Hyman*

8:30 a.m. – 11:00 a.m.

Panel

Regency Ballroom 1

Reward/Motivation Deficits in Attention Deficit Hyperactivity Disorder (ADHD) and the Effects of Medication

Chair: Wilson M. Compton

Co-Chair: James Swanson

- 8:30 a.m. Functional Connectivity of Reward Circuits in the Rat and Human Brain
Elliot A. Stein
- 8:30 a.m. Reward Circuitry, Risky Behaviors, and ADHD
Francisco Xavier Castellanos
- 9:30 a.m. Dopamine Reward Circuitry in Attention Deficit Disorder
Nora D. Volkow
- 10:00 a.m. Altered Sensitivity to Reinforcement in Individuals with ADHD: Implications for the Development of Aberrant Health Behaviors
Scott H. Kollins
- 10:30 a.m. Discussant: *James Swanson*

PA

Opioid and Cannabinoid Mechanisms in Alcohol Addiction: Recent Evidence from Functional Brain Imaging

Chair: Kent Hutchison

- 8:30 a.m. The Effects of Alcohol Consumption on Endogenous Opioid Release in the Human Orbitofrontal Cortex and Nucleus Accumbens
Jennifer Mitchell
- 9:00 a.m. Influence of Mu-Opioid Receptor (OPRM1) A118G Polymorphism on Pharmacological Effects of Alcohol: A Translational Approach
Vijay A. Ramchandani
- 9:30 a.m. CNR1 Variation is Associated with BOLD Response to Alcohol Cues and Alcohol Dependence Symptom Count
Kent Hutchison
- 10:00 a.m. Reduced Cannabinoid CB1 Receptor Binding in Alcohol Dependence Measured with Positron Emission Tomography
Markus Heilig
- 10:30 a.m. Discussant: *Raye Litten*

8:30 a.m. – 11:00 a.m.

Panel

Atlantic Ballroom 2

Beta-amyloid Neuropathology in Cognitively Normal Individuals: Preclinical Alzheimer's Disease or Cognitive Resilience?

Chair: Susan M. Resnick

Co-Chair: Dean F. Wong

- 8:30 a.m. The Detectability of Ab Amyloid by PET Imaging in Mouse Models of Alzheimer's Disease with Different Rate of Plaque Accumulation
Alena Savonenko
- 9:00 a.m. Preclinical AD: Evidence for Amyloid-associated Alterations in Brain Function and Structure
Reisa Sperling
- 9:30 a.m. Neuroimaging Predictors of Cognitive Impairment and Resilience: Insights from the Baltimore Longitudinal Study of Aging
Susan M. Resnick
- 10:00 a.m. Resilient Brain Aging: Neuropathological, Cellular and Biochemical Features of Pathological Alzheimer's Disease with Normal Cognition
Steven E. Arnold
- 10:30 a.m. Discussant: *Steven T. DeKosky*

PA

**Inhibition of Phosphodiesterases to Treat Psychiatric Disorders:
Advances through Innovation in Preclinical Models and
Feedback from the Clinic**

Chair: Nicholas Brandon

- 8:30 a.m. The Phosphodiesterase Isoform 4A5 (PDE4A5) is the Critical Mediator of Hippocampus-dependent Cognitive Impairments Induced by Sleep Loss
Ted Abel
- 9:00 a.m. Alcohol Drinking and Seeking Behaviors: Role of Phosphodiesterase-4 (PDE4)
Han-Ting Zhang
- 9:30 a.m. Phosphodiesterases Differentially Determine the Spatial and Temporal Modalities of cAMP Signal Integration in the Cortex and Striatum.
Pierre Vincent
- 10:00 a.m. Inhibition of Phosphodiesterase10A for the Treatment of Schizophrenia: Preclinical Rationale and Clinical Evaluation
Christopher J. Schmidt
- 10:30 a.m. Discussant: *Akira Sawa*

8:30 a.m. – 11:00 a.m.

Panel

Atlantic Ballroom 1

Dendritic Spine Plasticity in Depression and Addiction

Chair: Eric J. Nestler

Co-Chair: Scott Russo

- 8:30 a.m. Molecular Basis of Structural Plasticity of Nucleus Accumbens
Neurons Induced by Drugs of Abuse
Eric J. Nestler
- 9:00 a.m. Epigenetic Regulation of Synaptic Remodeling in Depression
Scott Russo
- 9:30 a.m. Subcellular Synaptic Connectivity in the Nucleus Accumbens
Adam Carter
- 10:00 a.m. Opposing Effects of Fear Conditioning and Extinction on
Dendritic Spine Remodeling in the Mouse Cortex
Wenbiao Gan
- 10:30 a.m. Discussant: *John Morrison*

PA

3:00 p.m. – 5:30 p.m.
Panel
Regency Ballroom 1

Circadian Rhythms and Mood Disorders: Clock Genes and New Treatment Implications

Chair: William Bunney

Co-Chair: Ellen Frank

- 3:00 p.m. Circadian Clock Genes in Control and Major Depressive Disorder Brain Tissue: Potential Role in Mode of Action of Ketamine
William Bunney
- 3:30 p.m. Patients' Self-reported Chronotype and Social Rhythm Changes after Treatment of Major Depression
Ellen Frank
- 4:00 p.m. Use of the Clock Mutant Mice to Identify New Mood Stabilizing Agents
Colleen A. McClung
- 4:30 p.m. The Circadian Transcriptional Network in Mammals
Joseph S. Takahashi
- 5:00 p.m. Discussant: *David J. Kupfer*

3:00 p.m. – 5:30 p.m.

Panel

Atlantic Ballroom 1

Neuronal Circuit Regulation of Ventral Tegmental Area Neurons

Chair: Garret Stuber

- 3:00 p.m. Subcellular Segregation of Dopamine and Glutamate Signaling
by a Subset of Ventral Tegmental Area Neurons
Marisela Morales
- 3:30 p.m. Anatomically-specific Ventral Tegmental Area Afferents Control
Reward and Aversion
Garret Stuber
- 4:00 p.m. Plasticity and Function of Distinct Subtypes of Dopamine
Neurons
Robert Malenka
- 4:30 p.m. Dopamine Neurons Modulate the Neural Encoding and
Expression of Depression-related Behavior
Kay M. Tye
- 5:00 p.m. Discussant: *Antonello Bonci*

PA

Applying Translational Research and Imaging to Treatment Strategies in Alcoholism

Chair: Rajita Sinha

- 3:00 p.m. Translational Neuroimaging Studies of Alcoholism: From Rats to Man
Adolf Pfefferbaum
- 3:30 p.m. Brain Mechanisms of Behavioral Changes Promoting Relapse in Alcohol Dependent Individuals: A Translational Approach
Theodora Duka
- 4:00 p.m. Altered Prefrontal Structure and Function Predicts Heavy Drinking and Alcohol Relapse: Are there Clues for Novel Treatment Strategies?
Rajita Sinha
- 4:30 p.m. Striatal-limbic Suppression during Anticipatory Anxiety in Alcohol-dependent Men
Bryon Adinoff
- 5:00 p.m. Discussant: *Charles P. O'Brien*

3:00 p.m. – 5:30 p.m.

Panel

Atlantic Ballroom 2

**Lesson from Animal Studies of Genetic Risk Factors for
Psychiatric Disorders of Neurodevelopmental Origin:
How can we Move Forward with our Research for Novel
Treatment Interventions?**

Chair: Atsushi Kamiya

- 3:00 p.m. Insulin-like Growth Factor 1 Therapy in Rett Syndrome: From
Animal Studies to Clinic
Daniela Tropea
- 3:30 p.m. Modeling Lissencephaly-From Pathogenesis to Therapies
Anthony Wynshaw-Boris
- 4:00 p.m. Regulatory Role of DISC1 for Excitatory Action of GABA
Signaling in Prefrontal Cortex Development and Function
Atsushi Kamiya
- 4:30 p.m. Pathological Mechanisms of Aberrant Neuregulin Signaling
Revealed by Temporal Control of Expression
Lin Mei
- 5:00 p.m. Discussant: *Patricio O'Donnell*

PA

Glucocorticoid Receptors as Pharmacologic Targets in Psychiatry

Chair: Alan F. Schatzberg

- 3:00 p.m. Corticosteroid-dependent Plasticity Mediates Compulsive Alcohol Drinking in Rats
George F. Koob
- 3:30 p.m. Working Memory is Modulated by Glucocorticoid Receptor and Dopaminergic Genes
Wissam El-Hage
- 4:00 p.m. A Human Laboratory Study of Mifepristone Treatment for Alcohol Dependence
Barbara J. Mason
- 4:30 p.m. The Role of Glucocorticoid Receptors in Bipolar Disorder
Allan H. Young
- 5:00 p.m. Discussant: *Mary L. Phillips*

3:00 p.m. – 5:30 p.m.

Panel

Regency Ballroom 2

Anxiety Disorders: New Evidence for Structural and Functional Connectivity Abnormalities

Chair: Ned H. Kalin

Co-Chair: Jennifer Blackford

- 3:00 p.m. Structural and Functional Alterations Predict Individual Differences in Behavioral Inhibition
Andrew S. Fox
- 3:30 p.m. Intrinsic Connectivity Abnormalities in Social Anxiety
Jennifer Blackford
- 4:00 p.m. Frontolimbic Connectivity in Generalized Anxiety Disorder
Jack B. Nitschke
- 4:30 p.m. Targeting the Medial Prefrontal Cortex in the Treatment of Pediatric Anxiety
Danny Pine
- 5:00 p.m. Discussant: *Scott Rauch*



Army STARRS Suicide Research: From Bench to Battlefield

Chair: Murray B. Stein
Co-Chair: Robert Ursano

- 3:00 p.m. The Army STARRS Study Plan
Robert Ursano
- 3:30 p.m. Executive Functioning and Suicidal Behavior among Soldiers:
Results from the Army STARRS Study
Matthew Nock
- 4:00 p.m. A Beating of Minds: Suicide and Traumatic Brain Injury
Murray B. Stein
- 4:30 p.m. TBI and Medical Illness as Predictors of Suicide Risk in US
Army Soldiers
Michael Schoenbaum
- 5:00 p.m. Discussant: *Thomas Insel*

Thursday Morning At A Glance

7:00 AM	Morning Break	Grand Ballroom Lobby
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7:00 AM – 8:00 AM	ACNP/AsCNP/CINP/ECNP/JSNP Meeting	Room 212
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Mini Panel Sessions

8:00 AM – 9:15 AM	Beyond Ketamine, Can Selective Targeting of the NMDA Receptor Produce Antidepressant Response without Psychotomimetic Effects: Clinical Results with Three Novel Compounds	Diplomat Ballroom 1 & 2
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9:15 AM – 10:30 AM	Beyond the NMDA Receptor-Alternative Glutamatergic Targets for Antidepressant Treatment	Diplomat Ballroom 1 & 2
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Panel Sessions

8:00 AM – 10:30 AM	Hippocampus and Addiction: New Neurons, New Circuits, and New Responses	Regency Ballroom 3
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8:00 AM – 10:30 AM	Neural Networks across Development in Health, Anxiety/Depression, and Treatment Implications	Regency Ballroom 2
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8:00 AM – 10:30 AM	Harnessing Cortical Plasticity for Therapeutic Purposes	Regency Ballroom 1
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8:00 AM – 10:30 AM	Balancing Benefits, Risks and Cost for New Treatments in Vulnerable Populations: Lessons from Child Psychiatry on the Need for a New Standard of Diverse Methodologies	Atlantic Ballroom 3
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8:00 AM – 10:30 AM	Metabotropic Glutamate Receptors (mGluRs) and Addiction	Atlantic Ballroom 2
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8:00 AM – 10:30 AM	Affective Neuroscience of Young Monkeys to Developing Population: Translational Studies of Brain Function Informing Interventions	Atlantic Ballroom 1
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9:00 AM – 12:00 PM	ACNP Council Meeting	Room 319-320
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10:30 AM – 12:00 PM	Brunch	Grand Ballroom Lobby
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PA-Panel MP-Mini Panel PL-Plenary SG-Study Group

Thursday Afternoon At A Glance

Panel Sessions

12:00 PM – 2:30 PM	Functional and Structural Alterations in the Insula are Central to the Pathophysiology of Both Anorexia Nervosa and Obesity	Diplomat Ballroom 1 & 2
12:00 PM – 2:30 PM	Neuroimaging Predictors of Treatment Effects in High-risk and Bipolar Individuals across the Lifespan	Atlantic Ballroom 2
12:00 PM – 2:30 PM	Sink or Swim: Take Your Raft and Fyns Down the STEPs to Navigate NMDA Receptor Pools in Neuropsychiatric Disorders	Atlantic Ballroom 3
12:00 PM – 2:30 PM	Non-invasive Brain Modulation to Enhance Inhibitory Control and Drive in Psychiatric Disorders: a Translational Approach with a Focus on Dopaminergic Modulation of Fronto-striatal Pathways	Regency Ballroom 2
12:00 PM – 2:30 PM	Neuronal Mechanisms for Behavioral and Psychiatric Vulnerability in Adolescents	Atlantic Ballroom 1
12:00 PM – 2:30 PM	The Orexins: Bench to Bedside and Beyond	Regency Ballroom 3
12:00 PM – 2:30 PM	Microdomain-Specific Proteome Abnormalities in Severe Mental Illness	Regency Ballroom 1

Beyond Ketamine, Can Selective Targeting of the NMDA Receptor Produce Antidepressant Response without Psychotomimetic Effects: Clinical Results with Three Novel Compounds

Chair: Gerard Sanacora

- 8:00 a.m. Beyond Ketamine: Next Generation NMDA Antagonists Show Rapid Antidepressant Effects, without Psychotomimetic Effects
Nancy Diazgranados
- 8:25 a.m. A Phase 2, Randomized, Double Blind, Single Intravenous Dose Study of GLYX-13, an NMDA Receptor Glycine Site Functional Partial Agonist, in Subjects with Major Depressive Disorder with Inadequate Response to Antidepressant Medication
Ronald M. Burch
- 8:50 a.m. Randomized Trial of AZD6765, an N-methyl-D-aspartate (NMDA) Channel Blocker, as Adjunct Treatment for Major Depression
Sanjeev Pathak

9:15 a.m. – 10:30 a.m.

Mini Panel

Diplomat Ballroom 1 & 2

Beyond the NMDA Receptor-Alternative Glutamatergic Targets for Antidepressant Treatment

Chair: Yogesh Dwivedi
Co-Chair: P. Jeffrey Conn

- 9:15 a.m. Altered Affective Behavior in Kainate Receptor Knockout Mice
Anis Contractor
- 9:40 p.m. RNA Editing of an AMPA Receptor Subunit is Altered in Major
Depression and Suicide
Monsheel Sodhi
- 10:05 a.m. Selective mGlu5 NAMs for the Treatment of MDD
Carrie K. Jones

Hippocampus and Addiction: New Neurons, New Circuits, and New Responses

Chair: Rita A. Fuchs

- 8:00 a.m. Role of the Dorsal Hippocampus in the Reconsolidation and Utilization of Associative Memories that Maintain Drug Context-induced Cocaine Seeking
Rita A. Fuchs
- 8:30 a.m. Individual Differences in Substance Abuse Liability: Implicating the Hippocampus
Huda Akil
- 9:00 a.m. Ventral Hippocampal Regulation of Medial VTA Dopamine System and its Role in Addiction
Anthony A. Grace
- 9:30 a.m. Linking Context with Reward: Hippocampal and Septal Circuit Projections to Ventral Tegmental Area Play Critical Roles in Cocaine Relapse
Gary Aston-Jones
- 10:00 a.m. Discussant: *Nora D. Volkow*

8:00 a.m. – 10:30 a.m.

Panel

Regency Ballroom 2

Neural Networks across Development in Health, Anxiety/ Depression, and Treatment Implications

Chair: Monique Ernst

- 8:00 a.m. Overcoming the Detrimental Effects of Motion on Resting State fMRI
Michael Milham
- 8:30 a.m. Altered Intrinsic Connectivity and Error-processing Function of Salience Network in Pediatric Obsessive Compulsive Disorder
Kate D. Fitzgerald
- 9:00 a.m. What can Amygdala Functional Connectivity Tell Us about the Development of Anxiety Disorders?
Amy K. Roy
- 9:30 a.m. From Correlation to Causation in Resting-state fMRI: Network Dynamics in Psychopathology and with Concurrent TMS/fMRI
Amit Etkin
- 10:00 a.m. Discussant: *Angus W. MacDonald*



Harnessing Cortical Plasticity for Therapeutic Purposes

Chair: Sophia Vinogradov

- 8:00 a.m. Shaping Brain Circuits with Auditory Experience
Etienne de Villers-Sidani
- 8:30 a.m. Directing Cortical Plasticity to Understand and Treat
Neurological Disease
Michael P. Kilgard
- 9:00 a.m. Long-lasting Enhancement of Visual Perceptual Learning in
Healthy Humans by the Cholinesterase Inhibitor Donepezil
Michael A. Silver
- 9:30 a.m. Harnessing Prefrontal Plasticity through Videogame Training to
Address Multitasking Deficits across the Adult Lifespan
Adam Gazzaley
- 10:00 a.m. Discussant: *Akira Sawa*

8:00 a.m. – 10:30 a.m.

Panel

Atlantic Ballroom 3

Balancing Benefits, Risks and Cost for New Treatments in Vulnerable Populations: Lessons from Child Psychiatry on the Need for a New Standard of Diverse Methodologies

Chair: Christoph U. Correll

Co-Chair: John W. Newcomer

- 8:00 a.m. One Year Follow-up Longitudinal Study with a Large Sample of Antipsychotic-naïve Children and Adolescents
Celso Arango
- 8:30 a.m. The Value of Randomized Clinical Trials: Data from the Metabolic Effects of Antipsychotics in Children (MEAC) Study
Ginger E. Nicol
- 9:00 a.m. Mixed Methods in Health Services Research to Understand Real-world Acceptance of Safety Recommendations for Psychopharmacologic Treatments
Elaine H. Morrato
- 9:30 a.m. Economic Evaluation in Child Psychiatry---An Example from the Metabolic Effects in Antipsychotic Treatment of Children Study
Steven M. Kymes
- 10:00 a.m. Discussant: *John W. Newcomer*



Metabotropic Glutamate Receptors (mGluRs) and Addiction

Chair: Jill B. Becker

- 8:00 a.m. Estradiol Influences Dopamine and GABA Release in the Striatum via mGluR5
Jill B. Becker
- 8:30 a.m. Estrogen Receptors Located at the Surface Membrane Activate Metabotropic Glutamate Receptor Signaling
Paul G. Mermelstein
- 9:00 a.m. Unique Roles for Ventral and Dorsal Striatum mGluR5 in Extinction Learning and Relapse to Cocaine Seeking
Lori A. Knackstedt
- 9:30 a.m. Restoration of Infralimbic mGluR2 Deficit Rescues Control Over Drug Seeking in Alcohol Dependence
Wolfgang H. Sommer
- 10:00 a.m. Discussant: *M. Foster Olive*

8:00 a.m. – 10:30 a.m.

Panel

Atlantic Ballroom 1

**Affective Neuroscience of Young Monkeys to Developing
Population: Translational Studies of Brain Function Informing
Interventions**

Chair: Mani Pavuluri

Discussant: Richard Davidson

- 8:30 a.m. Identification of Novel Targets in the Developing Primate
Amygdala for the Early Treatment of Childhood Anxiety
Ned H. Kalin
- 8:30 a.m. Translational Imaging Studies of Natural Products as Treatments
for Pediatric Depression
Perry Renshaw
- 9:00 a.m. Brain Functional Mechanisms of Treating Pediatric Mania
Mani Pavuluri
- 9:30 a.m. Amygdala Activation and Prefrontal Cortex Functional
Connectivity: Potential Targets for Treatment of Autism
Spectrum Disorders
Christopher S. Monk
- 10:00 a.m. Discussant: *Richard Davidson*



Functional and Structural Alterations in the Insula are Central to the Pathophysiology of Both Anorexia Nervosa and Obesity

Chair: Guido KW. Frank

Co-Chair: Walter Kaye

- 12:00 p.m. The Role of the Insular Cortex in Flavor Preference Formation
Dana Small
- 12:30 p.m. Pain and Pending Pictures: Increased Insula Response in Anorexia Nervosa
Alan Simmons
- 1:00 p.m. Regional Gray Matter Volumes in the Insula Distinguish Anorexia Nervosa and Obesity
Guido KW. Frank
- 1:30 p.m. Circuits Connecting Somatic/Visceral-Related Insular Areas with Eating/Reward Areas in the Ventromedial Prefrontal Cortex
Joseph L. Price
- 2:00 p.m. Discussant: *Martin Paulus*

12:00 p.m. – 2:30 p.m.

Panel

Atlantic Ballroom 2

Neuroimaging Predictors of Treatment Effects in High-risk and Bipolar Individuals across the Lifespan

Chair: Caleb Adler

- 12:00 p.m. Neurobiological and Genetic Risk Factors for Antidepressant-induced Mania in Youth at Risk for Bipolar Disorder
Kiki Chang
- 12:30 p.m. Neurofunctional Effects of Ziprasidone in Manic Adolescents with Bipolar Disorder
Melissa DelBello
- 1:00 p.m. Neurophysiological Effects of Bipolar Medications across Mood State
Caleb Adler
- 1:30 p.m. White Matter Correlates of Antipsychotic and Lithium Response in Bipolar Disorder: A Meta-analysis and Meta-regression of Diffusion Tensor Imaging findings
Sophia Frangou
- 2:00 p.m. Discussant: *Ellen Leibenluft*



Sink or Swim: Take Your Raft and Fyns Down the STEPs to Navigate NMDA Receptor Pools in Neuropsychiatric Disorders

Chair: Jacqueline F. McGinty

- 12:00 p.m. Genetic Manipulation of Striatal Enriched Phosphatase (STEP) Rescues Behavioral Abnormalities and seizures in a Mouse Model of Fragile X
Janice R. Naegele
- 12:30 p.m. Neuroprotective Role of STEP, a Brain-enriched Tyrosine Phosphatase, in Focal Cerebral Ischemia
Surojit Paul
- 1:00 p.m. Fyn, Tyrosine Phosphatases and Alcohol Drinking Behaviors
Dorit Ron
- 1:30 p.m. The Role of GluN2B Receptors and STEP in the ERK Shutoff Induced by Cocaine Self Administration in Rats
Jacqueline F. McGinty
- 2:00 p.m. Discussant: *Paul J. Lombroso*

12:00 p.m. – 2:30 p.m.

Panel

Regency Ballroom 2

Non-invasive Brain Modulation to Enhance Inhibitory Control and Drive in Psychiatric Disorders: a Translational Approach with a Focus on Dopaminergic Modulation of Fronto-striatal Pathways

Chair: Rita Goldstein

- 12:00 p.m. The Effects of Modafinil and Methylphenidate in Neuropsychiatric Disorders
Barbara J. Sahakian
- 12:30 p.m. Oral Methylphenidate Improves Inhibitory Control and Resting-state Functional Connectivity in Cocaine Addiction: An fMRI Study
Rita Goldstein
- 1:00 p.m. Noninvasive Nonpharmacological Approach to Modulate Cognition and Decision Making in Addiction
Felipe Fregni
- 1:30 p.m. Dopaminergic Modulation in a Preclinical Model of Risky Decision Making
Barry Setlow
- 2:00 p.m. Discussant: *Ruben Gur*



Neuronal Mechanisms for Behavioral and Psychiatric Vulnerability in Adolescents

Chair: Bitá Moghaddam
Co-Chair: Patricio O'Donnell

- 12:00 p.m. Neuronal Processing Differences in the Orbitofrontal Cortex and Striatum of Adolescents and Adults during Motivated Behavior
Bitá Moghaddam
- 12:30 p.m. Reward Network in Adolescents: Longitudinal Data and Functional Connectivity
Monique Ernst
- 1:00 p.m. Adolescent Maturation of Cortico-accumbens Circuits and Risk for Addictive Behavior
Patricio O'Donnell
- 1:30 p.m. Developmental Impairment of Local Prefrontal GABAergic Circuits by Altered Glutamatergic Transmission during Adolescence
Kuei Y. Tseng
- 2:00 p.m. Discussant: *Linda P. Spear*

12:00 p.m. – 2:30 p.m.

Panel

Regency Ballroom 3

The Orexins: Bench To Bedside and Beyond

Chair: David Michelson

- 12:00 p.m. The Hypocretin/Orexin System: Neuropeptides Involved in Sleep/Wake and Multiple Other Functions
Thomas S. Kilduff
- 12:30 p.m. Role of Hypocretin/Orexin Receptors in Arousal Control
Luis de Lecea
- 1:00 p.m. Efficacy and Safety of Suvorexant, an Orexin Receptor Antagonist, in Patients with Primary Insomnia: Results from Three Phase 3 Trials
W. Joseph Herring
- 1:30 p.m. Orexin Agonists and Antagonists Effects beyond Insomnia
Thomas Roth
- 2:00 p.m. Discussant: *Andrew Krystal*

Microdomain-Specific Proteome Abnormalities in Severe Mental Illness

Chair: Robert McCullumsmith

- 12:00 p.m. Playing in Traffic: Protein Trafficking and Membrane Domains
in Polarized Cells
Bettina Winckler
- 12:30 p.m. Identification and Characterization of a Putative Subcellular
Microdomain: Evidence for Disruption of the Coupling
of Glutamate Transporters and Glycolytic Enzymes with
Mitochondria in Schizophrenia
Robert McCullumsmith
- 1:00 p.m. Evidence from Proteomic Analysis of Schizophrenia Implicates
the Cellular Process of Clathrin Mediated Endocytosis in
Schizophrenia
David Cotter
- 1:30 p.m. PSD Protein Partitioning is Altered in the DLPFC of
Schizophrenia
Chang-Gyu Hahn
- 2:00 p.m. Discussant: *Joseph Coyle*



Poster Session I – Monday



Advocacy Affiliate - International Mental Health Resource Organization
“IMHRO Rising Star Awards”

Cindy Dyar, Brandon Staglin

1. Membership Advisory Task Force: Feedback, Challenges, and Solutions in 2012

Katherine E. Burdick, Linda Carpenter, Marlene Freeman, Vaishali Bakshi, Paul Holtzheimer, Lisa Monteggia, Thomas Schulze, Carlos Bolanos-Guzman, Kristin Cadenhead, Raymond Cho, Cynthia Crawford, Paul Kenny, Gregory Light, Gonzalo Laje

2. Blockade of Kappa Opioid Receptors Reduces Footshock Effects on Startle

Ashlee Van't Veer, Anita Bechtholt-Gompf, Sara Onvani, David Potter, Yujun Wang, Elena Chartoff, Uwe Rudolph, Lee-Yuan Liu-Chen, F. Ivy Carroll, Bruce Cohen, William Carlezon

3. Impaired Mesolimbic Regulation of Prefrontal Glutamate and Acetylcholine Release Accompanies Cognitive Inflexibility in Two Animal Models of Schizophrenia

Michelle Pershing, Dave Bortz, Ana Pocivavsek, Martin Sarter, Robert Schwarcz, John P. Bruno

4. Transition to Dorsolateral Striatal Dopamine Control of Cocaine Seeking Behavior is Predicted by Trait Impulsivity

Jennifer E. Murray, Ruth Dilleen, Yann Pelloux, Daina Economidou, Emily R. Jordan, Jeffrey W. Dalley, David Belin, Barry J. Everitt

5. Basolateral and Central Nuclei of the Amygdala Required for the Transition to Dorsolateral Dopamine Control over Habitual Cocaine Seeking

Jennifer E. Murray, David Belin, Barry J. Everitt

Poster Session I—Monday

6. GAD67 Downregulation in Specific Interneuronal Subpopulations Leads to Distinct Behavioral Phenotypes
Martin Schmidt, Krassimira Garbett, Szatmar Horvath, Karoly Mirnics
7. Brief Repeated Cortico-striatal Hyperstimulation Generates Chronic OCD-relevant Behavior
Susanne E. Ahmari
8. Deficiency of Schnurri-2, an MHC Enhancer Binding Protein, Induces Mild Chronic Inflammation in the Brain and Confers Molecular, Neuronal, and Behavioral Phenotypes Related to Schizophrenia
Tsuyoshi Miyakawa, Keizo Takao, Katsunori Kobayashi, Hideo Hagihara, Koji Ohira, Hirotaka Shoji, Satoko Hattori, Hisatsugu Koshimizu, Juzoh Umemori, Shun Yamaguchi, Tsuyoshi Takagi, Noah Walton, Hidenori Suzuki, Mitsuyuki Matsumoto, Shunsuke Ishii
9. Serine Racemase Knockout Mice, a Genetic Model of NMDA Receptor Hypofunction, Exhibit Impaired Hippocampal Neuroplasticity that can be Rescued by D-serine Treatment
Darrick T. Balu, Yan Li, Matthew D. Puhl, Vadim Bolshakov, Joseph Coyle
10. Inflammatory Th17 Cells Promote Depression-like Behavior in Mice
Eleonore Beurel, Laurie Harrington, Richard S. Jope
11. Effects of Baclofen and Naltrexone, Alone and in Combination, on Binge Eating in Rats
Nicole M. Avena, Mark S. Gold
12. Cariprazine Exhibits Dopamine D3 Receptor-Dependent Antidepressant-like Activity in the Chronic Unpredictable Stress Model of Anhedonia
Ronald S. Duman, Vanja Duric, Mounira Banasr, Nika Adham, Béla Kiss, István Gyertyan

Poster Session I—Monday

13. The Peripheral Immune System Functionally Contributes to Susceptibility to Repeated Social Defeat Stress

Georgia E. Hodes, Sam A. Golden, Daniel J. Christoffel, Madeline Pfau, Mitra Heshmati, Marylene Leboeuf, Miriam Merad, Scott Russo

14. Distinct CRF Protein Expression Patterns in Two Different CRF Overexpressing Mouse Models

Debra Bangasser, Zach Plona, Jodi Gresack, Mate Toth, Isabelle Mansuy, Emilio Merlo-Pich, Victoria Risbrough, Rita Valentino

15. Risk-taking in Adolescence: Relationships with Cocaine Self-administration and Involvement of Dopamine Signaling

Barry Setlow, Marci Mitchell, Virginia Weiss, Sofia Beas, Drake Morgan, Jennifer Bizon

16. The Hyperactive (HYPER) Rat: A Potential Animal Model of Bipolar Disorder

Jay M. Weiss, Katherine A. Boss-Williams

17. Opposite Effects of Tolcapone on Amphetamine-disrupted Startle Gating in Low vs. High COMT-expressing Rat Strains

Michelle R. Breier, Samantha R. Hines, Sebastian D. Herrera, Martin Weber, Neal R. Swerdlow

18. Astrocyte-specific Ablation in the Mouse Prefrontal Cortex Induces Depressive-like and Anxiety-like Deficits

Mounira Banasr, Meiyu Xu, Gerard Sanacora, Christopher Pittenger, Ronald S. Duman

Poster Session I—Monday

19. Optogenetic Control of Serotonergic Neurons and Anxiety-like Behavior

Yu Ohmura, Kenji Tanaka, Akihiro Yamanaka, Mitsuhiro Yoshioka

20. Depressive-like Phenotype in Transgenic Mice with CNS Overexpression of IL-6

Stacey J. Sukoff Rizzo, Zoe A. Hughes, Sarah Neal, J. Michael Roos, Sharon Rosenzweig-Lipson, Stephen J. Moss, Nicholas Brandon

21. Progress of the Lilly/Pfizer Partnership to Tackle Preclinical Model Development in Psychiatry: Does Chronic ACTH Treatment Render Mice Resistant to Antidepressants?

Zoe A. Hughes, Jeffrey M. Witkin, Taleen Hanania, Afshin Ghavami, Nicholas Brandon, David Bleakman, Kurt Rasmussen

22. Witnessing Social Defeat Induces an Anxiety and Depression-like State and Increases Nicotine Consumption

Brandon Warren, Lyonna Alcantara, Vincent Vialou, Eric J. Nestler, Carlos A. Bolanos-Guzman

23. Bacille Calmette Guérin Induces a Depressive Phenotype in ‘Susceptible’ Animals that is Sensitive to Antidepressants and Accompanied by Hypersensitivity to Pain Stimuli: A Preclinical Model of Comorbid Pain and Depression?

Brian Platt, Janet Schulenberg, Nicole Klee, Maryam Nizami, Bradley Nash, Urey Chow, James Barrett, Janet Clark

24. First Hospitalization Manic Youth Show Functional Alterations during a Task of Sustained Attention

Marguerite R. Schneider, Wade Weber, Jeffrey Welge, Caleb Adler, Strakowski Stephen, Melissa DelBello

Poster Session I—Monday

25. Subjective Cognitive Impairment, the Pre-mild Cognitive Impairment Stage of Eventual Alzheimer's Disease: Prospective Behavioral Markers of 2 Year Decline

Barry Reisberg, Carol Torossian, Ricardo Osorio, Santosh Ghimire, Kamalika Roy, Pravesh Sharma, Isabel Monteiro, Melanie B. Shulman, Iryna Lobach

26. Increased Risk for Suicide among Schizophrenia Patients with High Premorbid IQ

Mark Weiser, Ori Kapara, Nomi Werbeloff, Eyal Fruchter, Rinat Yoffe, Michael Davidson

27. Glutamate in the Associative Striatum Decreases after 4 Weeks of Antipsychotic Treatment in First-episode Psychosis: A Longitudinal 1H-MRS Study

Camilo de la Fuente, Pablo León-Ortiz, Mariana Azcárraga, Sylvana Stephano, Rafael Favila, Leonardo Díaz-Galvis, Patricia Alvarado-Alanis, Jesús Ramírez-Bermúdez, Ariel Graff-Guerrero

28. The Promigratory Chemokine CXCL12 is Negatively Related to Neuroinflammation in the Prefrontal Cortex of People with Schizophrenia

Samantha J. Fung, Stu Fillman, Cyndi Shannon Weickert

29. AAV Suppression of Dopamine D1 Receptors in the Striatum Impairs Probabilistic Learning via Affecting Reward-associations

Jared W. Young, Kerin Higa, Baohu Ji, David E. Nichols, Mark A. Geyer, Xianjin Zhou

30. Effects of Oxytocin on Social Cognition and Olfaction in Adults with Schizophrenia and Healthy Subjects

Josh Woolley, Brandon Chuang, Olivia Lam, Kate Rankin, Daniel H. Mathalon, Sophia Vinogradov

Poster Session I—Monday

31. Neuroanatomical Correlates of Apathy in Late Life Depression and Antidepressant Treatment Response

Genevieve S. Yuen, Faith Gunning-Dixon, Eric Woods, Matthew Hoptman, George S. Alexopoulos

32. Central Oxytocin in Social and Cued Fear Conditioning in Mice: Specific Role of the Dorsolateral Septum

Iulia Toth, David A. Slattery, Inga D. Neumann

33. Brain-derived Neurotrophic Factor, Interleukin-6, and Salivary Cortisol Levels in Patients with Major Depressive Disorder Treated With Desvenlafaxine vs Placebo

Philip Ninan, Richard C. Shelton, Weihang Bao, Christine Guico-Pabia

34. Baseline Working Memory Abnormalities in Current Major Depression as Detected by Magnetoencephalography

Mark J. Niciu, Allison C. Nugent, Craig Marquardt, Tom Holroyd, Giacomo Salvatore, Maura L. Furey, Carlos A. Zarate

35. Detecting Activity-evoked pH Changes in Human Brain

John A. Wemmie, Vincent A. Magnotta

36. Structure-functional Selectivity Relationship Studies of Beta-arrestin-biased Dopamine D2 Receptor Agonists

Jian Jin, Xin Chen, Maria F. Sassano, Vincent Setola, Meng Chen, William C. Wetsel, Bryan L. Roth

Poster Session I—Monday

37. Safety and Efficacy of Olanzapine/Fluoxetine Combination Versus Placebo in Patients Aged 10 to 17 in the Acute Treatment of Major Depressive Episodes Associated with Bipolar I Disorder

Melissa DelBello, Holland C. Detke, John Landry, Roland Usher, Rebecca Schroer, Mufassil Dingankar

38. Basolateral Amygdala Stimulation Produces Heterosynaptic Suppression of Inputs from Other Temporal Cortical Structures in the Prefrontal Cortex

Hugo Tejada, Patricio O'Donnell

39. A 0.23 Mb Region on Mouse Chromosome 11 Contains Three Possible Quantitative Trait Genes Influencing Methamphetamine Sensitivity

Camron D. Bryant, Clarissa C. Parker, Michael A. Guido, Loren A. Kole, Jackie E. Lim, Greta Sokoloff, Riyan Cheng, Abraham A. Palmer

40. Kinase Inhibition within the Bed Nucleus of the Stria Terminalis Potentiates Binge Alcohol Intake by C57BL/6J Mice

Melissa G. Wroten, Justin A. Courson, Amy R. Williams, Karen K. Szumlinski

41. Switching To Lurasidone in Patients with Schizophrenia: Tolerability And Effectiveness at 6 Weeks And 6 Months

Joseph P. McEvoy, Leslie Citrome, David Hernandez, Jay Hsu, Peter Warner, Andrei Pikalov, Josephine Cucchiaro, Christoph U. Correll, Antony Loebel

42. Neural Responses during Explicit and Implicit Face Processing Vary Developmentally in Bipolar Disorder

Christen M. Deveney, Melissa Brotman, Laura Thomas, Kendra Hinton, Eli Muhrer, Richard Reynolds, Nancy Adleman, Daniel S. Pine, Ellen Leibenluft

Poster Session I—Monday

43. Roles of Schizophrenia Susceptibility Gene KCNH2 3.1 Isoform in Regulation of Neuronal Excitability, Long-term Potentiation, Synaptogenesis and Cognition

Jingshan Chen, Peixiong Yuan, Qingjun Tian, Feng Yang, Grace Zhang, Jiemin Jia, Yun Wang, Jing Du, Paul Glineburg, Gregory V. Carr, Francesco Papaleo, James Pickel, Zheng Li, Daniel Weinberger

44. Neuronal Systems Underlying the Antidepressant Response to Ketamine

Daniel J. Lodge, Flavia Carreno, Amiksha Shah, Julianne Jett, Pedro L. Delgado, David A. Morilak, Alan Frazer

45. A Novel Serotonin-2 (5-HT₂) Modulator as a Candidate Drug to Treat Impulsive Behavioral Disorders and Psychoses without Weight Gain as a Side Effect

Drake Morgan, Clinton E. Canal, Krishnakanth Kondabolu, Rajeev Sakhuja, Kimberly Robertson, Neil E. Rowland, Raymond G. Booth

46. Relationship of Plasma Oxytocin Levels to Baseline Symptoms and Symptom Changes during Oxytocin Administration in Men and Women with Schizophrenia

Deanna L. Kelly, Heidi J. Wehring, Robert P. McMahon, Fang Liu, Jared Linthicum, Joseph G. Verbalis, Robert W. Buchanan, Gregory Strauss, Leah H. Rubin, Mary R. Lee

47. Drug-unpaired Environments Regulate Dendritic Spine Dynamics in the Nucleus Accumbens

Paul Vezina, Bryan Singer, Nancy Bubula, Vytautas Bindokas

Poster Session I—Monday

48. Understanding the Role of G-protein Dependent Signaling in the Indirect Striatal Pathway in Behavioral Inhibition using Targeted Viral-mediated Gene Transfer of DREADD Receptors
Susan Ferguson, Danielle Newcomer, Trevor W. Robbins, John F. Neumaier
49. Antipsychotic-like Actions of Amylin in Ventral Striatal Regions Enriched in RAMP-1 Gene Expression
Vaishali P. Bakshi, Sarah K. Baisley, Quentin Bremer, Brian A. Baldo
50. Role of Glycogen Synthase Kinase-3Beta and Beta-Catenin in the Ventral Tegmental Area in Stress and Anxiety Behaviors
Michelle S. Mazei-Robison, Raghu Appasani, Caroline Dias, Rachael Neve, Eric J. Nestler
51. Deficient Prepulse Inhibition in Schizophrenia Detected by the Multi-site Consortium on the Genetics of Schizophrenia (COGS)
Neal R. Swerdlow, Gregory A. Light, Joyce Sprock, David L. Braff, COGS Investigators
52. Pain-related Depression of the Mesolimbic Dopamine System in Rats: Expression, Blockade by Analgesics, and Role of Endogenous Kappa Opioids
Steve Negus, Michael Leitl, Matthew L. Banks
53. Hippocampal Function in KCNH2-3.1 Transgenic Mice
Gregory V. Carr, Audrey Bebensee, Randy Xun, Omoye Akhile, Qingjun Tian, Jingshan Chen, Francesco Papaleo, Daniel R. Weinberger

Poster Session I—Monday

54. Cocaine Craving and AMPA Receptor Plasticity: Modulation by Metabotropic Glutamate Receptors

Jessica A. Loweth, Andrew F. Scheyer, Mike Milovanovic, Xuan Li, Eden Flores-Barrera, M. Foster Olive, Kuei Y. Tseng, Marina E. Wolf

55. Modulation of Decision Biases by the Lateral Habenula

Colin M. Stopper, Stan B. Floresco

56. Pavlovian Conditioned Approach to a Reward Cue Predicts Fear Incubation

Jonathan D. Morrow, Stephen Maren, Terry E. Robinson

57. Hyperactivity and Increased Sociability in Mice lacking Fibroblast Growth Factor Receptor 2 in GFAP+ Cells During Critical Early Postnatal Period

Hanna E. Stevens, Flora M. Vaccarino

58. Adolescent Cannabinoid Exposure and Schizophrenia-like Deficits

Subroto Ghose, Kelly Gleason, Abhay Shukla, Shari Birnbaum

59. Social Interaction Familiarization, a Valid Preclinical Model of Social Processing and Behavioral Therapy for Anxiety

William Truitt, Elizabeth Lungwitz, Amy Dietrich, Pamela Minick, Anantha Shekhar

60. Alarm Pheromone Processing Areas are Involved in the Intergenerational Social Transfer of Emotional Trauma

Jacek Debiec, Regina M. Sullivan

Poster Session I—Monday

61. Adolescent Stressors to Epigenetic Modulation in Dopaminergic Neurons via Glucocorticoids: A Novel Model for Psychotic Depression

Minae Niwa, Akira Sawa

62. Enhanced Nicotine Self-administration in the Neonatal Ventral Hippocampal Lesion Rat Model of Schizophrenia without Nicotine Reversal of Spatial Working Memory Deficits

Sara Berg, Alena Sentir, Ben Cooley, R. Andrew Chambers

63. Convergence of Medial and Orbital Prefrontal Cortical Fibers in the Ventral Striatum Mediate DBS-enhancement of Fear Extinction

Jose Rodriguez-Romaguera, Fabricio H. Do Monte, Yoko Tanimura, Gregory J. Quirk, Suzanne Haber

64. Early Exposure to Antidepressants Does Not Recapitulate Constitutive Serotonin Transporter Deficiency

Stefanie Altieri, Hongyan Yang, Hannah O'Brien, Julie G. Hensler, Anne M. Andrews

65. Serotonin Transporter Genotype Modulates HPA Axis Output during Stress: Effect of Stress, Dexamethasone Test and ACTH Challenge

J. Dee Higley, Andrea Sorenson, John Capitanio, Sally Mendosa

66. Aberrant Light Impairs Mood and Learning through Melanopsin-expressing Neurons

Tara A. LeGates, Cara Altimus, Hui Wang, Sunggu Yang, Alfredo Kirkwood, Todd Weber, Samer Hattar

67. Traumatic Stress Reactivity Facilitates Excessive Alcohol Drinking and Prefrontal Cortex-Amygdala Synchronicity

Nicholas W. Gilpin, Scott Edwards, George F. Koob

Poster Session I—Monday

68. Context-dependent Neuronal Ensembles in the Amygdala, Prelimbic Area and Ventral Hippocampus after Fear Extinction in Rats

Caitlin Orsini, Chen Yan, Sheena Josselyn, Stephen Maren

69. GABAA and GABAB Receptor Subunits Display Altered Expression in Cerebella of Subjects with Schizophrenia, Bipolar Disorder, and Major Depression

S. Hossein Fatemi, Timothy Folsom

70. Abnormalities of the Ubiquitin-Proteasome System in the Superior Temporal Gyrus in Schizophrenia

Maria D. Rubio

71. ChIP-Seq Analysis Identifies Genome-wide Binding of Histone 4 Acetylated at Lysine 5 (H4K5ac) as a Mediator of the Acute Transcriptional Effects of Methamphetamine

Jean Lud Cadet, Christie Brannock, Michael McCoy, Subramaniam Jayanthi, Kevin Becker, Supriyo De, Elin Lehrman

72. Heat Shock Protein Hsp90 α : A Novel Target for Aripiprazole-induced Neurite Outgrowth

Kenji Hashimoto, Tamaki Ishima

73. Cognitive Impairments Induced by Brief Sleep Deprivation can be Prevented by Targeting a Single Phosphodiesterase Isoform Selectively in Excitatory Neurons in the Hippocampus

Robbert Havekes, Jennifer Choi, Vibeke Bruinenberg, George Baillie, Kyle Krainock, Sara Aton, Peter Meerlo, Miles Houslay, Ted Abel

Poster Session I—Monday

74. Childhood Adversity is Associated with Fewer Immature and Mature Dentate Gyrus Neurons in Treated and Untreated Depressed Subjects but not in Non-psychiatric Controls
- Giulia Bracci, Mihran J. Bakalian, Tanya H. Butt, Adrienne N. Santiago, Andrew J. Dwork, Gorazd B. Rosoklija, Rene Hen, Victoria Arango, Hadassah Tamir, J. John Mann, Maura Boldrini
75. Higher Nestin and GFAP Immunoreactivity in Superior Temporal Cortex in Developing Autism Donors 2-21 Yrs of Age Compared To Age-matched Controls, Evidence for Sustained Cell and Microvessel Proliferation
- Efrain C. Azmitia, Pooja P. Kothari, Mohammed F. Alzoobaee, Tanya H. Butt, Helen K. Lyo, Gordon Jiang, Patricia M. Whitaker-Azmitia, Probal Banerjee, Maura Boldrini
76. Stress, PACAP and Epigenetic Control of Adrenergic Function
- Dona Lee Wong, Robert Claycomb
77. Activity-dependent Phosphorylation of MeCP2 T308 Regulates Interaction with NCoR Co-repressor Complex
- Daniel H. Ebert, Michael E. Greenberg
78. Induced Pluripotent Stem Cell (iPSC) Models for Bipolar Disorder
- Melvin McInnis, Haiming Chen, Cindy DeLong, Sue O'Shea
79. Knockdown of TrkB in the Rat Nucleus Accumbens Alters BDNF Signaling in the Mesocorticolimbic Circuit and Prevents Effects of Social Defeat Stress on Amphetamine Cross-sensitization
- Ella Nikulina, Junshi Wang, Jeremy Kleiman, Ernest Terwilliger, Caroline Bass, Ronald Hammer

Poster Session I—Monday

80. N-methyl-D-aspartate Receptor Function and Cocaine-induced Conditioned Place Preference: Implications for Comorbid Schizophrenia and Substance Abuse

Matthew D. Puhl, Anita J. Bechtholt, Joseph Coyle

81. “Erasing” a Cocaine-cue Memory in Mice: Potential Implications for Relapse to Drug Taking

Sheena Josselyn, Hwa-Lin (Liz) Hsiang, Michel van den Oever, Chen Yan, Asim Rashid, Paul Frankland

82. Tricyclic Antidepressant Amitriptyline Indirectly Increases the Proliferation of Adult Dentate Gyrus-derived Neural Precursor Cells through Inducing FGF2 Secretion from Astrocytes

Shuken Boku, Kazue Hisaoka-Nakashima, Shin Nakagawa, Akiko Kato, Naoto Kajitani, Takeshi Inoue, Minoru Takebayashi

83. Redox Dysregulation in Fast-spiking Interneurons Disrupts Cortical Stability

Hirofumi Morishita, Harry J. Cabungcal, Ying Chen, Kim Q. Do, Takao K. Hensch

84. A New Role of Dopamine D2 Receptors in the Regulation of Synaptic Connections

Zheng Li

85. Altered Frontal Cortex Insulin Receptor Mediated Signaling and Associated Epigenetic Modifications in Alzheimer’s Patients but not during Aging

Jagadeesh S. Rao, Alisha Jamil, Stanley I. Rapoport

Poster Session I—Monday

86. Striatal Adenosine Signaling Regulates EAAT2 and Astrocytic AQP4 Expression and Alcohol Drinking in Mice

Moonnoh R. Lee, Christina L. Ruby, David J. Hinton, Sun Choi, Chelsea A. Adams, Na Young Kang, Doo-Sup Choi

87. Adolescent Social Isolation Impairs Decision-making and Elevates Deep-layer Prefrontal Cortical Dendritic Spine Density in Adulthood

Elizabeth A. Hinton, Shannon L. Gourley

88. Cell Adhesion Pathway is Implicated in Lithium Treatment for Adolescent Mania via DNA Methylation Alteration

Chunyu Liu, Jeffrey R. Bishop, Chunling Zhang, Meredith Wong, Shitalben Patel, Jonathan Leigh, Mani Pavuluri

89. Lipid Raft Sequestration of the G Protein, Gs α : A Protein-based Platelet Biomarker for Major Depressive Disorder

Jeffrey Sprouse, Alexander Jackson, Robert Donati, Lucio Tonello, Massimo Cocchi, Mark M. Rasenick

90. A Trial of Prazosin for Combat Trauma PTSD with Nightmares in Active Duty Soldiers Returned from Iraq and Afghanistan

Murray A. Raskind, Kris Peterson, Tammy Williams, Elaine R. Peskind

91. Reduced Mitochondrial Energy Production in Major Depressive Disorder: Associations with the Serotonin Transporter and Glutamine Synthetase Genes

Chadi G. Abdallah, Graeme F. Mason, Henk De Feyter, Madonna Fasula, Ben Kelmendi, Arthur Simen, Lihong Jiang, John H. Krystal, Douglas L. Rothman, Gerard Sanacora

Poster Session I—Monday

92. Social and Non-social Cognition in Bipolar Disorder and Schizophrenia: Relative Levels of Impairment

Junghee Lee, Lori Altshuler, David Glahn, David Miklowitz, Kevin Ochsner, Michael F. Green

93. Reducing GABA-A alpha 5 Receptor-mediated Inhibition Restores Synaptic Plasticity and Neuromorphological Deficits in a Mouse Model of Down Syndrome

Paula Martinez, Carmen Martinez-Cue, Noemi Rueda, Rebeca Vidal, Susana Garcia, Veronica Vidal, Andrea Corrales, Juan A. Montero, Angel Pazos, Jesus Florez, Andrew W. Thomas, Frederic Knoflach, Jose Luis Trejo, Joseph G. Wettstein, Maria C. Hernandez

94. Glutamate Antagonism and Alcohol Behaviors in Heavy Drinkers: Contrasting Effects of Opioid Antagonism

Suchitra Krishnan-Sarin, Stephanie O'Malley, Nicholas Franco, Dana Cavallo, Brian Pittman, Julia Shi, John H. Krystal

95. Occupancy of Naltrexone at Kappa Opioid Receptors may Predict Efficacy in Reducing Craving and Drinking in Alcoholics: A PET Imaging Study with a Novel Kappa Tracer

Evan Morris, Su Jin Kim, Nicholas Franco, Dana Cavallo, alisha jordan, Julia Gillard, Ming-Qiang Zheng, shu-fei lin, Stephanie O'malley, Yiyun Huang, Suchitra Krishnan-Sarin

96. Development of Personalized Small Molecule Modulator Screening Strategies: Upregulation of Alpha-L-iduronidase in Mucopolysaccharidosis Type I (MPSI) Patient Cells

Claude-Henry Volmar, Shaun P. Brothers, Claes Wahlestedt

Poster Session I—Monday

97. Trajectories of Depressive Symptoms During Medical Internship: Insights Into Classes of Depressive Symptoms Under Conditions of Chronic Stress
Srijan Sen, Constance Guille, Shaunna Clark, Ananda Amstadter
98. Lurasidone Adjunctive to Lithium or Valproate for the Treatment of Bipolar I Depression: Results of the 6-week, Double-blind, Placebo-controlled Prevail-1 Study
Joseph R. Calabrese, Antony Loebel, Josephine Cucchiaro, Robert Silva, Jay Hsu, Kaushik Sarma, Gary Sachs
99. Static and Dynamic Functional Network Connectivity during Resting State in Schizophrenia
Eswar Damaraju, Jessica Turner, Adrian Preda, Theo Van Erp, Daniel H. Mathalon, Judith M. Ford, Steven Potkin, Vincent Calhoun
100. Effect of Narp Deletion on Neophobia
Ashley Blouin, JongAh Lee, Bo Tao, Alexander Johnson, Dani Smith, Jay Baraban, Irving M. Reti
101. Combined Dexamethasone Suppression – Corticotrophin-releasing Hormone Stimulation Test in Unmedicated Major Depression and Healthy Volunteers
Leo Sher, Maria Oquendo, Thomas Cooper, J. John Mann
102. Inflammatory Biomarkers in Late-life Depression and White Matter Integrity
Nunzio Pomara, Davide Bruno, Jay Nierenberg, John Sidsitis, Henrik Zetterberg, Kaj Blennow

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103. Acute Nicotine Administration Improves White Matter Integrity and Associated Attention Performance

Peter Kochunov, Elliot A. Stein, Elliot Hong

104. The Unreliability of Reliability Statistics: A Primer on Calculating Interrater Reliability in CNS Clinical Trials

Danielle Popp, Craig H. Mallinckrodt, Janet BW. Williams, Michael J. Detke

105. Effects of Chronic Mild Stress and Electroconvulsive Seizure on Memory Functions in Rats

Ove Wiborg, Kim Henningsen, David Woldbye, Elena Bouzinova

106. Next-generation Sequencing Follow-up to a Genome Wide Association Scan for EEG Power in a Native American Population

Colin A. Hodgkinson, Polina Iarikova, Qiaoping Yuan, Cheryl Marietta, Zachary Hommer, Mary-Anne Enoch, David Goldman

107. Extracellular Administration of Apical Domain of CCT1 Inhibits Mutant Huntingtin Aggregation and Promotes Cell Survival in Vitro

Steven Potkin, Zhiqun Tan, Emily Mitchell Sontag, William E. Bunney, Leslie Thompson, Charles Glabe

108. Chronic Glucocorticoid Induces Parkin 2 – Mediated Ubiquitin-Proteasome Activity and TrkB Degradation

Anilkumar Pillai, Chirayu Pandya, Callie Jowers

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109. GLYX-13, a NMDA Receptor Glycine Site Functional Partial Agonist, Exerts its Antidepressant Effects by Acting at a Novel NMDA Receptor Modulatory Site

Joseph Moskal, Roger Kroes, Jeffrey Burgdorf, Amanda Gross, Xiao-lei Zhang, Ronald M. Burch, Patric Stanton

110. Next Generation Sequencing using ChIP-Seq Highlights an Essential Role for SIRT1 in Emotional Plasticity

Deveroux Ferguson, Ningyi Shao, Ja Wook Koo, Jian Feng, Vincent Vialou, Rachael Neve, Li Shen, Eric J. Nestler

111. Glucocorticoid Receptor Translocation is Attenuated in Olfactory Neuroepithelial Cells of Patients with Major Depressive Disorder

Karin Borgmann-Winter, Sarah Jefferson, Brooke Willis, Aziza Manceur, Ray Rabindranath, James Stefano, Jackie St. Louis, Michael Thase, Olivier Berton, Chang-Gyu Hahn

112. PSD Protein Partitioning is Drastically Altered in the Lateral Prefrontal Cortex of Schizophrenia

Matthew L. MacDonald, Gene Ciccimaro, Steven Siegel, Scott Hemby, Ian Blair, Chang-Gyu Hahn

113. Developmental Switch in Striatal Gene Expression in Rat: Implications for Schizophrenia

Gabriela Novak, Theresa Fan, Brian F. O'Dowd, Susan R. George

114. A Potential Mechanism of Behavioral Alteration by Genome Diversification: The Role of Neural MILI/piRNA Complexes on De Novo L1 Retrotransposition

Daisy Lin, Jay A. Gingrich

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115. Top-down Control of Raphe Circuits in Affective Resilience: Key Role of Raphe GABA Interneurons

Collin Challis, Julie Espallergues, Sheryl Beck, Olivier Berton

116. Ras Suppressor 1 Acts Downstream of Integrin to Regulate Rac1 Activity and Ethanol Consumption in Drosophila and Humans

Shamsideen Ojelade, Gunter Schumann, Adrian Rothenfluh

117. Cellular Mechanisms of Growth Arrest and DNA Damage-inducible 45b (Gadd45b) in Regulating Antidepressant-induced Adult Hippocampal Neurogenesis

Mi-Hyeon Jang, Heechul Jun, Guo-li Ming, Hongjun Song

118. Neuronal-glia Interactions in the Nucleus Accumbens following Morphine Administration: A Role in Relapse Behavior

Jaclyn M. Schwarz, Staci D. Bilbo

119. Limited Contribution of NMDA Receptor GluN1 Deletion in Cortical Excitatory Neurons to Schizophrenia-like Phenotypes

Gregory Rompala, Veronika Zsiros, Shuqin Zhang, Stefan M. Kolata, Kazu Nakazawa

120. Comparing Genome-wide Association Results for Conditioned Fear in Two Advanced Intercross Mouse Lines: Implications for the Genetic Mapping of Complex Psychiatric Traits

Clarissa C. Parker, Greta Sokoloff, Riyan Cheng, Shyam Gopalakrishnan, Natalia M. Gonzales, Joe Davis, Abraham A. Palmer

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121. Genomic Sequencing and Linkage Analysis in Selectively Bred Rat Lines Identify *Grm2* and *Lcn2* Stop Codons as Functional Alleles Influencing Alcohol Preference

Zhifeng Zhou, Tiebing Liang, Mitsuru Kimura, Qiaoping Yuan, Mary-Anne Enoch, Colin A. Hodgkinson, Francesca Ducci, Marjo-Riitta Järvelin, Anneli Pouta, Jenica Tapocik, Estelle Barbier, Markus Heilig, Howard Edenberg, David Goldman

122. Comparative Analysis of Differential Allele Gene Expression in the Mouse Brain

Seungeun Yeo, Zhifeng Zhou, Colin A. Hodgkinson, Qiaoping Yuan, Jeesun Jung, Mary-Anne Enoch, David Goldman

123. Open Field Testing of *Drosophila* Adults to Study CNS Stimulants

Charles D. Nichols, Indya Bruce, Jaime Becnel

124. Cortical Thickness, Regional Brain Volumes, and Symptom Severity in Body Dysmorphic Disorder

Sarah Madsen, Tara Pirnia, Alex Zai, Teena Moody, Jamie Feusner

125. Compared to What? Reappraising the Early Brain Overgrowth Hypothesis in Autism

Armin Raznahan, Rhoshel Lenroot, Audrey Thurm, Marta Gozzi, Sarah Spence, Susan Swedo, Jay Giedd

126. Nicotinic-mediated Effects on Brain Reward Function are Modulated by $\alpha 5$ -containing Nicotinic Acetylcholine Receptors

Christie D. Fowler, Paul Kenny

127. Targeted Deletion of the $\alpha 2$ Nicotinic Acetylcholine Receptor Subunit Gene (*Chrna2*) Potentiates Sexual Dimorphism in Emotional Processing

Shahrdad Lotfipour

Poster Session I—Monday

128. Serotonin Transporter Deficient Rats Exhibit Enhanced Acquisition and Disrupted Extinction of Conditioned Fear

Philip L. Johnson, Stephanie D. Fitz, Andrei Molosh, William Truitt,
Anantha Shekhar

129. Exposure to Chronic Stress During Pregnancy Prevents the Beneficial Effects of Motherhood on Dendritic Spines in the Hippocampus and Medial Prefrontal Cortex

Benedetta Leuner, Peter Fredericks, Connor Nealer

130. Cortisolemia, Psychopathology and Treatment Response in First-episode Schizophrenia

Eva Ceskova, Radovan Prikryl

131. Trauma Timing Predicts Variation in PTSD-related Outcomes

David T. George, Laura E. Kwako, Keva Garg, Joanna Sells, Erica N. Grodin, Melanie Schwandt, Daniel W. Hommer, Markus Heilig

132. The Relationship between Sleep Quality AND Morning/Eveningness, Seasonality, Activity Levels, and Dim Light Melatonin Onset in Depressed Patients with Bipolar Disorder

Dorothy Sit, Christopher Wiltrout, Evie Fowler, Maxwell Mitchell, Katherine L. Wisner, Howard Seltman

133. Brief Cognitive Intervention can Modulate ACTH Response to the Trier Social Stress Test

Stefanie Mayer, Thane Erickson, Hedieh Briggs, Jennifer Crocker, Israel Liberzon, James L. Abelson

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134. The Novel Vasopressin V1a Receptor Antagonist SRX246 Blocks Vasopressinergic Modulation of Emotion: An fMRI Study

Royce J. Lee, Emil F. Coccaro, Rosemary McCarron, Vernon Towle, Shi Fang Lu, Christophe Guillon, Karine Fabio, Michael Brownstein, Neal Simon

135. Antidepressant Efficacy of Ketamine in Treatment-resistant Major Depression: A Two-site, Randomized, Parallel-arm, Midazolam-controlled, Clinical Trial

James W. Murrough, Dan V. Iosifescu, Lee C. Chang, Rayan K. Al Jurdi, Charles M. Green, Syed Iqbal, Sarah Pillemer, Andrew M. Perez, Alexandra Foulkes, Asim Shah, Dennis S. Charney, Sanjay J. Mathew

136. Can Prediction of Psychosis be Improved? Findings from the 12-year Recognition and Prevention (RAP) Program

Barbara A. Cornblatt, Ricardo Carrion, Andrea Auther, Danielle McLaughlin

137. A Randomized Trial of a Low Trapping Non-selective N-methyl-D-aspartate Channel Blocker (AZD6765) in Treatment-resistant Major Depression

Carlos A. Zarate, Daniel Mathews, Nancy Brutsche, Libby Jolkovsky, Mark A. Smith, David Luckenbaugh

138. Impact of Sex and Gonadal Steroids on Neonatal Brain Structure

Rebecca Knickmeyer, Jiaping Wang, Hongtu Zhu, Xiujuan Geng, Sandra Woolson, Robert M. Hamer, Thomas Konneker, Martin Styner, John Gilmore

Poster Session I—Monday

139. Stress Response Systems in Adolescent Girls and Boys with Major Depression: A Multi-Modal Approach

Kathryn R. Cullen, Bonnie Klimes-Dougan, Alaa Hourri, Kelvin O. Lim

140. Stress Response in Adolescents with Childhood Maltreatment: Moderation by Gender and Genetic Factor

Uma Rao, Elena Gorodetsky, David Goldman

141. Salivary Cortisol, Pro-inflammatory Cytokines, and Grief-related Psychiatric Symptoms in Parentally Bereaved Children

Julie B. Kaplow, Kristen Wiese, James L. Abelson, Alan Prossin

142. Circadian Cortisol and Hypothalamic-subgenual Cortex Functional Connectivity in Major Depression with and without Psychotic Features

Keith Sudheimer, Jennifer Keller, Lakshika Tennakoon, Alan F. Schatzberg

143. Blood Glucagon-like Peptide-1 (GLP-1) Concentration Correlates Inversely with Alcohol Self-Administration in a Laboratory Study with Alcoholic Individuals: Preliminary Findings

Lorenzo Leggio, William Zywiak, Steven Edwards, Samuel Fricchione, Cynthia Vuittonet, Robert Swift, George Kenna

144. Neuroadaptive Changes after Chronic Lurasidone Treatment: Implication for Mood and Stress-related Disorders

Marco A. Riva, Alessia Luoni, Francesca Calabrese, Gianluigi Guidotti, Giorgio Racagni, Judith Homberg

Poster Session I—Monday

145. Substrate-selective Inhibition of COX-2 as a Novel Strategy for In Vivo Endocannabinoid Augmentation

Daniel Hermanson, Nolan Hartley, Lawrence Marnett, Sachin Patel

146. Modulation of the Inflammatory Response after the Antidepressant Agomelatine in Rats

Raffaella Molteni, Flavia Macchi, Claudia Zecchillo, Mario Dell'Agli, Marco A. Riva, Giorgio Racagni

147. Synergy between Melatonergic and 5-HT_{2C} Receptors in the Action of Agomelatine – Molecular and Cellular Evidence

Daniela Tardito, Marco A. Riva, Raffaella Molteni, Alessandra Mallei, Laura Musazzi, Francesca Calabrese, Maurizio Popoli, Giorgio Racagni

148. Spatial Memory Deficits in Adult c57BL6 Mice Exposed to Fluoxetine during Adolescence

Michelle J. Stone, Steven J. Nieto, Tiffany Aiello, Lace Riggs, Sergio Iñiguez

149. Subchronic Escitalopram Treatment Leads to SERT Internalization Rather than Down-regulation In Vitro and Ex Vivo

Walter E. Mueller, Jeanine Heiser, Kristina Leuner

150. An Examination of Involvement of the Dopamine Transporter (DAT), the Serotonin Transporter (SERT), and Monoamine Oxidase A (MAOA) in the Temperature and Locomotor Effects of 4-methylthiomethamphetamine (MTA)

Meredith A. Fox, Pablo R. Moya, Ramón Sotomayor-Zárate, Patricio Iturriaga-Vásquez, F. Scott Hall, Kevin Chen, Jean C. Shih, George R. Uhl, Miguel Reyes-Parada, Dennis L. Murphy

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151. Decreasing Activity of Neuronal Nicotinic Receptors can Improve Cognitive Function

Edward D. Levin, Marty C. Cauley, Amir H. Rezvani

152. CTP-354: A Novel Deuterated Subtype-selective GABA(A) Modulator for Treatment of Neuropathic Pain, Spasticity and Anxiety Disorders

Julie F. Liu, Scott Harbeson, Vinita Uttamsingh, Arturo J. Morales, Sophia Nguyen, Gary Bridson, Changfu Cheng, Ara Aslanian, Lijun Wu

153. Activation of Metabotropic Glutamate Receptor 7 (mGluR7) by AMN082 Attenuates the Rewarding Effects of Cocaine and Nicotine in Rats

Xia Li, Astrid Stoker, Zheng-Xiong Xi, Eliot Gardner, Athina Markou

154. Lithium Potentiates Long-term Synaptic and Antidepressant-like Effects of Ketamine

Rong-Jian Liu, Manabu Fuchikami, Jason Dwyer, Ronald S. Duman, George Aghajanian

155. Identification of a Novel Dopaminergic Agonist that Selectively Activates the D2 Dopamine Receptor

David R. Sibley, R. Benjamin Free, Jennie Conroy, Rebecca A. Roof, Trevor Doyle, Noel Southall, Marc Ferrer, Prashant Donthamsetti, Mayako Michino, Yang Han, Lei Shi, Jonathan A. Javitch

156. A Novel Nociceptin-1 Receptor Antagonist Produces Antidepressant-, Anxiolytic-, and Anti-ethanol-associated Effects in Rodent Models

Jeffrey M. Witkin, M Statnick, D L. McKinzie, L Rorick-Kehn, V N. Barth, J Pintar, K Perry, M A. Toledo, N Diaz, C Lafuente, A Jimenez, M A. Martinez-Grau

Poster Session I—Monday

157. Preclinical Studies of the Multimodal Antidepressant Vortioxetine Support a Potential Improvement of Cognitive Functions

Steven C. Leiser, Alan L. Pehrson, Paul J. Robichaud, Kristian GJ. Nielsen, Jesper B. Jensen, Gennady Smagin, Dekun Song, David Budac, Alan Frazer, Connie Sanchez

158. The Inositol Depletion Hypothesis of Lithium's Mechanism of Action is not Dead; Lithium Affects Mouse Brain Inositol Turnover with Behavioral Consequences in Bipolar-related Paradigms

Galila Agam, Yeala Sade

159. Altered Tonic and Phasic Glutamate in Prelimbic and Infralimbic Prefrontal Cortices of Anesthetized and Awake SHR Rats

Paul Glaser, Erin Miller, Francois Pomerleau, Greg Gerhardt

160. Evidence for Involvement of Nitric Oxide and GABA-B Receptors in MK-801-stimulated Glutamate Efflux in the Rat Prefrontal Cortex

Gary A. Gudelsky, Nicole Roenker, Rebecca Ahlbrand, Paul Horn, Neil M. Richtand

161. Effects of Chronic Cariprazine Administration on Serotonin and Glutamate Receptor Subtypes

Frank Tarazi, Yong Kee Choi, Nika Adham, Béla Kiss, István Gyertyan

162. Effects of a Novel Simplified Acute Tryptophan Depletion (SATD) vs. Acute Tryptophan Depletion (ATD Moja-De) Formulas on Serotonergic and Dopaminergic Content in C57BL/6J Mice

Cristina L. Sanchez, Amanda ED. Van Swearingen, Andrew E. Arrant, Cynthia Kuhn, Florian Daniel. Zepf

Poster Session I—Monday

163. Patterns in the Shape of Maturational Trajectories across the Human Cortex and between the Sexes

Aaron F. Alexander-Bloch, Philip T. Reiss, Nitin Gogtay, Jay Giedd

164. Drug Cue-induced Dopamine Release in Amygdala and Hippocampus: A High-resolution PET [18F]Fallypride Study in Cocaine Dependent Participants

Aryandokht Fotros, Kevin Casey, Kevin Larcher, Jeroen Verhaeghe, Sylvia Cox, Paul Gravel, Andrew Reader, Alain Dagher, Chawki Benkelfat, Marco Leyton

165. Lurasidone for Bipolar I Depression: Effects on Quality of Life and Functioning

Terence A. Ketter, Josephine Cucchiaro, Robert Silva, Peter Warner, Andrei Pikalov, Kaushik Sarma, Hans Kroger, Antony Loebel

166. Decreased Neuropeptide Y and Altered Neuropeptide Y - Corticotropin Releasing Hormone Balance in Cerebrospinal Fluid in Remitted Bipolar Patients can Predict Future Suicide Attempts

Aleksander Mathé, Johan Sandberg, Mikael Landén

167. Abnormal Threat Detection in AHI1 Mutant Mice: Translational Relevance to Schizophrenia and Autism

Amit Lotan, Tzuri Lifschytz, Alexandra Slonimsky, Yakov Fellig, Suzan Abedat, Hagit Cohen, Gadi Goelman, Bernard Lerer

168. Activation of Noradrenergic Locus Coeruleus Neurons Promotes Anxiety-like Behaviors

Jordan G. McCall, Edward R. Siuda, Chris P. Ford, Michael R. Bruchas

Poster Session I—Monday

169. Solanezumab Phase 3 Results: Testing the Amyloid Hypothesis

Richard C. Mohs, Eric Siemers, Christopher Carlson, Wahiba Estergard, Karen Sundell, David Henley, Jennifer Eads, Cora Sexton, Robert Dean, Brian Willis, Ronald DeMattos

170. Chronic Ethanol Upregulates Toll-like Receptors Increasing Neuroinflammation and Neurodegeneration Mimicking Human Alcoholic Brain

Fulton T. Crews, Liya Qin, Ryan P. Vetreno

171. MRS GABA and Glutamate Abnormalities in the Superior Temporal Gyrus and Their Association with Gamma Band Oscillation Abnormalities in Schizotypal Personality Disorder and Schizophrenia

Alexander Lin, Huijun Liao, Sai Merugumala, Margaret Niznikiewicz, Kevin M. Spencer, Yoji Hirano, Robert McCarley

172. Mutations in the X-linked Endosomal Alkali Cation/proton Exchanger 6 - A New Genetic Model to Study the Neurodevelopmental Biology of Severe Autism

Eric M. Morrow, Qing Ouyang, Julie Kauer, Michael Schmidt, Sofia Lizarraga

173. Resting Functional Connectivity of the Nucleus Accumbens in Apathetic Late-life Depression

George S. Alexopoulos, Genevieve S. Yuen, Matthew Hoptman, Faith Gunning-Dixon

174. The Ankyrin 3 (ANK3) Bipolar Disorder Gene Regulates Mood-related Behaviors and Stress Reactivity in Mice

Melanie Leussis, Erin Berry-Scott, Mai Saito, Ozan Alkan, Catherine Luce, Jon Madison, David Root, Tracey Petryshen

Poster Session I—Monday

175. Evidence for Diminished Interoceptive Response to Soft Touch in Adolescent and Adult Stimulant Users

Martin Paulus, Susan Tapert, Jennifer Stewart, April May, Robyn Migliorini

176. Sex Differences in Fear Learning and Memory: A Role for Dopamine

Rebecca Shansky, Colin Rey

177. Major Depressive Disorder with Mixed Features: Interim Baseline Characteristics of Subjects Enrolled in a 6-week, Double-blind, Placebo-controlled Trial of Lurasidone

Trisha Suppes, Josephine Cucchiaro, Andrei Pikalov, Peter Warner, Steven D. Targum, Antony Loebel

178. The Novel Brain Penetrant NPS Receptor Antagonist, NCGC00185684, Blocks Alcohol-Induced ERK-phosphorylation within the Central Amygdala, and Decreases Alcohol Self-administration in Rats

Annika Thorsell, Wei Zheng, Michelle Zook, Lauren Bell, Sam Patnaik, Juan Marugan, Ruslan Damadzic, Jenica Tapocik, Ke Liu, Seameen Dehdashti, Melanie Schwandt, Noel Southall, Christopher Austin, Robert Eskay, Roberto Ciccocioppo, Markus Heilig

179. ¹H-¹³C-Nuclear Magnetic Resonance Spectroscopy Measures of CP-101-606, Ro-256981 or Ketamine's Dose Effects on Amino Acid Neurotransmitter Metabolism

Golam M.I. Chowdhury, Kevin L. Behar, Eric Schaeffer, Linda Bristow, Douglas L. Rothman, Gerard Sanacora

180. Melanocortin 4 Receptor Signaling in the Ventral Striatum Affects Obsessive Compulsive Disorder-like Behaviors in Mice

Pin Xu, Huxing Cui, Brittany L. Mason, Andrew A. Pieper, Michael L. Lutter

Poster Session I—Monday

181. Monoamine Modulation of Separation-induced Ultrasonic Vocalizations in Preweanling F344 rats

Cynthia A. Crawford

182. Deuterium Enriched L-DOPA Displays Increased Behavioral Potency and Dopamine Output in an Animal Model of Parkinson's Disease: Comparison with the Effects Produced by L-DOPA and an MAO-B Inhibitor

Torgny H. Svensson, Torun Malmjöf, Kristin Feltman, Åsa Konradsson-Geuken, Björn Schilström

183. Impact of Sustained Administration of Aripiprazole on Neuronal Activity in Monoaminergic Systems in the Rat Brain

Chris Oosterhof, Mostafa El Mansari, Pierre Blier

184. Adenosine Receptor Stimulation during Extinction Training Produces Lasting Effects on Cocaine Seeking

Casey O'Neill, Benjamin Hobson, Sophia Levis, Ryan Bachtell

185. Pharmacologic and Genetic Manipulation of Trace Amine-associated Receptor 1 Signaling Demonstrates its Role in Methamphetamine-stimulated Locomotor Activity in Mice

David K. Grandy, Katie R. Tallman, Madeline S. Grandy, Ashley D. Kimbel, Olena Anoshchenko, William C. Grandy, Troy A. Wahl, Andrew Placzek, Thomas S. Scanlan, Ofelia Littrell, Wayne Cass, Greg Gerhardt, Aaron Janowsky, Greg Mark

186. Behavioral Interactions Between mGlu5 and 5-HT_{2A} Receptors in Mice

Adam L. Halberstadt, Susan B. Powell, Mark A. Geyer

Poster Session I—Monday

187. Chronic Exposure to Cocaine Produces Persistent Residual Effects on Functional Brain Activity in the Prefrontal Cortex: Evidence from a Nonhuman Primate Model of Cocaine Self-administration

Linda Porrino, Thomas Beveridge, Hilary R. Smith, Michael A. Nader

188. Uncoupling alpha7 nACh-NMDA Receptor Complex Blocks Cue-induced Reinstatement

Shupeng Li, Zhaoxia Li, Anh Le, Fang Liu

189. Activation of the μ - δ Opioid Receptor Heteromer in the Nucleus Accumbens Produces Anti-Depressant-like and Anxiolytic-like Effects

Noufissa Kabli, Tuan Nguyen, Gianfranco Balboni, Brian F. O'Dowd, Susan R. George

190. Glyoxalase 1 (Glo1) Increases Anxiety in Mice by Metabolizing Methylglyoxal, which is a Novel GABAA Receptor Agonist

Margaret G. Distler, Abraham A. Palmer

191. A Procedure for Studying Acute, Discontinuation-induced Benzodiazepine Withdrawal in Non-human Primates

Lisa R. Gerak, Martin Javors, Charles France

192. Chronic Ethanol and Nicotine Co-administration, but not Ethanol or Nicotine Self-administration, Increases the Reinforcing Properties of Nicotine within the Nucleus Accumbens Shell

Sheketha Hauser, Gerald A. Deehan, Jamie E. Toalston, Jesssica A. Wilden, William A. Truitt, William J. McBride, Zachary A. Rodd

Poster Session I—Monday

193. mTOR Altered by Chronic Alcohol Consumption: Effects on Signaling and Complex Formation in Mice

Michael J. Lewis, Darin Salloum, David Foster, Robin Clugson, William Blaner

194. Chronic Unpredictable Stress Modifies Cortical GABA and Induces Depression like Behavior in Rats: Reversal by Ketamine

Matthew P. Galloway, Farhad Ghoddoussi, George McKelvey, Shane A. Perrine

195. Dendritic Spine Plasticity Induced by the mGluR5 Positive Allosteric Modulator CDPPB

Amber L. LaCrosse, Sara B. Taylor, M. Foster Olive

196. Expression Profiles of Mitochondrial Genes in Frontal Cortex and Caudate Nucleus of Developing Humans and Mice Selectively Bred For High and Low Fear

Kwang Choi, Thein Le, Jennifer McGuire, Jennifer Coyner, Brandon Higgs, Suad Diglisic, Luke Johnson, David Benedek, Robert Ursano

197. Development of Antipsychotic Medications with Novel Mechanisms of Action Based on Computational Modeling of Hippocampal Neuropathology

Peter J. Siekmeier, David VanMaanen

198. Posttraining Optogenetic Stimulation and Inhibition of Basolateral Amygdala Activity, Respectively, Enhances and Impairs Retention of Inhibitory Avoidance Learning

Mary Huff, Rachel Miller, David Moorman, Ryan T. LaLumiere

Poster Session I—Monday

199. NMDA-receptor Antagonist Ketamine Induces Brain Hyperconnectivity at Rest

Naomi R. Driesen, Gregory McCarthy, Zubin Bhagwagar, Michael Bloch, Vincent Calhoun, Deepak D'Souza, Ralitza Gueorguieva, George He, Ramani Ramachandran, Alan Anticevic, Peter Morgan, John H. Krystal

200. Amygdala Projections to Lateral Bed Nucleus Stria Terminalis in Primate

Julie L. Fudge, Danielle M. deCampo

201. Role of the Nucleus Accumbens Shell to Lateral Hypothalamic (AcbSh-LH) Pathway in “Depressive-like” Behavior in Rats

Erin B. Larson, David W. Self

202. The Localization of the Excitatory Amino Acid Transporter EAAT2 in Prefrontal Cortex in Schizophrenia: A Postmortem Ultrastructural Study

Rosalinda C. Roberts, Joy Roche, Robert McCullumsmith

203. Within Subject Evaluation of the Effect of Neuroleptics on GABA Levels in Patients with Schizophrenia Spectrum Psychosis

Stefano Marengo, Katherine DeJong, Jan Willem van der Veen, Alan S. Barnett, Jose A. Apud, Karen F. Berman, Daniel R. Weinberger

204. Genetic Background Regulates the Effect of the Antidepressant Fluoxetine on Behavioral Despair and Hippocampal Neurogenesis in Mice

Brooke H. Miller, Zane Zeier, Thomas A. Lanz, Miguel Lopez-Teledono, Mathew Pletcher, Robin J. Kleiman, Claes Wahlestedt

Poster Session I—Monday

205. Functional Genetic Variants in the Vesicular Monoamine Transporter 1 (VMAT1) Modulate Emotion Processing

Falk W. Lohoff, Brian J. Mickey, Mary Heitzeg, Scott A. Langenecker, Jon-Kar Zubieta, Ryan Bogdan, Yulia Nikolova, Ahmad R. Hariri, Laura Bevilacqua, David Goldman, Glenn Doyle

206. Expression of Histone-modifying Genes in Response to Cocaine

Gregory C. Sartor, Shaun P. Brothers, Sari Izenwasser, Claes Wahlestedt

207. Glutamatergic Hyperexcitability during Alcohol Withdrawal-precipitated Aggression in Mice

Lara S. Hwa, Anna Nathanson, Keisha Dodman, Akiko Shimamoto, Joseph F. DeBold, Klaus A. Miczek

208. Opioid Modulation of Marijuana's analgesic, Subjective, Reinforcing, and Physiological Effects in Non-treatment Seeking Marijuana Smokers

Ziva D. Cooper, Sandra D. Comer, Gillinder Bedi, Divya Ramesh, Margaret Haney

209. Rostral vs. Caudal Anterior Cingulate Connectivities Differentiate Two Neurotherapeutic Targets Used for OCD and MDD

Sarah R. Heilbronner, Suzanne Haber

210. Experimental Sadness Induces Plasma IL-18 Elevation and Covarying Modulation of Limbic Endogenous Opioid Function in Major Depression

Alan Prossin, Alisa E. Koch, Steven S. Zalcman, Phillip L. Campbell, Jon-Kar Zubieta

Poster Session I—Monday

211. Long-term Potentiation of Visual Stimulus-induced EEG Phase Synchrony: Further Evidence for Impaired Visual Cortical Plasticity in Schizophrenia

Daniel H. Mathalon, Idil Cavus, Brian J. Roach, Ralitza Gueorguieva, Timothy J. Teyler, Wesley C. Clapp, John H. Krystal, Judith M. Ford

212. The Functional Contribution of Distal Amygdala Projections in Anxiety: An Optogenetic Circuit Dissection

Kay M. Tye, Ada C. Felix-Ortiz, Christopher Leppla

213. Oxytocin Associated with More Negative Evaluation of Neutral Faces after an Affective Learning “Gossip” Task for Men compared to Women

T.H. Eric Bui, Elizabeth Hoge, Eric Anderson, Laura Fischer, Lisa Feldman Barrett, Naomi M. Simon

214. Impaired Cognitive Flexibility following Single Prolonged Stress is Ameliorated by Pretreatment with D-Cycloserine

Sophie A. George, John Riley, James L. Abelson, Stan B. Floresco, Israel Liberzon

Tuesday Posters

Poster Session II – Tuesday



Advocacy Affiliate - International Mental Health Resource Organization
“IMHRO Rising Star Awards”

Cindy Dyar, Brandon Staglin

1. Membership Advisory Task Force: Feedback, Challenges, and Solutions in 2012

Katherine E. Burdick, Linda Carpenter, Marlene Freeman, Vaishali Bakshi, Paul Holtzheimer, Lisa Monteggia, Thomas Schulze, Carlos Bolanos-Guzman, Kristin Cadenhead, Raymond Cho, Cynthia Crawford, Paul Kenny, Gregory Light, Gonzalo Laje

2. Schizophrenic and Bipolar Tobacco Smoker/Nonsmoker Sex, Dopamine D2 Receptor Taq1A and OPRM1 A118G Genotype Differences

Edward F. Domino, Mika Hirasawa-Fujita, Michael Bly, Vicki Ellingrod, Gregory Dalack

3. Trauma and Post Traumatic Stress Disorder in an American Indian Community: Heritability, Electrophysiological Findings, and Comorbidity with Other Psychiatric Disorders

Cindy L. Ehlers, Ian Gizer, David Gilder, Rachel Yehuda

4. MicroRNA-137 Expression in Schizophrenia and Bipolar Disorder

Marquis P. Vawter, Ilaria Guella, Brandi Rollins, Theo Van Erp, Federica Torri, Pedro A. Sequeira, William E. Bunney, Steven Potkin, Fabio Macciardi

5. Gene x Withdrawal Effects on Responsiveness to Cocaine Stimuli in Cocaine Addiction

Scott J. Moeller, Muhammad Parvaz, Elena Shumay, Nicasia Beebe-Wang, Nora D. Volkow, Rita Goldstein

Poster Session II—Tuesday

6. Allele-specific DNA deMethylation in FKBP5: A Molecular Mediator of Gene x Environment Interactions with Childhood Trauma

Torsten Klengel, Divya Mehta, Christoph Anacker, Jens C. Pruessner, Carmine M. Pariante, Thaddeus WW. Pace, Kristina B. Mercer, Helen S. Mayberg, Bekh Bradley, Charles Nemeroff, Florian Holsboer, Christine M. Heim, Kerry J. Ressler, Theo Rein, Elisabeth Binder

7. APOE ϵ 4, an Alzheimer's Disease Susceptibility Allele, Predicts Relapse among Older Treatment-seeking Smokers

Rebecca L. Ashare, Jason Karlawish, E. Paul Wileyto, Angela Pinto, Caryn Lerman

8. The Catechol-O-methyltransferase (COMT) Val158Met Polymorphism Interacts with Childhood Trauma to Influence Aggression and Impulsivity among Treatment-seeking Alcohol Dependent Individuals

Melanie Schwandt, Markus Heilig, Daniel W. Hommer, David T. George, Colin A. Hodgkinson, Pei-Hong Shen, David Goldman, Vijay Ramchandani

9. eQTL Regulation by NATs in Alzheimer's Disease

Amanda Myers, Manuel Ramirez

10. Association of PACAP and PACAPR1 Gene Variants with Unipolar Depression and Panic Disorder

Angelika Erhardt, Susanne Lucae, Marcus Ising, Florian Holsboer, Elisabeth Binder

11. Genetic Variation in the PACAP-PAC1 Receptor (ADCYAP1R1) Gene is Associated with Increased Resting State Blood Flow in the Frontal and Limbic Regions among Adolescent Females with High Childhood Emotional Neglect

Amy E. Ramage, Suman Baddam, Megan N. Cullip, Rene L. Olvera, Douglas E. Williamson

Poster Session II—Tuesday

12. Exome Sequence Analysis of Finnish Patients with Clozapine-induced Agranulocytosis

Arun K. Tiwari, Anna C. Need, Clement C. Zai, Nabilah Chowdhury, Daniel J. Mueller (Müller), Anu Putkonen, Elia Repo-Tiihonen, Tero Hallikainen, A. Elif Anil Yağcıoğlu, Jari Tiihonen, James L. Kennedy, Herbert Y. Meltzer

13. Oxytocin Genotype may Modulate Reactivity to the Environment in Borderline Personality Disorder

M. Mercedes Perez-Rodriguez, Qiaoping Yuan, Zhifeng Zhou, Colin A. Hodgkinson, Laura Bevilacqua, Luis Ripoll, Marianne Goodman, Harold W. Koenigsberg, Pei-Hong Shen, David Goldman, Larry Siever, Antonia S. New

14. Genetic and Epigenetic Regulation of Catechol-O-Methyl-Transferase (COMT) are Associated with Impaired Fear Inhibition in Posttraumatic Stress Disorder

Seth D. Norrholm, Tanja Jovanovic, Alicia Smith, Elisabeth B. Binder, Torsten Klengel, Karen Conneely, Kristina B. Mercer, Jennifer Davis, Kimberly Kerley, Jennifer Winkler, Charles Gillespie, Bekh Bradley, Kerry J. Ressler

15. Relationship of SYNE1 and Processing Speed with Interference Resolution in Bipolar Disorder

Scott A. Langenecker, Aaron Vederman, Sebastian Zoellner, Masoud Kamali, Erika FH. Saunders, Melvin McInnis, Jon-Kar Zubieta, Margit Burmeister

16. Novel Repeat Polymorphism in the Catechol-O-Methyltransferase (COMT) Gene: Association with Cocaine Dependence and Age-related Changes in Brain Metabolism

Elena Shumay, Joanna Fowler, Nora D. Volkow

Poster Session II—Tuesday

17. Common and Rare Gain-of-function Alleles of the Serotonin Transporter Gene, SLC6A4, Associated with Tourette Disorder

Pablo R. Moya, Jens R. Wendland, Liza M. Rubenstein, Kiara R. Timpano, Anne M. Andrews, Gary A. Heiman, Jay A. Tischfield, Robert A. King, Sammanda Ramamoorthy, Francis J. McMahon, Dennis L. Murphy

18. An American Genetic Variant of DISC1 Disrupts NDEL1 Binding and Phosphorylation in Human Induced Pluripotent Stem Cell Derived Neural Progenitors

Lindsay Wilson, Sandra Engle, Zoe A. Hughes, Nicholas Brandon

19. Analysis of Genetic Effects and Heritability of SNPs and Their Interactions in Two Ethnical Populations for Smoking Dependence

Ming D. Li, Zhihong Zhu, Zhixiang Zhu, Jennie Z. Ma, Thomas J. Payne, Jun Zhu

20. The Effect of Distracting Noise on the Neuronal Mechanisms of Attention in Schizophrenia

Jason Tregellas, Jason Smucny, Lindsay Eichman, Donald Rojas

21. Safety, Pharmacokinetic and Positron Emission Tomography Evaluation of Serotonin and Dopamine Transporter Occupancy Following Multiple-dose Administration of the Triple Monoamine Reuptake Inhibitor BMS-820836

Ming Zheng, Lieuwe Appel, Roger Lane, David Burt, Feng Luo, Robert Risinger, Gunnar Antoni, Matthew Cahir, Sanjay Keswani, Wendy Hayes, Zubin Bhagwagar

Poster Session II—Tuesday

22. Differential Control of Learning and Anxiety along the Dorso-ventral Axis of the Dentate Gyrus

Mazen A. Kheirbek, Liam J. Drew, Daniel Costantini, Nesha Burghardt, Lindsay Tannenholz, Susanne E. Ahmari, Hongkui Zeng, Andre Fenton, Rene Hen

23. Anxiety Delays the Development of Fear Inhibition in Children at High Risk for Trauma Exposure

Tanja Jovanovic, Telsie Davis, Ami Smith, Jennifer Winkler, Seth D. Norrholm, Kerry J. Ressler, Bekh Bradley

24. A Non-competitive NMDA Antagonist AZD6765 Compared with Ketamine and Placebo on Pharmacology-MRI and Cognitive Mechanistic Biomarkers in Untreated Major Depressive Disorder; a Randomized Double-blind Controlled Trial

Bill Deakin, Steve Williams, Darragh Downey, Shane McKie, Guy Goodwin, Angela Rylands, Catherine Harmer, Kevin Craig, Colin Doursih, Gerard Dawson, Dennis McCarthy, Mark A. Smith

25. Ghrelin Intravenous Administration Increases Alcohol Craving in Alcohol-dependent Individuals: Preliminary Findings from a Human Laboratory Study

Lorenzo Leggio, William Zywiak, Samuel Fricchione, Steven Edwards, Robert Swift, George Kenna

26. Later Dinnertime is Associated with Abdominal Obesity in Patients with Bipolar Disorder

Isabella Soreca, Ellen Frank, David J. Kupfer

27. Differential Maturation of Subtypes of Perisomatic GABAergic Inputs in Monkey Prefrontal Cortex

Gil D. Hoftman, Kenneth Fish, David A. Lewis

Poster Session II—Tuesday

28. Adjunctive AZD6765, a Low-trapping NMDA Channel Blocker, in Treatment-resistant Major Depressive Disorder: A Randomized, Placebo-controlled Study

Gerard Sanacora, Mark A. Smith, Sanjeev Pathak, Hong-Lin Su, Dennis McCarthy

29. TRPA1 “Menthol Preference” Haplotypes are Associated with Levels of TRPA1 Expression and Smoking Cessation Success

George Uhl, Donna Walther, Frederique Behm, Jed Rose

30. Gene Expression Profiling in Selectively Bred Rat Lines that Differ in Addiction Liability

Shelly B. Flagel, Maria Waselus, Stanley J. Watson, Robert Thompson, Huda Akil

31. Just C NO to DREADDs: Inhibition of Ventral Pallidum Projection to Ventral Tegmental Area Blocks Cue-triggered Cocaine Seeking

Stephen V. Mahler, Elena M. Vazey, Jennifer Kaufling, Gary Aston-Jones

32. DLPFC Hyperactivation is Associated with the mir137 Schizophrenia Risk Genotype

Steven Potkin, Theo Van Erp, Ilaria Guella, Marquis P. Vawter, Federica Torri, Judith M. Ford, Kelvin O. Lim, Juan Bustillo, Ayse Belger, Adrian Preda, Dana Nguyen, Jessica Turner, Daniel H. Mathalon, Fabio Macciardi

33. Common Genetic Variation within Transcription Factor Binding Sites is Associated with Bipolar Disorder

David TW. Chen, Nirmala Akula, Liping Hou, Girma Hawariat, Sevilla Detera-Wadleigh, Xueying Jiang, BiGS Consortium, Francis J. McMahon

Poster Session II—Tuesday

34. Are Endophenotypes of Schizophrenia More or Less Heritable than the Disorder Itself in the COGS-1 Family Study?

Gregory A. Light, Tiffany A. Greenwood, Daniel J. Mathias, Neal R. Swerdlow, David L. Braff, COGS Investigators

35. Epigenetic Modulation of the Leukocyte Glucocorticoid Receptor and Childhood Parental Loss

Audrey R. Tyrka, Carmen Marsit, Lawrence H. Price, Yuliya I. Kuras, Noah S. Philip, Linda L. Carpenter

36. Excess Homozygosity in the MHC in Schizophrenia

Semanti Mukherjee, Saurav Guha, Anil Malhotra, Itsik Pe'er, Ariel Darvasi, Todd Lencz

37. Antipsychotic-induced Metabolic Abnormalities in Schizophrenia: The Prominent Role of Appetite Regulating Hypothalamic Genes

Daniel J. Mueller, Arun K. Tiwari, Nabilah I. Chowdhury, Natalie Freeman, Jeffrey A. Lieberman, Herbert Y. Meltzer, James L. Kennedy

38. eQTLs are Conserved across Brain Regions and Inform Genetic Association Results of Bipolar Disorder

Margit Burmeister, Viktoriya Strumba, Benjamin Keller, Ellen Schmidt, Matthew Flickinger, Elsbietta Sliwerska, Alan F. Schatzberg, Jack Barchas, William E. Bunney, Richard M. Myers, Stanley J. Watson, Jun Li, Laura Scott, Michael Boehnke, Huda Akil

39. The Heritability of Intelligence in a Combined Sample Community Controls and People with Schizophrenia

Dwight Dickinson, Joey Trampush, Ningping Feng, Bhaskar Kolachana, Richard Straub, Daniel R. Weinberger

Poster Session II—Tuesday

40. Behavioral Neurogenetics: Length of CAG Repeat in Huntingtin Below Disease Threshold Predicts Motor Skill and Behavior in Children

Peggy C. Nopoulos, Kathy Mathews, Eric Epping, Jane Paulsen

41. Pharmacogenetic Moderators of Methylphenidate and Guanfacine Response in Children and Adolescents with ADHD

Erika L. Nurmi, Karyn S. Mallya, James McGough, Sandra K. Loo, Robert M. Bilder, Fiona Whelan, James T. McCracken

42. Convergent Functional Genomics of Schizophrenia: From Comprehensive Understanding to Genetic Risk Prediction

Mikias Ayalew, Helen Le-Niculescu, Daniel Levey, Alan Breier, Anantha Shekhar, John Nurnberger, Mark A. Geyer, Ming Tsuang, Daniel Salomon, Nicholas Schork, Ayman Fanous, Michael C. O'Donovan, Alexander B. Niculescu

43. Identifying Rare Variants for Obsessive-Compulsive Disorder Using Family-based Whole Exome Sequencing Analysis: Preliminary Findings

Paul Arnold, Bingbin Li, Christian Marshall, Anath Lionel, Stephen Scherer, Gregory L. Hanna

44. De Novo Mutation of the Dopamine Transporter Gene Reveals a Novel Component of Autism Pathogenesis

James S. Sutcliffe, Peter Hamilton, Kevin Erreger, Andrea Belovich, Mark J. Daly, Aurelio Galli

45. The Genome-wide Supported Variant MicroRNA-137 Predicts Phenotypic Heterogeneity within Schizophrenia

Tristram Lett, Mallar Chakravarty, Virginia Goncalves, Arun K. Tiwari, Daniel Felsky, Jason Lerch, Eva Brandl, Jeffrey Lieberman, Herbert Y. Meltzer, James L. Kennedy, Aristotle Voineskos

Poster Session II—Tuesday

46. A System Level Transcriptomic Analysis in Schizophrenia Postmortem Brain

Panos Roussos, Pavel Katsel, Kenneth L. Davis, Larry Siever, Vahram Haroutunian

47. Elevated Omega-6/Omega-3 Fatty Acid Ratio Predicts Depression Development Following Interferon-Alpha Treatment: Relationship with Interleukin-6

Francis E. Lotrich, Barry Sears, Charles Reynolds, Robert McNamara

48. Low Vascular Endothelial Growth Factor and Interleukin-8 in Cerebrospinal Fluid of Suicide Attempters

Jussi Jokinen, Josef Isung, Shahin Aeinehband, Björn Mårtensson, Peter Nordström, Fredrik Piehl, Marie Åsberg

49. Influence of Acute Tryptophan Depletion on Attentional Performance in Adults with ADHD

Florian Daniel Zepf, Christian Mette, Marco Grabemann, Mona Abdel-Hamid, Jennifer Uekermann, Caroline Biskup, Jens Wiltfang, Bernhard Kis

50. DHEA and DHEAS as Potential Biomarker Candidates: Investigations in Over 600 Male OEF/OIF Era Veterans

Steven T. Szabo, Jason D. Kilts, Gillian Parke, Rajendra A. Morey, Lawrence Shampine, Jennifer Naylor, Robert M. Hamer, Christine E. Marx

51. Plasma Markers of Inflammation Correlate Directly Aggression and are Elevated in Intermittent Explosive Disorder

Emil F. Coccaro, Royce Lee, Mary Coussons-Read

Poster Session II—Tuesday

52. Elevated Transcript Levels for Viral Restriction Factors in Cortical Endothelial Cells in Schizophrenia

David W. Volk, Benjamin I. Siegel, Elizabeth J. Sengupta, Jessica R. Edelson, David A. Lewis

53. Broader Autism Phenotype: Relationships between Maternal/Paternal BAP, Parental SSRI Treatment, WB 5-HT and Child's Autism Symptoms

Tal Levin-Decanini, Nell Maltman, Guter Stephen, Edwin H. Cook, Suma Jacob

54. Toll Like Receptors in the Prefrontal Cortex of Depressed Suicide Victims

Ghanshyam N. Pandey, Hooriyah S. Rizavi, Xinguo Ren, Yogesh Dwivedi

55. Reduced μ -opioid Response to Social Rejection in Major Depressive Disorder

David T. Hsu, Benjamin J. Sanford, Kortni K. Meyers, Kathleen E. Hazlett, Brian J. Mickey, Scott A. Langenecker, Jon-Kar Zubieta

56. Neurochemical Effects of Ketamine Administration in Healthy Humans: An MRS Time-course Study

Lawrence S. Kegeles, Xiangling Mao, Najate Ojeil, Raffael Massuda, Mariana Pedrini, Chi-Ming Chen, Anissa Abi-Dargham, Mark Slifstein, Matthew Milak, Carolyn I. Rodriguez, Dikoma C. Shungu

57. Effects of 4mg Pioglitazone on Mnemonic Hippocampal Function: A Pharmacologic BOLD fMRI study in Healthy Elderly Adults

Ahmad Hariri, Annchen Knodt, Adam Gorka, James Burke, Kathleen Welsh-Bohmer, Brenda Plassman, Daniel Burns, Stephen Brannan, Michael Kukulka, Allen Roses

Poster Session II—Tuesday

58. Circadian Clock Gene Expression Rhythms in Cells from Patients with Bipolar Disorder Differ in Response to Lithium

Michael J. McCarthy, Hongbing Wei, Ryan Darvish, Donna McPhie, Bruce Cohen, David Welsh

59. Adjunctive Treatment with Asenapine Augments the Escitalopram-induced Effects on Monoaminergic and Glutamatergic NMDA as well as AMPA Receptor-mediated Transmission in the Medial Prefrontal Cortex

Monica M. Marcus, Olivia Frånberg, Carl Björkholm, Anna Malmerfelt, Kent Jardemark, Torgny H. Svensson

60. The Functional Significance of Antipsychotic-related Cortical Thinning in First Episode Schizophrenia

Tyler A. Lesh, Costin Tanase, Tara Niendam, Jong Yoon, J Daniel Ragland, Michael Minzenberg, Marjorie Solomon, Cameron Carter

61. GR-independent Corticosterone-Dopamine Interactions in the Nucleus Accumbens Mediate the Stress-induced Potentiation of Cocaine Seeking in Rats

Paul J. Gasser, Evan N. Graf, David A. Baker, Jayme McReynolds, Jonathan Hill, Amanda Ebben, Chung-Lung Chan, Mykel Robble, Oliver Vranjkovic, Daniel S. Wheeler, Robert A. Wheeler, John R. Mantsch

62. Basolateral Amygdala Microinfusion of Neuropeptide S (NPS) Rescues Behavior in a Rat Model of posttraumatic Stress Disorder (PTSD) by Increasing Expression of brain Derived Neurotrophic Factor and Neuropeptide YY1 Receptor

Hagit Cohen, Nitsan Kozlovsky, Zeev Kaplan, Joseph Zohar, Aleksander Mathé

Poster Session II—Tuesday

63. Inhibition of the Fatty Acid Amide Hydrolase (FAAH) in Acute Schizophrenia – A Mechanistically New Approach to its Treatment

F. Markus Leweke, Martin Hellmich, Franziska Pahlisch, Laura Kranaster, Dagmar Koethe

64. A Pilot Study on the Effects of Single Dose Intranasal Oxytocin on Social Cognition in Schizophrenia

Michael C. Davis, Junghee Lee, William Horan, Michael F. Green, Stephen R. Marder

65. Alterations in Synaptic Potentiation and Glutamatergic Signaling in Nicotine Abuse

Cassandra Gipson, Yonatan Kupchik, Kathryn Reissner, Peter Kalivas

66. Serum IgG Antibodies against the NR1 Subunit of the NMDA Receptor not Detected in Schizophrenia

Joseph C. Masdeu, Ana González-Pinto, Carlos Matute, Sonia Ruiz de Azúa, Aitor Palomino, Jose de Leon, Karen F. Berman, Josep Dalmau

67. Abnormalities of Glutamate Microdomains in Schizophrenia

Robert McCullumsmith, Dan Shan, Joy Roche, Vahram Haroutunian, James Meador-Woodruff, Rosalinda C. Roberts

68. Upregulation in the Expression of Key Serotonergic, Noradrenergic, and Cholinergic Genes in the Lower Brainstem in Major Depression: Neurobiological Substrate for Somatic Symptoms?

Aneesh Tyle, Nina S. Amilineni, Danielle A. Simpson, Edward G. Jones, William E. Bunney, Huda Akil, Stanley J. Watson, Ilan A. Kerman

Poster Session II—Tuesday

69. Alterations of Endogenous Cannabinoids in Complex PTSD and Borderline Personality Disorder

Juliane K. Mueller, Carola Schaefer, Frank Enning, J. Malte Bumb, Martin Hellmich, Dagmar Koethe, Christian Schmahl, Martin Bohus, F. Markus Leweke

70. Glutamate Signaling is Reduced and GABA Signaling Unaffected in Hippocampus in Schizophrenia

Ana D. Stan, Subroto Ghose, Yan Fang, Perry Mihalakos, Stephanie Morris, Sandeep Ganji, Changho Choi, Carol A. Tamminga

71. Functional Brain Basis of Hypnotizability

David Spiegel, Fumiko Hoeft, John DE Gabrieli, Susan Whitfield-Gabrieli, Brian Haas, Roland Bammer, Vinod Menon

72. Changes in Fusiform Gyrus Associated with Face Processing in Social Anxiety Disorder, before and after Treatment

Ardesheer Talati, Spiro Pantazatos, Joy Hirsch, Franklin R. Schneier

73. Markedly Reduced mGluR5 Receptor Binding in Smokers and Ex-smokers Determined by [11C]ABP688 Positron Emission Tomography

Gregor Hasler, Funda Akkus, Simon M. Ametamey, Valerie Treyer, Cyrill Burger, Anass Johayem, Daniel Umbricht, Baltazar Gomez Mancilla, Judit Sovago, Alfred Buck

74. Imaging the Sensitivity of [123I]5-IA-85380 to Increases in Acetylcholine at the Beta2-Nicotinic Acetylcholine Receptors in Human Subjects

Irina Esterlis, Jonas Hannestad, Frederic Bois, Andrew Sewell, Rachel Tyndale, John Seibyl, Marina Picciotto, John H. Krystal, Marc Laruelle, Richard Carson, Kelly Cosgrove

Poster Session II—Tuesday

75. Neurobiological Differences in Mentalization in Anorexia Nervosa

Carrie J. McAdams, Daniel C. Krawczyk, Graham Emslie

76. Neural and Behavioral Markers of Treatment Response in PTSD: A Longitudinal fMRI Study

Yuval Neria, Gregory M. Sullivan, Erel Shvil, Scott Schafer, Mariana Neria, Miriam Campeas, John C. Markowitz, Tor D. Wager, Mohammed R. Milad

77. Prefrontal Brain Activity Predicted by Dopaminergic Genes in Healthy Adults is Modulated by Antipsychotics in Schizophrenia

Thomas Weickert, Ans Vercammen, Ashley Skilleter, Rhoshel Lenroot, Cynthia S. Weickert

78. Neural Changes Associated with Attention Bias Modification Treatment: Implications for Anxiety Disorders

Jennifer Britton, Jenna Suway, Michelle Clementi, Yair Bar-Haim, Nathan Fox, Daniel S. Pine

79. Effects of Nicotine and Cannabis Use on Structural Connectivity in Brain's Reward-motivation Circuitry

Francesca M. Filbey, Sina Aslan, Arvind Caprihan, Judith Segall, Vincent Calhoun

80. Longitudinal Assessment of Neurochemical Changes in First-episode Mania following Lithium Treatment: An Interim MRS Analysis from the Bipolar Disorder Imaging and Treatment Research Center

David E. Fleck, Wen-Jang Chu, Richard Komoraski, James Eliassen, Jing-Huei Lee, Matthew Norris, Michael Cerullo, Caleb Adler, Melissa DelBello, Stephen Strakowski

Poster Session II—Tuesday

81. White Matter Abnormalities in Skin Picking Disorder: A Diffusion Tensor Imaging Study
Jon E. Grant, Brian L. Odlaug, Samuel R. Chamberlain, Adam Hampshire
82. Dopaminergic Networks: Brain Maturation and ADHD
Dardo Tomasi, Nora D. Volkow
83. Odor Modulation during an Alcohol Cue-reactivity Paradigm: A Functional Magnetic Resonance Imaging (fMRI) Preliminary Investigation
Bernadette M. Cortese, Thomas W. Uhde, Konstantin E. Voronin, Scott Henderson, Joseph P. Schacht, Qing X. Yang, Raymond F. Anton
84. Brain GABA Levels and Vitamin D3 in Manic Children and Adolescents
Elif Sikoglu, Ana Liso, Debra Starr, Michael Cirillo, Benjamin Nwosu, Ryan Rogan, Martha Castro, Richard Edden, Jean King, David Kennedy, Constance M. Moore, Jean Frazier
85. Altered Activation in Fronto-Striatal Circuits during Sequential Processing in Unmedicated Adults with Obsessive-Compulsive Disorder
Rachel Marsh, Bradley S. Peterson, Helen Blair Simpson, Zhishun Wang, Nidhi Parashar, Guillermo Horga
86. Brain Arachidonic Acid Metabolism is Upregulated in Rat Model of Human HIV-1 Infection and can be Dampened by Chronic Lithium: Relation to Bipolar Disorder
Stanley I. Rapoport, Mireille Basselin, Ipolia Ramadan

Poster Session II—Tuesday

87. Gray Matter Volumes in Pediatric Generalized Anxiety Disorder: A Voxel-Based Morphometry Study

Jeffrey Strawn, Anna M. Wehry, Michael Cerullo, James Eliassen, Stephen Strakowski, Caleb Adler, Melissa DelBello

88. An Examination of White Matter Aberrations in Youth at High-risk for Bipolar Disorder: A Tract-based Spatial Statistics Analysis

Donna Roybal, Naama Barnea-Goraly, Ryan G. Kelley, Spencer Boucher, Meghan Howe, Dylan Alegria, Kiki Chang

89. The use of MRI to Detect Regionally Specific Changes in White Matter Integrity Following Extended Methamphetamine Self-Administration in Rats

Carmela M. Reichel, Saeid Taheri, Ronald E. See

90. Functional Activation Differences during an Impulsivity Task Occur in Subjects with Bipolar Disorder Compared to Major Depressive Disorder during a Depressive Episode

Michael Cerullo, Martine Lamy, Christopher Smith, Brenda Milburn, David E. Fleck, James Eliassen, Jeffrey Strawn, Caleb Adler, Melissa DelBello, Stephen Strakowski

91. Self-regulation of Amygdala Activity with Real-time fMRI Neurofeedback in Patients with Depression

KyMBERLY Young, Raquel Phillips, Vadim Zotev, Wayne C. Drevets, Jerzy Bodurka

Poster Session II—Tuesday

92. Assessment of Brain Kappa Opioid Receptor Occupancy after Single Oral Doses of LY2456302 as Measured by PET with the Radioligand LY2879788 in Healthy Subjects

Johannes Tauscher, Francois Vandenhende, Jennifer Witcher, Mohini Ranganathan, Ming-Qiang Zheng, Mika Naganawa, Yiyun Huang, Alexander Neumeister, Richard E. Carson

93. Association of Depression with Hippocampal Volume: Results from an Epidemiological Sample

E. Sherwood Brown, Carroll W. Hughes, Roderick McColl, Ronald Peshock, A. John Rush, Julia Knypinski

94. Resting State Network Dynamics Predict Relapse in Cocaine-dependent Individuals

Meredith J. McHugh, Hong Gu, Yihong Yang, Jacquelyn Braud, Michael D. Devous, Richard W. Briggs, N. Robrina Walker, Bryon Adinoff, Elliot A. Stein

95. Subcortical Food Motivation Circuitry Dysfunction Associated with Endogenous Active Ghrelin Levels in Women with Anorexia Nervosa

Laura M. Holsen, Elizabeth A. Lawson, Kara Christensen, Anne A. Klibanski, Jill M. Goldstein

96. Resting-state Functional Connectivity and Neuroactive Steroids in Postpartum Depression

Kristina M. Deligiannidis, Elif M. Sikoglu, Scott A. Shaffer, Blaise Frederick, Constance M. Moore, Anthony J. Rothschild

97. Neural Mechanisms of Social Influence in Young Adult Drug Use

Jodi M. Gilman, Sang Lee, John Kuster, Byoungwoo Kim, Myoung Joo Lee, Paul Wighton, Andre van dew Kouwe, Anne Blood, Hans C. Breiter

Poster Session II—Tuesday

98. Damage Control: The Neural Basis of Reappraisal in Generalized Anxiety Disorder

Carmen Andreescu, Lei K. Sheu, Dana L. Tudorascu, Greg Siegle, Douglas Mennin, Howard J. Aizenstein

99. Diffusional Kurtosis Imaging of Frontal White Matter in Alcohol-dependent Young Adults with and without Attention Deficit Hyperactivity Disorder

Joseph P. Schacht, Ali Tabesh, Konstantin E. Voronin, Raymond F. Anton

100. APOE Genotype Modulates 1H-MRS Metabolites in the Aging Brain

Jesus J. Gomar, Marc L. Gordon, Peter B. Kingsley, Aziz M. Ulug, Koppel Jeremy, Concepcion Conejero-Goldberg, Peter Davies, Terry E. Goldberg

101. Membrane Phospholipid Turnover in First Degree Relatives of Schizophrenia Subjects

Konasale Prasad, Dhruvan Goradia, Krishna Pancholi, Steven Goodnow, Brent Oliveros, Matcheri Keshavan, Vishwajit Nimgaonkar, Jeffrey Stanley

102. Cortical, Diencephalic and Midbrain Gray Matter Volume in Alcoholism Measured by VBM: Effect of Co-morbid Substance Abuse

Daniel W. Hommer, Erica N. Grodin, Reza Momenan

103. Functional Connectivity between Broca's Area and the Default Mode Network is Associated with Auditory Hallucinations: An FBIRN Study

Judith M. Ford, Steven Potkin, Theo Van Erp, Brian J. Roach, Harshad Shanbhag, Adrian Preda, Ayse Belger, Vince Calhoun, Jessica Turner, Bryon Mueller, F BIRN, Daniel H. Mathalon

Poster Session II—Tuesday

104. Regional Brain Volume in ADHD and its Relationship to Ataxia and Intrasubject Variability

Eve Valera, Thomas Zeffiro, Rebecca Spencer, Nikos Makris, Stephen Faraone, Larry Seidman, Jeremy Schmahmann

105. White Matter Trajectories from Childhood into Late Adulthood

Bart D. Peters, Philip R. Szeszko, Toshikazu Ikuta, Kimberly Cameron, Patricia Gruner, Pamela DeRosse, John Cholewa, Anil Malhotra

106. Stimuli Presentation Modification Improves Diagnostic Accuracy at the Individual Level for Functional MRI Detection of Deception

Steven Laken, Andrew Lai, Kevin Johnson, Paul Morgan, Kimberly Levine, Michael Fernald, Ketan Patel, Kristy Hass, Jennifer Cahill, F. Andrew Kozel

107. A Mechanism of Ventricular Enlargement in an Alcohol Binge Model

Natalie M. Zahr, Edith Sullivan, Adolf Pfefferbaum

108. Sex Differences in the Effect of Childhood Emotional Abuse on Hippocampal Volume

Pamela DeRosse, Erin Samplin, Toshikazu Ikuta, Anil Malhotra, Philip R. Szeszko

109. A Study of Glutamatergic Function in Adolescent Males with High-functioning Autistic Disorder using Magnetic Resonance Spectroscopy: A Pilot Study at 4T

Gagan Joshi, Rachel Goldin, Dave Crowley, Scott Lukas, Atilla Gonenc

Poster Session II—Tuesday

110. Intrinsic Connectivity Abnormalities in Social Anxiety

Jennifer Blackford, Jacqueline Clauss, Suzanne Avery, Ross VanDerKlok

111. Brain Response to “Unseen” Cannabis Cues among Cannabis-dependent Individuals

Reagan Wetherill, Julian Bender, Kanchana Jagannathan, Yin Li, Charles P. O’Brien, Anna Rose Childress, Teresa R. Franklin

112. Impact of Genetic Risk for Bipolar Disorder on Emotion Regulation Circuitry in a Population of Symptomatic Youth

Danella Hafeman, Genna Bebeko, Michele A. Bertocci, Henry Chase, Susan Perlman, Amanda Hinze, Mary Kay Gill, Christine Demeter, Vaibhav Diwadkar, Jeffrey Sunshine, David A. Axelson, Boris Birmaher, Robert Kowatch, Robert L. Findling, Mary L. Phillips

113. Emotional Face Encoding in Youth at Risk for Bipolar Disorder: A Preliminary fMRI Study

Melissa A. Brotman, Brian L. Bones, Aviva K. Olsavsky, Nancy E. Adleman, Christen M. Deveney, Daniel P. Dickstein, Daniel S. Pine, Ellen Leibenluft

114. Multivariate Analysis of Neural Activity Patterns Measured by Functional MRI Reveals Distinct Signatures of Antipsychotic Drug Action

Thomas Mueggler, Celine Risterucci, Andreas Bruns, Basil Kuennecke, Edilio Borroni, Emilio Merlo Pich, Jean-Luc Moreau, Wettstein G. Joseph, Markus von Kienlin

115. Network-level Disruptions in Intrinsic Functional Connectivity across Psychotic Disorders

Justin T. Baker, Avram J. Holmes, Randy L. Buckner, Dost Ongur

Poster Session II—Tuesday

116. Brain Response to Stress and to Smoking Cues Predicts Relapse to Tobacco Smoking

Christian G. Schütz, Martin Landsberg, Marcel Daamen, Gisela Bopp, Lukas Scheef

117. Greater Activation in the Anterior Cingulate Cortex is Related to Less Social Anxiety in Young Adults at Temperament-based Risk

Jacqueline A. Clauss, Suzanne Avery, Ross VanDerKlok, Jennifer Urbano Blackford

118. Neuroanatomical Correlates of Altered Immune Function in Late-life Depression

Olusola Ajilore, Ghanshyam N. Pandey, Rebecca Charlton, Laura Korthauer, Melissa Lamar, Shaolin Yang, Xinguo Ren, Anand Kumar

119. Naltrexone Modulates Anticipatory Responses to Reward

Tiffany Love, Chelsea Cumminford, Brian J. Mickey, Joseph Heffernan, Evan Chang, Jon-Kar Zubieta

120. Genetic Studies of PTSD in Ohio National Guard Soldiers Deployed to Iraq and Afghanistan: Replication of G x E interaction in FKBP5 and 5-HTTLPR

Anthony P. King, Peng Zhang, Sebastian Zoellner, Michael Camilleri, Sandro Galea, Marijo Tamburrino, Joseph R. Calabrese, Israel Liberzon

121. Auditory Steady State Response and Auditory Cortex Volume Deficits in Schizophrenia

Yoji Hirano, Naoya Oribe, Robert W. McCarley, Kevin M. Spencer

Poster Session II—Tuesday

122. Transcriptional Profiling of Multiple Brain Regions from Matched Cohorts of Subjects with Schizophrenia, Bipolar Disorder, and Major Depressive Disorder

Thomas A. Lanz, Simon Xi, Diogo M. Camacho, Susan E. Bove, Veronica Reinhart, Max Kuhn, Phillip Yates, Dmitri Volfson, David A. Lewis, Robin J. Kleiman, Nicholas Brandon

123. High Rate of Rare and of Disease Related Copy Number Variants in Childhood Onset Schizophrenia

Judith L. Rapoport, Kwangmi Ahn, Nitin Gogtay, Peter Gochman, Tiffany Andersen

124. A Factor Analysis of GABAergic Gene Expression across 16 Brain Regions in Human Postmortem Samples Identifies Specificity in Response to Chronic Alcohol and Cocaine Exposure

Mary-Anne Enoch, Basel Baghal, Qiaoping Yuan, David Goldman

125. Caudate Activation Changes and Dose Response during Attention and Learning Tasks in Antipsychotic Treatment Studies of First Episode Schizophrenia

Sarah Keedy, James Reilly, Margret Harris, Jeffrey R. Bishop, Peter Weiden, John Sweeney

126. Buprenorphine Implants for the Treatment of Opioid Dependence: Six and 12 Month Outcomes

Katherine L. Beebe, Steven Chavoustie, Walter Ling, Stacey Sigmon, Deborah Leiderman, Genie Bailey

127. Does the MATRICS Consensus Cognitive Battery Detect Pro-cognitive Effects of Amphetamine in Healthy Adults?

Hsun-Hua Chou, Jo A. Talledo, Sarah N. Lamb, Wesley K. Thompson, Neal R. Swerdlow

Poster Session II—Tuesday

128. Advanced Paternal Age and DNA Methylation Abnormalities in Psychiatric Disease

Maria H. Milekic, Yurong Xin, Anne O'Donnell, Kevin Kumar, John Edwards, Timothy Bestor, Victoria Haghighi, Jay A. Gingrich

129. Distinguishing Bipolar Disorder from Major Depressive Disorder in Adolescents: An FMRI Study

Jill Russo, Fei Wang, Hilary Blumberg

130. Pre-attentive Information Processing and Impulsivity in Bipolar Disorder

Alan C. Swann, Marijn Lijffijt, Scott Lane, Joel Steinberg, F. Gerard Moeller

131. Extracellular Matrix Abnormalities in Olfactory Epithelium Tissue from Subjects Diagnosed with Schizophrenia

Harry Pantazopoulos, Anne Boyer-Boiteau, Steven E. Arnold, Sabina Berretta

132. A Specific Blood Gene Expression Pattern is Correlated with a Neurophysiological Abnormality in Deficit Syndrome of Schizophrenia

Yasushi Kajii, Ikwunga Wonodi, Elliot Hong, Gunvant K. Thaker

133. Neurocognitive Functioning and Impairment in Awareness of Illness in Schizophrenia: Baseline Correlations and Treatment Effects

Philip D. Harvey, Cynthia Siu, Josephine Cucchiaro, Andrei Pikalov, Antony Loebel

134. N-Acetylcysteine in the Treatment of Pediatric Trichotillomania: A Randomized, Double-blind, Placebo-controlled Trial

Michael Bloch, Kaitlyn Panza, Jon E. Grant, James Leckman, Christopher Pittenger

Poster Session II—Tuesday

135. Evidence of an Inflammatory Pathway Leading to Psychosis in Bipolar Disorder

Mikael Landén, Carl Sellgren, Magdalena Kegel, Carl-Johan Ekman, Patrick Sullivan, Jordan W. Smoller, Pamela Sklar, Göran Engberg, Sophie Erhardt

136. Psychometric Evaluation of the Brown Assessment of Beliefs Scale in Body Dysmorphic Disorder and Obsessive-Compulsive Disorder

Katharine A. Phillips, Ashley S. Hart, Jane L. Eisen, William Menard, Nicholas J. Sibrava, Steven Rasmussen

137. Polymorphisms within PP-fold Polypeptide Pathway May Contribute to Susceptibility of Eating Disorders and related Endophenotypes

Pei-an Betty Shih, Nicholas Schork, Wade Berrettini, Andrew Bergen, Pierre Magistretti, Cinnamon Bloss, Ashley Van Zeeland, Walter Kaye

138. The Sex Biased Phosphoproteome: A Novel Approach Towards Understanding The Molecular Basis for Sex Differences in Neuropsychiatric Diseases

Rita Valentino, Debra Bangasser, Zach Plona, Hua Ding, Christopher McKennan, Steven Seeholzer

139. Gamma Ventral Capsulotomy for Severe Obsessive-Compulsive Disorder: A Double-blind, Randomized Controlled Trial

Euripedes Constantino Miguel, David Pauls, Benjamin Greenberg, Marcelo Hoexter, Marcelo Batistuzzo, André Gentil, Roseli G. Shavitt, Carlos A. Pereira, Juliana Belo. Diniz, Antonio C. Lopes, Georg Noren, Steven Rasmussen

Poster Session II—Tuesday

140. Resting Regional Cerebral Blood Flow to Predict Relapse in Cocaine Dependent Individuals

Hong Gu, Meredith J. McHugh, Yihong Yang, Conner Shope, Jacquelyn Braud, Michael D. Devous, Richard W. Briggs, N. Robrina Walker, C. Munro Cullum, Elliot A. Stein, Bryon Adinoff

141. Polygenic Variation across the Folate Metabolic Pathway Influences Frontal Cortical Thickness: Implications for Altered Neurodevelopment in Schizophrenia

Joshua L. Roffman, Noah J. Silverstein, Avram J. Holmes, Phil H. Lee, Marisa O. Hollinshead, Jordan W. Smoller, Randy L. Buckner

142. Convergence of Two Key Pathways of Schizophrenia: DISC1-Serine Racemase Interaction in Astrocytes

Mikhail V. Pletnikov, Martin Ma, Sofya Abazyan, Bagrat Abazyan, Akira Sawa, Solomon H. Snyder

143. Effects of Two-week Chronic Treatment with Phendimetrazine or its Primary Active Metabolite, Phenmetrazine, on Choice between Cocaine and Food in Rhesus Monkeys

Matthew L. Banks, Bruce Blough, Steve Negus

144. Altered Functional and Structural Brain Connectivity in First-episode Psychosis

Tom A. Hummer, John D. West, Yang Wang, Nikki Mehdiyoun, Jenifer L. Vohs, Michael M. Francis, Emily Liffick, Brenna C. McDonald, Andrew J. Saykin, Alan Breier

145. Monoamine Oxidase A in Subtypes of Depression: A [C-11] Harmine Positron Emission Tomography Study

Lina Chiucciariello, Sylvain Houle, Laura Miler, Robert G. Cooke, Robert D. Levitan, Stephen J. Kish, Alan A. Wilson, Pablo Rusjan, Jeffrey H. Meyer

Poster Session II—Tuesday

146. Increased Expression of Neurokinin-1 Receptors in the Central Nucleus of the Amygdala Mediates Escalated Alcohol Self-administration and Increased Potency of Neurokinin-1 Receptor Antagonism in Alcohol Preferring P Rats

Jesse R. Schank, Hui Sun, Courtney King, Jenica Tapocik, Estelle Barbier, Kejun Cheng, Kenner Rice, Markus Heilig

147. Major Depressive Disorder is Associated with Abnormal Responses in the Dorsal Mid-insula during Attention to Interoceptive States

William K. Simmons, Jason Avery, Joel Barcalow, Scott Mosemann, Jerzy Bodurka, Wayne C. Drevets

148. Smooth Pursuit Eye Movement, Prepulse Inhibition, and Auditory Paired Stimuli Processing Endophenotypes across the Schizophrenia - Bipolar Disorder

Elena I. Ivleva, Amanda F. Moates, Jordan Hamm, Ira Bernstein, Darwynn Cole, Brett Clementz, Guntavt Thaker, Carol A. Tamminga

149. Adjunctive Topiramate in Patients with Schizophrenia-spectrum Disorders: Results from a Meta-analysis of 9 Randomized Controlled Trials

Christoph U. Correll, Lawrence Maayan, Marc De Hert, Dan Cohen

150. Alcohol-induced Conditioned Place Preference in Healthy Moderate Drinkers

Emma L. Childs, Harriet de Wit

151. Increased Developmental Serotonin Influences Response to Psychotomimetics in Adulthood

Caitlin E. McOmish, Elena Y. Demireva, Natalie A. Diacovo, Alice M. Rolland, Jay A. Gingrich

Poster Session II—Tuesday

152. Epigenetics, Neurodevelopment, and Risk for Anxiety and Depression in Model Rats
Sarah M. Clinton, Rebecca K. Simmons, Matthew E. Glover, Phyllis C. Pugh, Huda Akil
153. Phospholipase C Signaling within the Central Nucleus of the Amygdala Maintains Binge Alcohol Drinking by Mice
Justin A. Courson, Emily Lum, Karen K. Szumlinski
154. Juvenile Methylphenidate Interacts with Dopamine and Estrogen Levels to Modulate Dopamine Receptors in Prefrontal Cortex of Female Rats
Jodi Lukkes, Britta S. Thompson, Kai Sonntag, Susan L. Andersen
155. Ovarian Hormones Modulate Working Memory Related Hippocampal-DPLFC Connectivity in Women - A Positron Emission Tomography (PET) Study
Katherine Damme, Shau-Ming Wei, Erica Baller, Jeffrey Bloch, Phillip Kohn, Shane Kippenhan, David Rubinow, Pedro Martinez, Peter Schmidt, Karen Berman
156. Effects of Cocaine Self-administration and Extinction on Astrocyte Content and Protein Expression in the Nucleus Accumbens, and Relationship to Reinstatement
Kathryn Reissner, Heather Boger, Peter Kalivas
157. Effects of Early Prefrontal Cortex NMDA Receptor Dysfunction on Impulsivity in Adulthood
Janet Finlay, Ginger Dunham, Robert Greene
158. Guided by Your Heart: Cardiac Cycle Modulates the Awareness and Perceived Intensity of Fear Stimuli
Sarah N. Garfinkel, Hugo D. Critchley

Poster Session II—Tuesday

159. Insight, Treatment Outcomes and Recovery in First-episode Schizophrenia

Ofer Agid, Cynthia Siu, Robert B. Zipursky, Gary Remington

160. Precision Delivery of Viral Vectors within Discrete Brain Substructures of the Rodent

Miles G. Cunningham, Sivan Subburaju, Andrew Coleman, Nand Kishore, Francine Benes

161. Genetic Variation in Endocannabinoid Signaling Moderates the Association between Childhood Maltreatment and Amygdala Habituation

Ryan Bogdan, Caitlin E. Carey, Emily M. Drabant, Arpana Agrawal, Sean D. Kristjansson, Ahmad R. Hariri

162. A Randomized, Double-blind, Placebo-controlled Add-on Treatment of Benzoate, a D-Amino Acid Oxidase Inhibitor, for Schizophrenia

Guochuan Emil. Tsai, Hsien-Yuan Lane, Michael F. Green

163. Unlike Serotonin, Clozapine Induced, 5-HT_{2A}R-mediated Signaling and Behavioral Events are Beta-Arrestin2 Independent

Cullen L. Schmid, John M. Streicher, Laura M. Bohn

164. Cannabinoid Facilitation of Extinction Recall Via Increased Recruitment of Prefrontal-hippocampal Circuitry in Healthy Humans

Christine A. Rabinak, Mike Angststadt, Chandra Sripada, Mohammed R. Milad, Israel Liberzon, K. Luan Phan

165. Detecting Disease-specific Differences in Mitochondrial Morphology and Distribution in Fibroblasts from Patients with Bipolar, Schizophrenic, and Major Depressive Disorders

Donna L. McPhie, David Logan, Laura Sargent, John-Thomas Berry, Caitlin Ravichandran, Anne Carpenter, Bruce Cohen

Poster Session II—Tuesday

166. Emotional Processing Changes in Depressed Patients following Antidepressant Treatment: An fMRI Study

Sidney Kennedy, Sakina Rizvi, Jakub Konarski, Jonathan Downar, Roger McIntyre, Tim Salomons

167. Appraisal of Immediate and Delayed Reward is Impaired with Amygdalar-Striatal Abnormalities in Pediatric Bipolar Disorder

Alessandra M. Passarotti, Minjie Wu, Aneesh Nandam, Mani Pavuluri

168. Depressed Adolescents Show Less Enhancement of Positive Affect and Weaker Fronto-striatal Connectivity while Recalling a Positive Experience

Dana L. McMakin, Jennifer Silk, Thomas Olino, Ronald Dahl, Kyung Hwa Lee, Erika E. Forbes, Neal Ryan, Greg Siegle

169. Lipid Correlates of Antipsychotic-induced White Matter Changes in First-episode Psychosis

Philip R. Szeszko, Delbert Robinson, Toshikazu Ikuta, Bart D. Peters, Juan A. Gallego, John Kane, Anil Malhotra

170. Depot-Naltrexone Modulates Limbic Brain Activity and Reduces Smoking In Heroin Dependent Patients

Daniel D. Langleben, Kanchana Jagannathan, Igor Elman, Catherine P. Koola, An-Li Wang, Shira Blady, Anna Rose Childress, Charles P. O'Brien

171. Dopamine Activity and Reward Processing in Smokers before and after Smoking Cessation: Combined [18F]FDOPA/fMRI Studies

Lena Rademacher, Susanne Prinz, Katja N. Spreckelmeyer, Oliver Winz, Jörn Schmaljohann, Felix Mottaghy, Ingo B. Vernaleken, Gerhard Gründer

Poster Session II—Tuesday

172. Functional Connectivity During Reward Processing in Adolescents with Bipolar I Disorder

Manpreet K. Singh, Ryan G. Kelley, Erica Marie Sanders, Meghan Howe, Ian Gotlib, Kiki Chang

173. Greater MAO-A Binding in Borderline Personality Disorder: An [11C] Harmine PET Study

Nathan J. Kolla, Alan A. Wilson, Sylvain Houle, Paul Links, Shelley McMain, Michael Bagby, Jeffrey H. Meyer

174. Developmental Trajectory of Sensory Cortical Gamma Synchrony Using Steady State Auditory Evoked Potentials

Raymond Y. Cho, Christopher Walker, Nicola Polizzotto, Catherine Fissell, Thomas Wozny, Chi-Ming Chen

175. Subgenual Cingulate Function in Borderline Personality Disorder and Major Depression

Brian J. Mickey, Alexander Andrews, Chelsea Cumminford, Mary Heitzeg, Scott A. Langenecker, Tiffany Love, Benjamin J. Sanford, Kenneth Silk, Jon-Kar Zubieta

176. Chronic HDAC Inhibitor Treatment Alters Brain Glucose Metabolism in Rats: Examining 18F-Fluorodeoxyglucose Uptake with Positron Emission Tomography

Frederick A. Schroeder, Michael Granda, Anna Cha, Chris Moseley, Stephen J. Haggarty, Jacob M. Hooker

177. Experimentally Delayed Puberty Interacts with Social Stress to Produce Widespread Effects on the Development of Brain White Matter Tracts and Behavior in Rhesus Monkeys

Jodi R. Godfrey, Brittany Howell, Xiaodong Zhang, Govind Nair, Xiaoping Hu, Mark Wilson, Mar Sanchez

Poster Session II—Tuesday

178. Regional Analysis of Naltrexone Effects on Mu-Opioid Availability In Vivo
Chelsea Cummmiford, Tiffany Love, Robert Koeppe, Jon-Kar Zubieta
179. Multivariate Pattern Classification Reveals Distributed Effects of Oxycodone on the Resting Brain Connectome in Humans
Chandra Sripada, Daniel Kessler, Mike Angstadt, Robert C. Welsh, Scarlet Guo, K. Luan Phan
180. Effects of Levodopa-Carbidopa-Entacapone on Neural Responses to Cocaine-related Stimuli and Non-drug Rewards in Regular Cocaine Smokers
Gillinder Bedi, Nehal P. Vadhan, Edward V. Nunes, Richard W. Foltin, Adam Bisaga
181. Evocative Cues Trigger Limbic Motivational Circuits and Differentially Disrupt “Cognitive Control” in Real-time Functional Magnetic Resonance Imaging
Anna Rose Childress, Jeremy Magland, Oscar Bartra, Jessie Lupardus, Robert Fabianski, Shing Chun Lam, Kimberly Young, Jesse Suh, Teresa Franklin, Daniel Langleben, Charles P. O’Brien
182. Anomalous Insula - Amygdala Functional Connectivity in Borderline Personality Disorder Patients is Associated with Impaired Psychological Habituation to Negative Stimuli
Harold W. Koenigsberg, Bryan Denny, Jin Fan, Xun Liu, Liza Rimsky, Hannah Pakray, Antonia S. New, Larry J. Siever
183. Effects of GABAergic Manipulation of Resting State Brain Networks in Psychotic and Healthy Subjects: A Connectomic Pattern Classification Analysis
Chandra Sripada, Robert C. Welsh, Daniel Kessler, K. Luan Phan, Stephan F. Taylor

Poster Session II—Tuesday

184. Pretreatment Orbitofrontal Thickness as a Measure to Classify Patients with Obsessive-Compulsive Disorder in Responders and Non-responders

Marcelo Q. Hoexter, Antonio C Lopes, Roseli G. Shavitt, Marcelo Batistuzzo, Darin D Dougherty, Fabio L S Duran, Juliana Belo Diniz, Geraldo F Busatto, Euripedes C Miguel, Rodrigo A Bressan, João R Sato

185. Amygdala, Prefrontal Cortex, and Cingulate Relationships in Combat Veterans with Traumatic Brain Injury and Post-Traumatic Stress Disorder

Monte S. Buchsbaum, Alan Simmons, Alex DeCastro, Scott Matthews

187. Association of Glutamate and N-Acetylaspartate in Schizophrenia Before and after Treatment with Risperidone: A Proton Magnetic Resonance Spectroscopy Study

Adrienne C. Lahti, Meredith A. Reid, Nina V. Kraguljac, David M. White, Jan den Hollander

188. Multimodal Neuroimaging Correlates of Repetitive Mild Traumatic Brain Injury in Iraq and Afghanistan War Veterans

Eric C. Petrie, Donna J. Cross, Vasily Yarnykh, Todd Richards, Nathalie Martin, Satoshi Minoshima, Murray A. Raskind, Elaine R. Peskind

189. Early Life Stress and Functional Connectivity within Major Depression

Merida Grant, Jennifer Hadley, David M. White, Richard C. Shelton

190. Dopaminergic Activity and Altered Reward Modulation in Anorexia Nervosa

Ursula F. Bailer, Julie C. Price, Carolyn C. Meltzer, Angela Wagner, Chester A. Mathis, Walter H. Kaye

Poster Session II—Tuesday

191. Imaging Emotion Circuits to Predict Treatment Outcomes in Major Depressive Disorder: The International Study to Predict Optimized Treatment in Depression

Leanne Williams, Mayuresh Korgaonkar, Yun Ju Song, Sarah Eagles, Anthony Harris, Stephen Koslow, Stuart Grieve, Amit Etkin

192. Strength of Frontal-hippocampal Functional Coupling Predicts Hippocampal Responses during Fear Learning in Schizophrenia

Garth Coombs, Donald Goff, Mohammed R. Milad, Daphne J. Holt

193. Functional Connectivity in Ventromedial Prefrontal Cortex is Altered in Depression: A Novel Biomarker Supported by Parallel Rodent and Human Neuroimaging Studies

Conor Liston, Logan Grosenick, Karl Deisseroth, Wenbiao Gan, Marc Dubin

194. In Vivo Serotonin Transporter Binding Predicts Reactivity to Emotional Conflict in Prefrontal Cortex

Julia Sacher, Swen Hesse, Marianne Patt, Georg Becker, Franziska Moeller, Karsten Mueller, Matthias L Schroeter, Arno Villringer, Osama Sabri

195. The Autism Brain Imaging Data Exchange: Towards Large-scale Evaluation of the Intrinsic Brain in Autism

Adriana Di Martino, ABIDE Consortium

196. The Efficacy of an Adenovirus-based Anti-cocaine Vaccine to Reduce Cocaine Self-administration in Rhesus Monkeys using a Choice Procedure

Suzette M. Evans, Richard W. Foltin, Martin J. Hicks, Jonathan B. Rosenberg, Bishnu P. De, Kim D. Janda, Stephen M. Kaminsky, Ronald G. Crystal

Poster Session II—Tuesday

197. Controlled Trial of Cognitive Remediation in Psychotic Bipolar Disorder: Feasibility, Tolerability, and Development of a Web-based Computer Control

Kathryn E. Lewandowski, Bruce Cohen, Sarah Sperry, Dost Ongur, Matcheri Keshavan

198. Exercise Improves Physical Capacity in Obese Patients with Schizophrenia: Pilot Study

Martin T. Strassnig, John W. Newcomer, Philip Harvey

199. Is Repetitive Transcranial Magnetic Stimulation Effective in Treatment Resistant Depression?: A Systematic Review

Bradley N. Gaynes, Stacey Lloyd, Linda Lux, Gerald Gartlehner

200. Functional Connectivity of Attention Network Relates to Drink Volume in Youth

Barbara Weiland, Robert Welsh, Mary Soules, Crosby Modrowski, Robert Zucker, Jon-Kar Zubieta, Mary Heitzeg

201. Comparison of the Neurocognitive Effects between Acute Course Magnetic Seizure Therapy and Electroconvulsive Therapy: A Preliminary Report

Shawn M. McClintock, Mustafa M. Husain, C. Munro Cullum, Bruce Luber, Paul Croarkin, Angel Peterchev, Kenneth Trevino, Mohamed Aly, Louis Stool, Ahmad Raza, Sarah Lisanby

202. Electroconvulsive Therapy Potentiates the Inhibition of Gamma Oscillations in Treatment Resistant Depression

Mera S. Barr, Lakshminarayan V. Chinta, Natasha Radhu, Marina V. Frantseva, Daniel M. Blumberger, Andrea J. Levinson, Zafiris J. Daskalakis

Poster Session II—Tuesday

203. Changes in Default Mode Resting State fMRI Following ECT in Major Depression

Georgios Petrides, Miklos Argyelan, Styliani Kaliora, Philip R. Szeszko

204. Prefrontal rTMS Versus tDCS Effects on Perceived Controllability and the Emotional Dimension of Pain: Implications for Mechanisms of Technology-specific Action

Jeffrey J. Borckardt, Scott Reeves, Alok Madan, Jennifer Naylor, Sarah Fredrich, Heather Frohman, Kelly Barth, Mark S. George

205. Oxytocin Differentially Decreases Methamphetamine Intake and Reinstatement to Methamphetamine Seeking in Male and Female Rats

Carmela M. Reichel, Brittney Cox, Amy Young, Ronald E. See

206. Maternal Experience Affects Drug Abuse Vulnerability in the Female Rat

Jennifer Cummings, Jill B. Becker

207. Phenotypic Heterogeneity Reduces the Power of Genomewide Association Studies

Martin Alda, Jeff Cullis, Mirko Manchia, Rudolf Uher

208. Assessing Motivation in Schizophrenia in a Virtual Environment: Development of a Novel Ecologically Valid, Objective Assessment Methodology

George Foussias, Ishraq Siddiqui, Nasteho Hasan, Krysta McDonald, Sathesan Thavabalasingam, Christina Plagiannakos, John Zawadzki, Konstantine K. Zakzanis, Paul Fletcher, Albert Wong, Gary Remington

Poster Session II—Tuesday

209. Brain Region Specific Circadian Disruptions in a Mouse Model of Major Depressive Disorder

Nicole Edgar, Andrea Gillman, Colleen A. McClung

210. Sex, Stress and Cocaine: Role of Corticotropin Releasing Factor in Behavioral and Dopaminergic Sensitization to Cocaine

Elizabeth N. Holly, Akiko Shimamoto, Joseph F. DeBold, Klaus A. Miczek

211. Sex Differences in Fear Extinction in Men and Women Exposed to Trauma

Sabra S. Inslicht, Thomas Metzler, Mohammed R. Milad, Scott P. Orr, Charles R. Marmar, Thomas Neylan

212. Expression Patterns of Genes Hemideleted in Williams Syndrome: Developmental and Allelic Variation Effects in Human Brain

Chao Li, Barbara Lipska, Thomas M. Hyde, Ran Tao, Shane Kippenhan, Andrew Jaffe, Liqin Wang, Tianzhang Ye, Carlo Colantuoni, Bhaskar S. Kolachana, Venkata S. Mattay, Daniel R. Weinberger, Joel E. Kleinman, Karen F. Berman

214. Frontostriatal Activation Abnormalities during Sustained Attention and Inhibitory Control among Patients with Late Life Depression

Sara L. Weisenbach, Bridget Cornett, Erich Avery, Brennan Haase, Jon-Kar Zubieta, Scott Langenecker

Wednesday Posters

Poster Session III – Wednesday



Advocacy Affiliate - International Mental Health Resource Organization
“IMHRO Rising Star Awards”

Cindy Dyar, Brandon Staglin

1. Membership Advisory Task Force: Feedback, Challenges, and Solutions in 2012

Katherine E. Burdick, Linda Carpenter, Marlene Freeman, Vaishali Bakshi, Paul Holtzheimer, Lisa Monteggia, Thomas Schulze, Carlos Bolanos-Guzman, Kristin Cadenhead, Raymond Cho, Cynthia Crawford, Paul Kenny, Gregory Light, Gonzalo Laje

2. Treatment with Adjunctive Aripiprazole Results in Significant Improvement Compared with Continued Antidepressant Monotherapy in Patients with Mild, Moderate, and Severe Major Depressive Disorder

J. Craig Nelson, Thomas D. Stewart, Ainslie Hatch, Kimberly Largay, Elizabeth E. Bellochio, Sabrina V. Marler, Ross A. Baker, John Sheehan, Robert M. Berman

3. Genomic Predictors of Response to Antidepressant Treatment in Geriatric Depression using Genome-wide Expression Analyses: A Pilot Study

Helen Lavretsky, Ascia Eskin, Stanley Nelson, Steve Cole

4. The Acetylcholinesterase Inhibitor, Rivastigmine, but not Huperzine A, Improves Verbal Learning/Episodic Memory and Working Memory in Cocaine-dependent Volunteers

James Joseph Mahoney, Ari Kalechstein, Thomas Newton, Ryan Bennett, Nicholas Arnoudse, Richard De La Garza

5. Treatment of Depression with Botulinum Toxin A: A Randomized, Double-blind, Placebo Controlled Trial

Eric Finzi, Norman Rosenthal

Poster Session III—Wednesday

6. Adjunctive Aripiprazole More than Doubles the Rate of Early and Sustained Response across Multiple Measures in Patients with MDD who have an Inadequate Response to Antidepressant Monotherapy

Daniel E. Casey, Kimberly Laubmeier, Eudicone James, Ronald N. Marcus, Ross A. Baker, John Sheehan, Robert M. Berman

7. A Randomized Controlled Crossover Trial of Ketamine in Obsessive-Compulsive Disorder

Carolyn I. Rodriguez, Lawrence S. Kegeles, Amanda Levinson, Sue Marcus, Helen Blair Simpson

8. Safety and Tolerability of Atomoxetine Hydrochloride in a Placebo-controlled Randomized Withdrawal Study in Adults with Attention-Deficit/Hyperactivity Disorder

Himanshu P. Upadhyaya, Angelo Camporeale, J. Antoni Ramos-Quiroga, David Williams, Yoko Tanaka, Jeannine Lane, Robert R. Conley, Rodrigo Escobar, Paula Trzepacz, Albert J. Allen

9. Efficacy and Safety of Cariprazine in Acute Exacerbation of Schizophrenia: A Phase III, International, Randomized, Double-blind, Placebo-controlled Trial

Stephen R. Zuckin, John Kane, Andrew J. Cutler, Yao Wang, Oksana Mokliatchouk, Krisztián Nagy, István Lászlószky, Suresh Durgam

10. Rasagiline in the Treatment of the Persistent Negative Symptoms of Schizophrenia

Robert W. Buchanan, Elaine Weiner, Deanna L. Kelly, Robert P. McMahon, James M. Gold, David Gorelick

11. Effects of Suvorexant, an Orexin Receptor Antagonist, on Next Day Driving Performance in Healthy Volunteers

Annemiek Vermeeren, Eric Vuurman, Anita Van Oers, Cees Van Leeuwen, Stefan Jongen, An Bautmans, Xiaodong Li, Tara Siringhaus, Ingeborg Heirman, Tine Laethem, Matthew Troyer, David Michelson, Hong Sun

Poster Session III—Wednesday

12. Effects of the Mu-Opioid Receptor Antagonist GSK1521498 on Brain Responses to Food Cues, Hedonic and Consummatory Eating Behaviour and Bodyweight: A Proof of Mechanism Study in Binge Eating Obese Subjects

Pradeep Nathan, Hisham Ziauddeen, Samuel R. Chamberlain, Victoria Cambridge, Mark Bush, Wenli Tao, Annelize Koch, Chris Dodds, Kay Maltby, Andrew Skeggs, Lucy Cheke, Sadaf Farooqi, Stephen O’Rahilly, Paul Fletcher, Edward Bullmore

13. Mindfulness Training Improves Resilience: Reductions in Adrenocorticotrophic Hormone (ACTH) Response to Laboratory Stress

Elizabeth Hoge, T.H. Eric Bui, Christina Metcalf, Mark H. Pollack, Naomi M. Simon

14. A Phase III, Double-blind, Placebo-controlled, Flexible-dose Study of Levomilnacipran SR in Patients With Major Depressive Disorder

Angelo Sambunaris, Anjana Bose, Carl Gommoll, Changzheng Chen, William M. Greenberg, Stephen R. Zukin, David V. Sheehan

15. Relapse Risk after Discontinuation of Risperidone in Alzheimer’s Disease

Davangere P. Devanand, Susan Schultz, Jacobo Mintzer, David Sultzer, Gregory Pelton, Howard Andrews, Danilo de la Pena, Sanjay Gupta, Corbett Schimming, Sylvia Colon, Bruce Levin

16. Predictors of Middle-of-the-night Dosing in Primary Insomnia Subjects (Sleep Maintenance Type) Participating in a 4-Week Outpatient Clinical Trial of 3.5 mg Sublingual Zolpidem Tartrate versus Placebo

Thomas Roth, Wallace Mendelson, Nikhilesh Singh, Frank J. Steinberg, Margaret Moline

Poster Session III—Wednesday

17. Partial Adherence to Antipsychotic Treatment and Return of Positive Symptoms in First-episode Schizophrenia Patients

Juan A. Gallego, Delbert G. Robinson, Majnu John, Perihan E. Guvencek-Cokol, Jessica L. Greenberg, John Kane

18. ALKS 5461, a Novel Opioid Receptor Modulator, Reduces the Subjective Effects of Cocaine and was Safe and Well Tolerated during Concurrent Cocaine Administration

Ryan Turncliff, Bernard Silverman, Yangchun Du, Bradley Vince, Edward Sellers, Nancy Chen, Megan Shram, Mark Todtenkopf, Elliot W. Ehrich

19. The Failed CNS Studies Phenomenon: Is the Final Factor Looking Us Right in the Eye?

Charles S. Wilcox, Judy L. Morrissey, Nader Oskooilar, Mellissa M. Henry, Daniel E. Grosz, My-Linh Tong, Don F. De Francisco

20. GLYX-13, an NMDA Receptor Functional Partial Agonist, Reduced Depression Scores without Psychotomimetic Effects in Subjects with Major Depressive Disorder who had Failed Another Antidepressant Agent During the Current Episode

Sheldon Preskorn, Matthew Macaluso, Benji Kurian, Raymond Manning, Vishaal Mehra, Gary Zammit, Joseph Moskal, Jeffrey Burgdorf, Ronald M. Burch

21. Attrition and Retention among African Americans in a Pharmacological Treatment Study of Depression: Insights from the STAR*D Study

Eleanor Murphy, Layla Kassem, A. John Rush, Gonzalo Laje, Francis J. McMahon

Poster Session III—Wednesday

22. Assessment of Safety, Cardiovascular and Subjective Effects after Intravenous Cocaine and Lofexidine

Richard De La Garza, Gantt Galloway, Thomas Newton, John Mendelson, Colin Haile, Ekaterina Dib, Rollin Hawkins, Chwen-Yuen Chen, James Joseph Mahoney, Jurij Mojsiak, Guifang Lao, Ann Anderson, Roberta Kahn

23. In a 2-Year Placebo-controlled Randomized Trial, Galantamine-treated Patients had Lower Mortality Rates and Slower Decline in Cognition and Activities of Daily Living

Klaus Hager, Alan S. Baseman, John H. Han, Mary Sano, Henry M. Richards

24. Keeping it Real: Dissemination of Clinical Trial Results Using Clinician-friendly Effect Size Measures

Leslie Citrome

25. GIRK Channel Inhibitors Decreased Preference in Mice and Relapse Risk Scores in Alcoholics

Nagisa Sugaya, Yasukazu Ogai, Yuzo Aikawa, Mitsuru Umeno, Yosuke Yumoto, Mihoko Takahama, Miho Tanaka, Yoichi Kakibuchi, Eiichi Senoo, Yukio Takamatsu, Hideko Yamamoto, Yoko Hagino, Athina Markou, Kazutaka Ikeda

26. A Randomized Controlled Trial of Cognitive-behavioral Therapy Versus Risperidone for Augmenting Serotonin Reuptake Inhibitors in Obsessive-Compulsive Disorder

Helen Blair Simpson, Edna Foa

Poster Session III—Wednesday

27. Empirical Development of Depression Subtypes Using Factor Analysis of Subject Self-reports Across Symptom Domains

Marisa Toups, Guanghua Xiao, Yang Xie, Thomas Carmody, Benji Kurian, Madhukar Trivedi

28. Varenicline Attenuates Methamphetamine-induced Subjective Effects in Methamphetamine-dependent Volunteers

Christopher D. Verrico, James Joseph Mahoney, Ryan S. Bennett, Thomas F. Newton, Richard De La Garza

29. A Double-blind, Placebo-controlled, Multicenter trial of Adjunctive Armodafinil in Adults with Major Depression Associated with Bipolar I Disorder

Joseph R. Calabrese, Mark A. Frye, Ronghua Yang, Terence A. Ketter

30. Efficacy and Safety of Lisdexamfetamine Dimesylate in Treatment of Adults with Binge Eating Disorder: A Randomized, Double-blind, Placebo-controlled Trial

Susan McElroy, James Mitchell, Denise Wilfley, Maria Gasior, M. Celeste Ferreira-Cornwell, Joseph Gao, Jiannong Wang, Strakowski Stephen, James Hudson

31. Kynurenic Acid Correlates with Neopterin in Hepatitis C Virus Patients: Implications for the Efficacy and Psychological Side-effects of Interferon-Alpha Treatment

Waldemar Turski, Wojciech Zgrajka, Gregory F. Oxenkrug

32. Functional Connectivity Following GABAergic Challenge: Neural Correlates of Sedation and Intoxication

Stephanie Licata, Lisa Nickerson, Amy Janes, Steven Lowen, George Trksak, Scott Lukas

Poster Session III—Wednesday

33. Genetic Risk of Suicidal Behavior in Bipolar-spectrum Disorder: Analysis of 737 Pedigrees

Mirko Manchia, Tomas Hajek, Claire O'Donovan, Valeria Deiana, Caterina Chillotti, Martina Ruzickova, Maria Del Zompo, Martin Alda

34. Effect of Chronic Haloperidol on Glutamate Metabolism in Rat Cortex

Glenn Konopaske, Alo Basu, Joseph Coyle

35. Synaptic and Mitochondrial Changes in the Anterior Cingulate Cortex in Schizophrenia

Keri A. Barksdale, Joy Roche, Rosalinda C. Roberts, Adrienne C. Lahti

36. Persistently Increased Danger Signaling in the Adult Prefrontal Cortex following Adolescent Intermittent Ethanol is Associated with Reversal Learning Deficits

Ryan P. Vetreno, Liya Qin, Fulton T. Crews

37. Association of Anticipatory Connectivity in Insula with the Clinical Course of Major Depression: A Follow-up fMRI Study

Irina A. Strigo, Elena Kosheleva, Alan A. Simmons

38. Exercise Reduces Cocaine Abuse and Enhances Pharmacotherapy: Sex Differences

Marilyn E. Carroll, Natalie E. Zlebnik, Amy T. Saykao

39. In Vivo Binding of the Dopamine-1 Receptor PET tracers [11C] NNC112 and [11C]SCH23390: A Comparison Study in Individuals with Schizophrenia

Ragy R. Girgis, Eline Poels, Mark Slifstein, Anissa Abi-Dargham

Poster Session III—Wednesday

40. Aripiprazole Once-monthly for the Treatment of Schizophrenia: A Double-blind, Randomized, Non-inferiority Study vs. Oral Aripiprazole

W. Wolfgang Fleischhacker, Raymond Sanchez, Pamela P. Perry, Na Jin, Timothy Peters-Strickland, Brian R. Johnson, Ross A. Baker, Anna Eramo, Robert D. McQuade, William H. Carson, John M. Kane

41. Inflammatory Genes Expression as Biomarkers for Personalised Treatment in Psychiatry

Annamaria Cattaneo, Christoph Anacker, Nilay Hepgul, Mark Horowitz, Valeria Mondelli, Ksenia Musaelyan, Patricia Zunszain, Carmine M. Pariante

42. “Nonlinear Techniques as an Approach to Understand Mood Regulation in Bipolar Disorder”

Abigail Ortiz, Kamil Bradler, Julie Garnham, Claire Slaney, Martin Alda

43. A Re-analysis Using a Population-Enrichment Strategy of a Double-blind, Placebo-controlled Study of Chromium Picolinate in Atypical Depression

Maurizio Fava, Roberto Gomeni, James Komorowski, John Docherty, Cristina Cusin, David Soskin, David Mischoulon

44. Multi-site Validation of Touch Screen-based Translational Assays of Cognition and their Pharmacological Sensitivity

Rouba Kozak, Linda Mulryan, Brian J. Eastwood, Douglas S. Chapin, Theresa Ballard, Gary Gilmour, Francois Gastambide, Sophie Dix, Sanna K. Janhunen, Niels Plath, John Talpos, Laetitia Fellini, Linda Lerdrup, Sophie Billa, Iida Ahokas, Lisa M. Saksida, Eric G. Mohler, Lynne E. Rueter, Nadia Malik, Roger Wyler

Poster Session III—Wednesday

45. Targeting Cortical Oscillations with Non-invasive Brain Stimulation in Computer Simulations and Humans
Kristin Sellers, Mohsin Ali, Michael Boyle, Bradley Vaughn, John Gilmore, Flavio Frohlich
46. Mapping Brain Metabolic Connectivity in Awake Rats with MicroPET and Optogenetic Stimulation
Panayotis Thanos, Lisa Robison, Eric Nestler, Ronald Kim, Mike Michaelides, Mary Kay Lobo, Nora D. Volkow
47. A Brief Monetary Progressive Ratio Task Predicts Clinical Amotivation and Ventral Striatum Activation in Schizophrenia
Jacob Kantrowitz, Natalie Katchmar, Theodore Satterthwaite, Lillie Vandekar, Ruben Gur, Raquel Gur, Daniel Wolf
48. The Relationship between Early Life Stress and Brain Volume in Treatment-seeking Alcoholics
Laura Kwako, Melanie Schwandt, Victoria Brown, Erica N. Grodin, Markus Heilig, Daniel W. Hommer, Vijay Ramchandani
49. Chronic Naltrexone Modulates Marijuana's Reinforcing, Subjective and Cardiovascular Effects
Margaret Haney, Gillinder Bedi, Ziva Cooper
50. Increased Hippocampal Glutamate Correlates with Hippocampal Volumetric Deficits in Unmedicated Patients with Schizophrenia
Nina V. Kraguljac, David M. White, Meredith A. Reid, Jan den Hollander, Adrienne C. Lahti

Poster Session III—Wednesday

51. Altered Cortical Activation during Episodic Memory Encoding and Recognition in First-episode Psychosis

Michael M. Francis, Brenna C. McDonald, John D. West, Nikki Mehdiyoun, Tom A. Hummer, Jenifer L. Vohs, Emily Liffick, Andrew J. Saykin, Alan Breier

52. Transitive Inference in Relatives of Schizophrenia Patients

Obiora E. Onwuameze, Beng Ho

53. Regionally Specific Theta, Alpha, and Gamma Resting State Abnormalities in Schizophrenia

Yu-Han Chen, Breannan Howell, J. Christopher Edgar, Mingxiong Huang, Michael Hunter, Emerson Epstein, José Cañive

54. Association between the Change in Electrodermal Activity after Acute Tryptophan Depletion and Mood, Aggression and Venturesomeness in Young People with Attention Deficit Hyperactivity Disorder

Georg von Polier, Caroline Biskup, Wiebke F. Kötting, Sarah Bubenzer, Katrin Helmbold, Albrecht Eisert, Tilman J. Gaber, Florian Daniel Zepf

55. Longer-term Efficacy and Safety of Olanzapine and Fluoxetine Combination Versus Fluoxetine Monotherapy Following Successful Combination Therapy of Treatment-resistant Depression

Mauricio Tohen, Elizabeth Brunner, Olawale Osuntokun, John Landry, Rebecca Schroer, Michael Thase

56. Uridine Reduces Symptoms in Adolescent Bipolar Depression: A Phosphorus-31 Magnetic Resonance Spectroscopy Study

Douglas G. Kondo, Xian-Feng Shi, Young-Hoon Sung, Tracy L. Hellem, Rebekah S. Huber, Bethany R. Nickerson, Lauren N. Forrest, Perry F. Renshaw

Poster Session III—Wednesday

57. A Randomized Trial Administering Aspirin, Minocycline or Pramipexole vs Placebo as Add-on to Antipsychotics in Patients with Schizophrenia or Schizoaffective Disorder

Mark Weiser, Shimon Burshtein, Liliana Fodoreanu, Roxana Chiriță, Ghiorghe Talău, Diana Cirjaliu, John M. Davis, Michael Davidson

58. The Value of Risk Reduction in CNS Drug Development: Use of Net Present Value as One Model

Walter Greenblatt, Craig H. Mallinckrodt, Janet B.W. Williams, Danielle Popp, John Kane, Michael J. Detke

59. Personality Traits and Treatment of Major Depressive Episodes Associated with Bipolar I Disorder

Gary S. Sachs, Cynthia Siu, Josephine Cucchiaro, Robert Silva, Fred Grossman, Jay Hsu, Amir Kalali, Antony Loebel

60. Do Short-term Effects of Cholinesterase Inhibitors Predict Long-term Outcomes?

Bonnie Davis, Mary Sano

61. Efficacy of Adjunctive Quetiapine SR in a Randomized, Double-blind, Placebo-controlled Study of Mixed States (MS) in Bipolar Disorder (BD)

Vivek Singh, Charles Bowden

62. Feasibility of Centralized Ratings for Mental Health Safety Screening in a Dermatology Trial

Janet B.W. Williams, Daniel Davis, Danielle Popp, Jason Gross, Donna Salvucci, Michael J. Detke

Poster Session III—Wednesday

63. Imaging Cognition Circuits for Treatment Prediction in Major Depressive Disorder: Results from the International Study to Predict Optimized Treatment in Depression (iSPOT-D)

Anett Gyurak, Mayuresh Korgaonkar, Stuart Grieve, Leanne Williams, Amit Etkin

64. Six-month Depot Naltrexone Treatment Reduces Relapse in Parolees Formerly Addicted to Opioids

James Cornish, Courtney D. Nordeck, Daniel D. Langleben, Donna Coviello, Kevin Lynch, Tamara Boney, Charles P. O'Brien

65. Non-steroidal Anti-inflammatory Drug Use is Associated with Lower Remission Rate with Escitalopram but not with Other Antidepressants

Madhukar Trivedi, Marisa Toups, Thomas Carmody, Benji Kurian, Jennifer Warner-Schmidt, Kimberly E. Vanover, A. John Rush, Paul Greengard

66. Sensory-specific Satiety in Schizophrenia

James A. Waltz, Jaime K. Brown, Thomas J. Ross, Betty J. Salmeron, James M. Gold, Elliot A. Stein

67. Reward Circuitry Function in Euthymic Adults with a History of Major Depressive Disorder

Gabriel S. Dichter, Crystal Schiller, Moria Smoski

68. Oxytocin Influences Response Bias in Men but not Women in a Signal Detection Emotion Perception Task

Naomi M. Simon, Spencer Lynn, Elizabeth Hoge, Laura Fischer, Lisa Feldman Barrett

Poster Session III—Wednesday

69. Deficits in Functional Capacity and Impaired Glycemic Control: Urban African Americans with Type 2 Diabetes

Dominique L. Musselman, Marcia McNutt, David Ziemer, Erica Royster, Philip Harvey

70. Impulsivity in Relation to Emotional Stress in Borderline Personality Disorder

Sylvia Cackowski, Annegret Krause-Utz, Anne-Christine Reitz, Christian Schmahl

71. Neural Mechanisms Associated with Switching Attention from an Internal to an External Focus

Emily R. Stern, Alexandra F. Muratore, Stephan F. Taylor, James L. Abelson, Patrick R. Hof, Wayne K. Goodman

72. Enhancing Self Control of Smoking with Real Time fMRI

Luke Stoeckel, Satra Ghosh, Anisha Keshavan, Julia Stern, Susan Whitfield-Gabrieli, John DE. Gabrieli, A. Eden Evins

73. Lifetime Comorbidity of Obsessive-Compulsive Disorder and Sub-threshold Obsessive-Compulsive Symptomatology in the Community: Impact, Prevalence, Socio-demographic and Clinical Characteristics

Naomi A. Fineberg, Michael Hengartner, Carmel Bergbaum, Tim M. Gale, Kiri Jefferies, Wulf Rössler, Jules Angst

74. Attention and Positive Affect in Bipolar Disorder: An fMRI Study

Thilo Deckersbach, Andrew Corse, Elizabeth Greiter, Amanda Arulpragasam, Navneet Kaur, Tina Chou, Scott Rauch, Andrew A. Nierenberg, Darin Dougherty

Poster Session III—Wednesday

75. Differences in Neural Response to Extinction Recall in Young Adults Characterized as Behaviorally Inhibited/noninhibited during Early Childhood

Tomer Shechner, Nathan Fox, Jaime Mash, Carolyn Spiro, Melanie Hong, Ellen Leibenluft, Daniel S. Pine, Britton Jenifer

76. The Neurosteroids Allopregnanolone and DHEA Enhance Emotion Regulation Neurocircuits and Modulate Memory for Emotional Stimuli

Rebecca K. Sripada, Christine E. Marx, Anthony P. King, Sarah N. Garfinkel, James L. Abelson, Israel Liberzon

77. The Nicotinic Partial Agonist Varenicline Attenuates Visuospatial Working Memory Deficits in Schizophrenia Induced by Cigarette Smoking Abstinence

Victoria C. Wing, Caroline E. Wass, Tony P. George

78. Decision Making and Psychological Pain in Acutely Suicidal Depressed Patients

Ricardo Caceda, Philip Harvey, Dante Durand, Edmi Cortes, Stefania Prendes, Justyna Wojas, Charles Nemeroff

79. The Effects of Cannabis Abstinence on Neurocognitive and Clinical Symptoms in Cannabis Dependent Individuals with and without Schizophrenia

Rachel A. Rabin, Christiana Stefan, Konstantine K. Zakzanis, Tony P. George

80. Conflict Monitoring and Adaptation in Individuals at Familial Risk for Developing Bipolar Disorder

Luis R. Patino Duran, Caleb M. Adler, Neil Mills, Strakowski Stephen, David Fleck, Jeffrey Welge, Melissa DelBello

Poster Session III—Wednesday

81. Theta Power Modulation of Selective Encoding in Schizophrenia
J Daniel Ragland, Joshua Phillips, Michael Minzenberg, Tyler A. Lesh, Tara Niendam, Marjorie Solomon, Jong Yoon, Cameron Carter, Charan Ranganath
82. Differential Roles of Basic Visual Information in Fear and Happiness Perception in Schizophrenia
Yue Chen
83. Parents of Low Socioeconomic Status: Brain Function and Structure is Affected by Perceived Social Status and Early Life Experience
James E. Swain, S. S. Ho, Gary W. Evans, Xin Wang, Robert Varney, Israel Liberzon
84. Olanzapine, but not Fluoxetine, Treatment Increases Survival in Activity-based Anorexia in Mice
Stephanie C. Dulawa, Stephanie Klenotich, Mariel Seiglie, Matthew McMurray, Jamie Roitman, Daniel Le Grange, Priya Dugad
85. Antipsychotic use, Cardiometabolic health and Care Coordination: Implementation of Metabolic Monitoring Guidelines in a Large Outpatient Psychiatry Clinic
Jayesh Kamath, Ksenia Nawrocki, Rebecca Andrews
86. Brain GABA in Schizophrenia and Bipolar Disorders
Juan Bustillo, Hongji Chen, Thomas Jones, Nicholas Lemke, Chris Abbott
87. A Brain-selective Prodrug of 17 β -Estradiol in the Male Mouse: Implications for the use of Estrogen in Men who Suffer from Mood and Anxiety Disorders
Michal Arad, Sean C. Piantadosi, Katalin Tatrai-Prokai, Istvan Merchenthaler, Laszlo Prokai, Todd D. Gould

Poster Session III—Wednesday

88. Haste or Speed? Balancing Speed and Accuracy during a Motivated Reaction Time Task in Remitted and Depressed Patients with Bipolar Disorder

Henry W. Chase, Haris Aslam, Jorge Almeida, Mary L. Phillips

89. An Improved Framework for Confound Regression and Filtering for Control of Motion Artifact in the Preprocessing of Resting-State Functional Connectivity Data

Theodore D. Satterthwaite, Mark Elliott, Raphael Gerraty, Kosha Ruparel, James Loughhead, Monica Calkins, Simon Eickhoff, Hakon Hakonarson, Ruben Gur, Raquel Gur, Daniel Wolf

90. Effects of Yoga on Cognition, Psychiatric Symptoms, Weight and Biochemical Changes in Chronic Schizophrenic Patients

Robert C. Smith, Merlyn Mathew, Lawrence Maayan, Patricia L. Gerbarg, Richard Brown, Elizabeth Visceglia, Henry Sershen, Abel Lajtha, Sylvia Boules, James Auta, Alessandro Guidotti, John M. Davis

91. Depression-associated Risk Variant in GRM7 Predicts Vulnerability in Offspring at Risk for Major Depression and is Associated with Cortical Thickness Patterns

Guia Guffanti, Priya Wikramaratne, Ardesheer Talati, Ravi Bansal, Susan E. Hodge, Carolyn Erdman, Virginia Warner, Philips Adams, Marc Gameroff, Zagaa Odgerel, Bradley S. Peterson, Myrna M. Weissman, Steven P. Hamilton

92. Assessments of Everyday Functioning in Schizophrenia

Samir Sabbag, Felicia Gould, Dante Durand, Philip Harvey

Poster Session III—Wednesday

93. Genetic Association between Dopamine Signaling Molecules and Striatal Presynaptic Dopamine
Daniel P. Eisenberg, Joseph C. Masdeu, Philip D. Kohn, Bhaskar S. Kolachana, Daniel R. Weinberger, Karen F. Berman
94. Correlation of Cortisol but not BDNF Levels with Hippocampal Activation in Depressed Patients
Shigeru Toki, Yasumasa Okamoto, Masahiro Takamura, Tomoya Matsumoto, Shinpei Yoshimura, Tetsuya Yamamoto, Shigeto Yamawaki
95. Electroconvulsive Therapy Response in Major Depressive Disorder: A Pilot Functional Network Connectivity Resting State fMRI Investigation
Chris Abbott, Nicholas Lemke, Shruti Gopal, Robert Thoma, Juan Bustillo, Vincent Calhoun, Jessica Turner
96. Methylome Sequencing in the Alcohol Post-dependent Rat Model
Estelle Barbier, Jenica Tapocick, Nathan Jurgens, Jesse R. Schank, Kornel Schuebel, Zhifeng Zhou, Qiaoping Yuan, David Goldman, Markus Heilig
97. Effects of Pharmacogenetic Manipulation of the Nucleus Accumbens on Neuronal Activity and Alcohol Seeking Behaviors
Angela R. Ozburn, Joseph Kern, Kush Purohit, Colleen A. McClung
98. Anhedonia and Neural Response to Social Reward in Adolescents
Erika E. Forbes, Kati Healey, Thomas Olino, Erin Coffman

Poster Session III—Wednesday

99. Characterizing the Splicing Variants of ZNF804a in Human Brain

Ran Tao, Chao Li, Joo Heon Shin, Bin Xie, Yuan Gao, Tianzhang Ye, Andrew Jaffe, Barbara Lipska, Joel E. Kleinman, Daniel R. Weinberger, Thomas M. Hyde

100. Antidepressant Effects of Optogenetic Control of Nucleus Accumbens Neurons

T. Chase Francis, Jeffrey D. Lenz, Eric Finkel, Dipesh Chaudhury, Ming-Hu Han, Mary Kay Lobo

101. Ketamine and Neurocognition in Depression: The Modulating Effects of Lamotrigine

James W. Murrough, Le-Ben Wan, Benjamin Glicksberg, Katherine A. Collins, Sanjay J. Mathew, Dennis S. Charney, Dan V. Iosifescu, Katherine E. Burdick

102. A New Animal Model of the Pathophysiology of Tourette Syndrome: Parallel Characterization of Humans and Mice with a Disruption of the Histidine Decarboxylase (Hdc) Gene

Lissandra Baldan, Kyle Williams, Jean-Dominique Gallezot, Michael Crowley, Vladimir Pogorelov, Roxanne Gorczyca, George Anderson, Bennett Leventhal, Hiroshi Ohtsu, Zoe A. Hughes, John H. Krystal, Linda Mayes, Ivan de Araujo, Yu-Shin Ding, Matthew State, Christopher Pittenger

103. The Effects of Heterozygous Knockout of the Vesicular Monoamine Transporter (VMAT2) and Transgenic Overexpression of Alpha-Synuclein (SYN) on Locomotor Behavior in Mice

Frank S. Hall, Maria Perona, Charles Tsouvalas, Michael Baumann, Eliezer Masliah, George Uhl

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104. Adult Hippocampal Neurogenesis Modulates Excitability of the Dentate Gyrus

Amar Sahay, Taruna Ikrar, Sally Levinson, Antoine Besnard, Alexis Hill, Rene Hen, Xiangmin Xu

105. Neuronal Signatures of Self-control in Anterior Cingulate Cortex

Benjamin Hayden, Tommy Q. Blanchard

106. GLYX-13, an NMDA Receptor Glycine-site Functional Partial Agonist, Induces Rapid Antidepressant-like Effects without Ketamine-like Side Effects

Jeffrey Burgdorf, Xiao-lei Zhang, Katherine Nicholson, Robert Balster, J. David Leander, Patric Stanton, Roger Kroes, Joseph Moskal

107. Chronic Unpredictable Stress Dysregulates Glutamate Neurotransmission, Neuronal Plasticity and Cognitive Flexibility in the Rat Medial Prefrontal Cortex

Julianne Jett, Brian Bingham, David A. Morilak

108. Cognitive Features after Long-term Breast Cancer Survival: Longitudinal Evaluation of Neuropsychological Function into Later Life

Susan Schultz, Christopher M. Nguyen, Natalie L. Denburg, Torricia H. Yamada, Leigh Beglinger

109. Nonconscious Color Priming Reveals Intact Feedforward Visual Processing in Schizophrenia

Jonathan K. Wynn, Carol Jahshan, Bruno Breitmeyer, Michael F. Green

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110. Neuropsychological Performance in Phenotypes of Pediatric Bipolar Disorder

Daniel P. Dickstein, David A. Axelson, Shirley Yen, Jeff Hunt, Heather Hower, Mary Kay Gill, Lauren Weinstock, Tina Goldstein, Benjamin Goldstein, Neal Ryan, michael strober, Boris Birmaher, Martin Keller

111. Impaired Reward Responsiveness during Nicotine withdrawal in Rats and Humans Assessed in a Translational Behavioral Procedure

Andre Der-Avakian, Michele L. Pergadia, Manoranjan S. D'Souza, Pamela AF. Madden, Andrew C. Heath, Saul Shiffman, Diego A. Pizzagalli, Athina Markou

112. Loss Aversion with Respect to Prospect Theory and Relative Preference Theory

Sang Lee, Myoung Joo Lee, Jodi M. Gilman, Byoungwoo Kim, John Kuster, Anne Blood, Hans C. Breiter

113. Probabilistic Reinforcement Learning in Young Adults with Autism Spectrum Disorders Reflects Cognitive Control Deficits

Marjorie Solomon, Michael J. Frank, Anne Smith, J Daniel Ragland, Jong Yoon, Tara Niendam, Tyler A. Lesh, Cameron Carter

114. Respective Contributions of Alcoholism and Bipolar Disorder to Neurocognitive Deficits in Adults with Co-occurring Bipolar Disorder and Alcohol Dependence: Impact of Abstinence

Bryan K. Tolliver, James J. Prisciandaro, Delisa G. Brown, Kathleen T. Brady

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115. Does Targeted Social Cognitive Training Enhance Cortical and Subcortical Activation in Schizophrenia during Reward Processing?

Sophia Vinogradov, Karuna Subramaniam, Srikantan Nagarajan,
Christine Hooker

116. Catechol-O-Methyltransferase (COMT) Genotype as a Predictor of Response to Computerized Cognitive Remediation in Schizophrenia and Schizoaffective Disorders

Jean-Pierre Lindenmayer, Herbert Lachman, Susan McGurk,
Saurabh Kaushik, Anzalee Khan-Rhodes

117. Multimodal Neuroimaging of Networks Associated with Working Memory Performance

Katherine H. Karlsgodt, Gerhard Helleman, Jeanette Mumford,
Eliza Congdon, Catherine A. Sugar, Angelica Bato, Fred Sabb,
Edythe London, Russell Poldrack, Robert M. Bilder, Tyrone Cannon

118. Low Frequency Neural Oscillations Associated with Age and Processing Speed in Early- and Adult-onset Schizophrenia

Peter Bachman, Zachary D. Moran, Maria Jalbrzikowski, David C.
Glahn, Tyrone Cannon, Carrie Bearden

119. Relationship between Anxiety and Vascular Function in Older Individuals with and without Atherosclerotic Vascular Disease

Ashley N. Stillman, David J. Moser, Jess Fiedorowicz, Heather M.
Robinson, Peggy C. Nopoulos, William G. Haynes

120. Expectation and Hedonics Activate Specific Neural Networks during Reward

Arielle D. Stanford, Sarah Lisanby, Dolores Malaspina, Yael M.
Cycowicz

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121. Inhibitory Control Deficits in Autism Spectrum Disorders (ASD)

Matthew W. Mosconi, Michael E. Ragozzino, Lauren Schmitt,
Edwin H. Cook, John Sweeney

122. Violation of Temporal Expectancy Triggers Lability of Amygdala-dependent Memories

Lorenzo Diaz-Mataix, Raquel Martinez, Glenn Schafe, Joseph LeDoux, Doyere Valerie

123. Fine-grained Working Memory Load Manipulation Reveals Absence of Normative Inverted-U Activation in Schizophrenia

Jared X. Van Snellenberg, Ragy R. Girgis, Christina Read, Judy L. Thompson, Jochen Weber, Tor D. Wager, Mark Slifstein, Jeffrey A. Lieberman, Anissa Abi-Dargham, Edward E. Smith

124. If I do it, it Must be Important: An Inhibition Deficit Model of Obsessive Compulsive Disorder

Gideon E. Anholt, Omer Linkovski, Eyal Kalanthroff, Avishai Henik

125. Selective Manipulation of Striatal Circuitry with Serotonin-6 Receptors: Pathway Specific Targeting of 5-HT₆ Receptor Overexpression has Opposing Effects on Behavioral Acquisition and Expression of Habits

Daniel Eskenazi, John F. Neumaier

126. A Novel Task Probing Neural Substrates of Approach-avoidance Conflict: Implications for Understanding Anxiety Disorders

Robin L. Aupperle, Sarah Sullivan, Andrew Melrose, Martin Paulus, Murray B. Stein

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127. Relationship between Plasma Peptide YY and Cardiometabolic Disease Risk Factors in a Cohort of Older Psychiatric Patients

Pei-an Betty Shih, Hua Jin, Sunder Mudaliar, Robert R. Henry, Dilip V. Jeste

128. The Influence of the Brain-derived Neurotrophic Factor Val66Met Genotype and HMG-CoA Reductase Inhibitors on Insulin Resistance in the Schizophrenia and Bipolar Populations

Kyle Burghardt, Rodica Pop-Busui, Michael Bly, Tyler Grove, Stephan Taylor, Vicki Ellingrod

129. Relationship between Reactivity to Heroin-related Cues, Craving, and Self-administration in Buprenorphine-maintained Heroin Abusers

Sandra D. Comer, Adam Bisaga, Jermaine Jones, Shanthi Mogali, Jeanne Manubay, Maria Sullivan, Suzanne Vosburg, Ziva Cooper, Perrine Roux, Jessica Fogel, Laura Shiffrin

130. Combined Effects of Antipsychotic Medication and Hypoglycemia upon Microglial Activation

Neil M. Richtand, Amanda Isom, Rebecca Ahlbrand, Gary A. Gudelsky

131. Substance use during Cannabis Withdrawal in People with Schizophrenia

Maju Koola, Douglas Boggs, Deanna L. Kelly, Fang Liu, Jared Linthicum, Hailey Turner, Robert P. McMahon, David Gorelick

132. Familial Internalizing and Externalizing Liability Factors Contribute to Borderline Personality Disorder

James Hudson, Mary Zanarini, Karen Mitchell, Lois Choi-Kain, Ming Tsuang, John Gunderson

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133. Depression Symptoms Associated with Cannabis Dependence in an Adolescent American Indian Community Sample

David Gilder, Cindy L. Ehlers

134. Mental Disorders in Adolescence and Young Adulthood: Homotypic and Heterotypic Longitudinal Associations

Katja Beesdo-Baum, Daniel S. Pine, Roselind Lieb, Hans-Ulrich Wittchen

135. Migraine Comorbidity Worsens Course of Illness in a Longitudinal Study of Bipolar Disorder

Erika FH. Saunders, Masoud Kamali, Alan J. Gelenberg, Melvin McInnis

136. Childhood Sexual and Emotional Abuse Related to Multiple Unfavorable Bipolar Disorder Illness Characteristics

Shefali Miller, Soumya Parameshwaran, Farnaz Hooshmand, Po Wang, Terence A. Ketter

137. PTSD is Associated with an Increased Prevalence of Autoimmune Disorders

Aoife O'Donovan, Beth Cohen, Daniel Bertenthal, Mary Margaretten, Karen Seal, Thomas Neylan

138. Metabolic Syndrome and Outcomes in Individuals with Bipolar II Disorder

Holly Swartz, Andrea Fagiolini, Paola Rucci, Ellen Frank, David J. Kupfer

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139. Juvenile Antioxidant Treatment Prevents Behavioral and Prefrontal Cortical Deficits in a Developmental Model of Schizophrenia

Danielle S. Counotte, Harry J. Cabungcal, Patrick Piantadosi, Elyse Sullivan, Eastman Lewis, Gwendolyn G. Calhoun, Hugo Tejada, Michel Cuenod, Kim Q. Do, Patricio O'Donnell

140. RNA Editing of an AMPA Receptor Subunit is Altered in Major Depression and Suicide

Monsheel Sodhi, Daniel Mount, Thomas M. Hyde, Joel E. Kleinman

141. Higher Insula Connectivity in Abstinent Stimulant Users

Jazmin Camchong, Sheila Specker, Valerie Slaymaker, Bryon Mueller, Angus MacDonald, Kelvin O. Lim

142. Schizophrenia Subtypes: Going, Going, Gone

David L. Braff, Anthony J. Rissling, Stacy Langton, William Carpenter

143. Seasonality and Diurnal Rhythms of Sleep and Activity assessed by Mobile Technologies in a Community Based Population Study

Adrienne D. Taylor, Femke Lamers, Jihui Zhang, Ruth Benca, Kathleen R. Merikangas

144. Pediatric Bipolar Disorder and Mixed Mood Symptoms: Moderately Prevalent in Community Mental Health and Underdiagnosed by Practitioners

Eric Youngstrom, Jennifer Youngstrom, Guillermo Perez Algorta, Robert L. Findling

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145. Clinical Characteristics of Visual Hallucinations in Psychotic Disorders:
A Cross-sectional Study

Virginie-Anne Chouinard, Dost Ongur, Guy Chouinard, Bruce M. Cohen

146. Risk-taking in Schizophrenia and Controls with and without Cannabis
Dependence

Bernard A. Fischer, Robert P. McMahon, Deanna L. Kelly, Heidi J. Wehring, Walter Meyer, Stephanie Feldman, William Carpenter, David Gorelick

147. The Influence of Social Setting on Acute Alcohol Effects in Humans

Matthew Kirkpatrick, Harriet de Wit

148. Anatomy of the “Neuropsychiatric Translational Research Revolution:”
Challenges Abound

David L. Braff, Lara Braff

149. Utilization of Patient-derived Neuronal Cells for Disease Modeling and
Hypothesis Building in Neuropsychiatric Disease Research

Shinichi Kano, Gustavo Maegawa, Zhifeng Zhou, Ross Cardarelli, Carlo Colantuoni, Qiaoping Yuan, Fang Han, Ashley Wilson, Nicola Cascella, Hongkai Ji, Patricio O’Donnell, David Goldman, Akira Sawa

150. Prepulse Inhibition Detects Evidence of Neurodevelopmental Differences
in Individuals at Greatest Risk for Psychosis: Findings from the North
American Prodromal Longitudinal Study (NAPLS)

Kristin Cadenhead, Jean Addington, Tyrone Cannon, Barbara Cornblatt, Daniel H. Mathalon, Thomas McGlashan, Diana Perkins, Larry Seidman, Ming Tsuang

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151. Relationships between Mismatch Negativity, Auditory Emotional Processing, and Psychosocial Functioning in Schizophrenia

Barbara Breteinstein, Wei-li Chang, Anthony J. Rissling, Richard F. Sharp, Catherine A. Sugar, Ricki-Leigh Malaguti, Marlena Pela, Joyce Sprock, David L. Braff, Gregory A. Light

152. Relationship between Auditory Processing and Affective Prosody in Schizophrenia

Carol Jahshan, Jonathan K. Wynn, Justina Avila, Michael F. Green

153. Diffusion Tensor Imaging of the Thalamus in Cocaine Dependent Subjects: Association with fMRI and Treatment Response

F. Gerard Moeller, Khader M. Hasan, Joel Steinberg, Liangsuo Ma, Joy M. Schmitz, Scott Lane, Ponnada A. Narayana

154. Tolcapone Modulates Working Memory Load Related DLPFC Activity and Coupling in Patients with Schizophrenia

Roberta Rasetti, Venkata S. Mattay, Christopher J. Li, Qiang Chen, Jesse Hochheiser, Joseph H. Callicott, Karen F. Berman, Jose A. Apud, Daniel R. Weinberger

155. Behavioral Economics for Standardized Metrics in CNS Drug Development and Regulation

Jack E. Henningfield, Steven R. Hursh, Peter G. Roma, Edward J. Cone, August R. Buchhalter, Reginald V. Fant, Sidney H. Scholl

156. The Effect of Stereotype Threat, Perceived Discrimination, and Examiner-examinee Racial Discordance: Simple as Black and White?

April D. Thames, Robert M. Bilder, Desiree A. Byrd, Charles H. Hinkin, Kimberley Duff

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157. microRNA-206 Regulates Alcohol Consumption and Preference

Jenica Tapocik, Meghan Flanigan, Matthew Solomon, Courtney King, Estelle Barbier, Jesse Schank, Molly Heyer, Paul Kenny, Markus Heilig

158. Developmental Differences in the Neural Correlates of Trial-to-trial Variance in Response Time

Nancy E. Adleman, Richard Reynolds, Gang Chen, Varun Razdan, Daniel S. Pine, Ellen Leibenluft

159. Differentiating Neural Networks with Interleaved TMS-BOLD Imaging: Insight into Addiction

Colleen A. Hanlon, Melanie Canterberry, Joseph Taylor, Truman Brown, Mark S. George

160. The Dopamine D1-D2 Receptor Heteromer Regulates Glycogen Synthase Kinase-3 β Activity Independent of Akt in Rodent Prefrontal Cortex: Potential Relevance to Cognitive Function

Melissa L. Perreault, Jace Jones-Tabah, Brian F. O'Dowd, Susan R. George

161. Interleaved TMS/fMRI after rTMS: How to Make the Prefrontal Cortex Sing?

Joseph Taylor, Colleen A. Hanlon, Xingbao Li, Mark S. George

162. Cognitive De-biasing Strategies Significantly Improve the Assessment of Pediatric Bipolar Disorder

Melissa Jenkins, Eric Youngstrom

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163. Economic Evaluation of Antipsychotic Treatment in Pediatric Disruptive Behavior Disorders: An Example from the Metabolic Effects of Antipsychotics in Children (MEAC) Study

Ginger E. Nicol, Josh Edler, Steven M. Kymes, John W. Newcomer

164. Preference for Future Gains and Losses in Patients with Major Depression is Modulated by the Presence of Comorbid Posttraumatic Stress Disorder

Boadie Dunlop, Britta Maciuba, Jan Engelmann

165. Effect of Meta-Chlorophenylpiperazine (mCPP) on Appetite and Satiety and on Emotional Processing and Mood in Healthy Volunteers

Colin T. Dourish, Jason M. Thomas

166. Current Treatment of Obsessive-Compulsive Disorder (OCD): A Cross-Sectional Analysis of OCD Treatment in 9 International Outpatient Settings

Michael Van Ameringen, William Simpson, Beth Patterson

167. Impact of Treatment Approach on Maternal and Neonatal Outcome in Pregnant Opioid-maintained Women

Verena Metz, Reinhold Jagsch, Nina Ebner, Johanna Würzl, Anna Pribasnig, Constantin Aschauer, Gabriele Fischer

168. Discontinuations Following a Switch from Risperidone, Olanzapine, or Aripiprazole to Iloperidone in Patients with Schizophrenia: The i-FANS Study

Leslie Citrome, Farid Kianifard, Xiangyi Meng, Adam Winseck, Marla Hochfeld, Stephen Stahl

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169. The Reinforcing and Subjective Effects of Intravenous and Intranasal Buprenorphine in Heroin Users

Jermaine Jones, Sandra D. Comer

170. MDMA Buffers against Cues of Social Rejection

Margaret C. Wardle, Charles G. Frye, Harriet de Wit

171. Neuroprotective Effects of Long-term Lithium Treatment on Amyloid Deposition in Older Adults with Bipolar Disorder

Ariel Gildengers, Julie Price, Butters Meryl, James Becker, Tamer Ibrahim, William Klunk, Charles Reynolds

172. Differences in the Magnitude of BOLD Response to Implicit Emotional Faces Predict Antidepressant Response to Scopolamine in Major Depressive Disorder

Maura L. Furey, Wayne C. Drevets, Ashish Khanna, Carlos A. Zarate

173. ALKS 5461, a Novel Opioid Receptor Modulator, Normalizes Human EEG Responses in an Auditory Oddball Task after Cocaine Administration as Indicated by a Brain Network Activation Analysis

Edward Sellers, Ryan Turncliff, Amit Reches, Dalia Dickman, I Laufer, Keren Ziv, Y Stern, Z Halabi, Bradley Vince

174. Pharmacokinetic Modeling of ALKS 9070 (ALKS 9072), a Novel Once-monthly Prodrug of Aripiprazole

Ryan Turncliff, Wen-i Li, Helen Pentikis

175. Long Acting Injectable vs. Oral Antipsychotics in Schizophrenia: A Systematic Review and Meta-analysis of Mirror-image Studies

Taishiro Kishimoto, Masahiro Nitta, Michael Borenstein, John Kane, Christoph U. Correll

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176. Pharmacokinetic Profile of ITI-007, A Novel Approach for the Treatment of Schizophrenia and Other Psychiatric and Neurological Disorders

Kimberly E. Vanover, Robert E. Davis, Lawrence P. Wennogle, Paul Greengard, Sharon Mates

177. Nicotine Metabolism: Sex Differences in African American and Caucasian Smokers

Andrea King, Lingjiao Zhang, Daniel Roche, Dingcai Cao, Rachel Tyndale

178. Impact on Hospitalization after Initiating Long-acting Injectable Antipsychotics for a Longer vs. Shorter Time Period among Medicaid Insured Patients with Schizophrenia

Rimal Bera, Craig Karson, Steve Offord, Donna Zubek, Gina Lau, Jay Lin

179. Healthcare Impact of Initiating Long-acting Injectable Antipsychotic Therapy among Medicaid Insured Patients with Schizophrenia

Craig Karson, Rimal Bera, Steve Offord, Donna Zubek, Gina Lau, Jay Lin

180. Do Benzodiazepines Really Induce Negative Cognitive Effects in Elderly Psychiatric Patients?

Lars H. Tanum, Gudrun Hoiseth, Marit Tveito, Kari Kristiansen, Kristin Kvande, Bernhard Lorentzen, Helge Refsum, Jorgen Bramness

181. The Efficacy of the Glutamate NMDA Receptor Antagonist Memantine For Cognitive Deficits In Euthymic Subjects with Bipolar Disorder

Dan V. Iosifescu, William S. Gilmer, Alexander Fan, Atilla Gonenc, Constance M. Moore, Christopher Randolph, Mark Hyman, Rapaport, Thilo Deckersbach, Andrew A. Nierenberg

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182. Hydrocortisone and Intrusive Memories in Posttraumatic Stress Disorder

Petra Ludäscher, Christian Schmahl, Martin Bohus

183. Lurasidone Monotherapy for the Treatment of Bipolar Depression: Results of the 6-week, Double-blind, Placebo-controlled PREVAIL-2 Study

Antony Loebel, Josephine Cucchiaro, Robert Silva, Kaushik Sarma, Hans Kroger, Jay Hsu, Joseph R. Calabrese, Gary Sachs

184. Effect of Lurasidone Monotherapy or Adjunctive Therapy on Anxiety Symptoms in Patients with Bipolar I Depression

Robert M A. Hirschfeld, Josephine Cucchiaro, Andrei Pikalov, Peter Warner, Jay Hsu, Hans Kroger, Antony Loebel

185. Evaluation of Glycine Transporter Inhibitor Org 25935 for the Prevention of Relapse in Alcohol-dependent Patients: A Multisite, Randomized, Double-blind, Placebo-controlled Trial

Armin Szegedi, Andrea deBejczy, Kari Nations, Frank Ruwe, Bo Söderpalm, David Michelson

186. Metabolism and Pharmacokinetics of the Antidepressant Amitifadine in Humans

Randall D. Marshall, Jurgen Venitz, Pierre Tran, David T. Wong, Franklin P. Bymaster

187. The Tryptophan-Kynurenine Pathway in Major Depression: Neuroprotective Effects of Treatment

Angelos Halaris, Aye-Mu Myint, Edwin Meresh, Vidushi Savant, Gilles Guillemin, James Sinacore

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188. Insulin Resistance as a Shared Pathophysiology between Mood and Cardiometabolic Disorders: Relevance of PPAR- γ Agonism

David Kemp, Keming Gao, Timothy Warneka, Carla Conroy, Stephen Ganocy, Faramarz Ismail-Beigi, Joseph R. Calabrese

189. The Space of Common Psychiatric Disorders

Carlos Blanco, Robert Krueger, Deborah Hasin, Shuai Wang, Mark Olfson

190. Levomilnacipran SR 40 mg and 80 mg in Major Depressive Disorder: A Phase III, Randomized, Double-blind, Fixed-dose, Placebo-controlled Study

David Bakish, Carl Gommoll, Changzheng Chen, William M. Greenberg, Rene Nunez, Michael Liebowitz, Arif Khan

191. The Efficacy of Vilazodone on Anxiety Symptoms in Patients with Major Depressive Disorder: A Post Hoc Analysis of Two Randomized Controlled Trials

Michael Thase, John Edwards, Dalei Chen, Adam Ruth

192. Predictive Deficits Underlie Auditory Verbal Hallucinations in Schizophrenia: A Model-based fMRI Study

Guillermo Horga, Kelly Schatz, Anissa Abi-Dargham, Bradley S. Peterson

193. Effect of Paternal Age on Schizophrenia-related Endophenotypes: Examination of Data from the Consortium on the Genetics of Schizophrenia (COGS)

Allen David. Radant, Steve Millard, Debby Tsuang, Michelle Esterberg, David L. Braff, Neal R. Swerdlow, Michael F. Green, Keith H Nuechterlein, Larry Siever, Jeremy M Silverman, Bruce I Turetsky, Laura Lazzeroni, Monica Calkins, Larry Seidman, Ann Olincy

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194. Brain Structural Community Abnormalities Revealed using PLACE (Path Length Associated Community Estimation)

Alex D. Leow, Johnson Jonaris GadElkarim, Olusola Ajilore, Liang Zhan, Jamie Feusner, Teena Moody, Paul Thompson, Anand Kumar, Lori Altshuler

195. Testing Glutamatergic Strategies in Early Psychosis: Longitudinal Rodent Pharmacological MRI Studies and a Randomized, Double-blind, Placebo-controlled Proof-of-concept Trial in Patients at Clinical Risk for Psychosis

Scott A. Schobel, Nashid Chaudhury, Usman Khan, Martin Styner, Beatriz Paniagua, Cheryl M. Corcoran, Jeffrey A. Lieberman, Holly Moore, Scott A. Small

196. Transient Ziprasidone's Dopamine D2/3 Receptor Occupancy - A Clinical 24-hour [11C]- Raclopride PET Study

Ariel Graff, Takefumi Suzuki, Hiroyuki Uchida, Gary Remington, Fernando Caravaggio, Carol Borlido, Bruce Pollock, Benoit Mulsant, Vincenzo DeLuca, Zahinoor Ismail, David Mamo

197. Modafinil Modulates Activation and Functional Coupling of the Prefrontal Cortex During Working Memory in Patients with Schizophrenia

Jose A. Apud, Roberta Rasetti, Christopher Li, Qiang Chen, Xi Cheng, Jesse Hochheiser, Joseph H. Callicott, Karen F. Berman, Daniel R. Weinberger, Venkata Mattay

198. Neural Correlates of the Discrepancy between Verbal and Performance IQ

Amy Margolis, Ravi Bansal, XueJun Hao, Cole Erickson, Bradley S. Peterson

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199. Role of Hypocretin-Dynorphin Co-transmission in Motivated Behavior

John Muschamp, Jonathan Hollander, Jennifer Thompson, Sara Onvani, Linda Hassinger, Theodore Kamenecka, Stephanie Borgland, Paul Kenny, William Carlezon

200. Functional Capacity Assessment in Older Adult Populations

Sara J. Czaja, Philip D. Harvey, David Loewenstein

201. Differences in Amphetamine-evoked Dopamine Release in Cortex and Striatum

Hank P. Jedema, Rajesh Narendran, Charles W. Bradberry

202. Remission during 12 Months of Double-blind Treatment with Lurasidone vs. Quetiapine XR in Patients with Schizophrenia

Antony Loebel, Josephine Cucchiaro, Jane Xu, Jay Hsu, Kaushik Sarma, Peter Warner, Andrei Pikalov, John M. Kane

203. Antidepressant-like Effects of Buprenorphine and Kappa Receptor Antagonists in Rodent Behavioral Tests

Irwin Lucki, Gregory V. Carr, Bethany R. Brookshire, Danielle Bracco, Edgardo Falcon-Morales

204. Effect of L-Methylfolate on Core Symptoms of Major Depression from a Randomized Clinical Trial

George Papakostas, Richard C. Shelton, John Zajecka, Maurizio Fava

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205. BDNF Val66Met Modulates BOLD Response to Affective Instrumental Learning in Humans

Mbemba Jabbi, Brett Cropp, Tiffany Nash, Philip Kohn, Raghav Mattay, Shane Kippenhan, Bhaskar S. Kolachana, Daniel R. Weinberger, Karen F. Berman

206. Reduced Default Mode Functional Connectivity between Medial Prefrontal and Superior Temporal Gyrus Regions in Youths with Bipolar Disorder

Melissa Lopez-Larson, Deborah Yurgelun-Todd

207. Heritability and Linkage Analysis of Personality in Bipolar Disorder

Tiffany A. Greenwood, Judith Badner, William Byerley, BiGS Consortium

208. Effects of Aromatase Inhibition and Androgen Activity on Serotonin and Behavior in Male Macaques

Cynthia L. Bethea, Arubala L. Reddy, Nicola Robertson, Kristine Coleman

209. Multiple Obesity-related Genes are Associated with Antipsychotic-induced Weight Gain in Drug Naïve Pediatric Patients

Jianping Zhang, Todd Lencz, Christoph Correll, Anil Malhotra

210. Reduced Risk of Neurological Disorders in Patients Receiving Lithium Treatment

James M. Prosser, Monica L. Gilbert, Ronald R. Fieve

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211. Double-blind, Placebo-controlled Trial of Pramipexole Augmentation in Treatment-resistant Major Depressive Disorder

Maurizio Fava, Cristina Cusin, Andrew A. Nierenberg, Dan V. Iosifescu, Nadia Iovieno, A. John Rush, Roy Perlis

212. Experience Integrating Ketamine into the Healthcare System: Outpatient and Inpatient Treatment of Post-partum Depression, Suicidal Ideation and Comorbid Chronic Pain- Mood Disorders

Marlon Quinones, Nancy Diazgranados, Maxim Eckmann, Somayaji Ramamurthy, Charles Bowden, Pedro L. Delgado

213. Tissue Specific Effects of Psychological Stress on the Development of Acute Insulin Resistance

Li Li, Joseph Messina, Xiaohua Li

214. Serotonergic Modulation of Neuronal Excitability in the BNST: Effects of Chronic Alcohol

Catherine Marcinkiewicz, Nora McCall, Josh Jennings, Thomas Kash

215. A Six Month Randomized Controlled Trial of Long Acting Injectable Risperdone 50 and 100 mg in Treatment Resistant Schizophrenia

Herbert Y. Meltzer, Jean-Pierre Lindenmayer, Joseph Kwentus, D. Barrett Share, Rania M. Shebaro, Karu Jayathilake

216. A Clinical Laboratory Model of Allostasis of the Brain Reward System Diurnal Cortisol and Sleep in Recently Detoxified Opioid Dependent Patients, Normal Control Subjects & Patients Drug Free for 60-90 Days

Scott Bunce, Roger Meyer, Edward Bixler, Jonathan Harris

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217. Neuropeptide Y Signaling in the Bed Nucleus of the Stria Terminalis
Regulates Binge-like Alcohol Drinking

Thomas Kash, Kristen Pleil, Emily Lowery, Todd Thiele

218. Association between Microstructural Integrity of the Frontostriatal Tracts
and School Functions: Inattention Symptoms and Sustained Attention as
Mediators

Susan Shur-Fen Gau, Kathleen Merikangas, Wen-Yih Isaac Tseng



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- 1) Seibyl JP, Krystal JH, Charney DS. Dopamine and noradrenergic reuptake inhibitors in treatment of schizophrenia. Patent #:5,447,948. September 5, 1995
- 2) Vladimir, Coric; Krystal, John H.; Sanacora, Gerard – Glutamate Agents in the Treatment of Mental Disorders No. 11/399,188 April 5, 2006 (Pending)
- 3) Intranasal Administration of Ketamine to Treat Depression (pending)

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Program Committee Members with No Disclosures:

Ted Abel	Michael Davidson	Kerry Ressler
Victoria Arango	Jay Gingrich	Etienne Sibille
Aysenil Belger	Paul Kenny	Arielle Stanford
Karen Berman	Thomas Lehner	Daniel Weinberger
Randy Blakely	Arnold Mandell	Rachel Yehuda
Hilary Blumberg	Karoly Mirnics	
Kristin Cadenhead	Bitu Moghaddam	

ACNP 2012 Presenter Disclosures

Bryon Adinoff: *Part 1:* Auxilium Pharmaceuticals, Shook, Hardy & Bacon LLP (medical malpractice consultant, tobacco companies), *Part 2:* Department of Veterans Affairs, UT Southwestern Medical Center

Caleb Adler: *Part 1:* I have participated in multi-site trials including AstraZeneca, Eli Lilly, Pfizer, Otsuka, Forest, Sunovion, Novartis, Glaxo Smith-Kline, and Amylin. I have received research support from AstraZeneca. I have been a speaker and consultant for Merck., *Part 2:* Merck, *Part 3:* Merck, *Part 4:* AstraZeneca

Evdokia Anagnostou: *Part 1:* Consultation fee from SeaSide Therapeutics Consulted without fees to Novartis, *Part 4:* Forest has provided free drug and placebo for a clinical trial

Celso Arango: *Part 1:* Dr. Arango. has been a consultant to or has received honoraria or grants from Astra Zeneca, Bristol-Myers Squibb, Janssen Cilag, Lundbeck, Merck, Otsuka, Pfizer, Roche, Servier and Schering Plough., *Part 4:* Dr. Arango has received grants from Roche and Lundbeck.

Steven Arnold: *Part 1:* Bristol Myers Squibb [advisor board], *Part 4:* Dr. Arnold has received research support from the NIH (R01AG039478, R01AG15819, P30AG10161, P30AG10124, P50MH64045, R01MH075916, R01 DA023210, R01 DA023210, R01 DA025201), the Marian S. Ware Charitable Giving Fund, the Penn Pfizer Alliance, Johnson & Johnson, Neuronetrix, Eli Lilly, American College of Radiology Imaging Network.

David Baker: *Part 1:* Promentis Pharmaceuticals (2010-present), *Part 2:* Promentis Pharmaceuticals (2010-present), *Part 3:* Promentis Pharmaceuticals (2010-present), *Part 4:* Promentis Pharmaceuticals (2010-present)

Deanna Barch: *Part 1:* I have research grants from Novartis and Allon, and I served as a consultant for Pfizer., *Part 4:* Allon and Novartis

Ruth Benca: *Part 1:* Consultant to Merck and Sanofi-aventis.

John Blangero: *Part 4:* Eli Lilly and Co.

Floyd Bloom: *Part 1:* I am a Founder and Director of Alkermes,plc. and as such I am also a shareholder in this publicly traded company., *Part 2:* I am retired. My income sources are from general investments and 401k plans, and from two chairitable remainder trusts., *Part 3:* I am a consultant to Elan Pharma, Inc, as well as a Director of Alkermes,plc. Nither relationship nor any of my other smaller consulting agreements amounts to 5% of my personal income.,

Johannes Bohacek: *Part 4:* Roche (post-doctoral fellowship)

Michael Bogenschutz: *Part 2:* Salary from the University of New Mexico

Christopher Bowie: *Part 1:* I have been a consultant and advisory board member for Abbott Pharmaceuticals from 2010 to present.

Nicholas Brandon: *Part 1:* I am a full time employee of Pfizer, *Part 2:* I am a full time employee of Pfizer, *Part 3:* I am a full time employee of Pfizer

ACNP 2012 Presenter Disclosures (continued)

Edward Bullmore: *Part 1:* GlaxoSmithKline Employee 0.5 FTE, *Part 2:* Income and equity from GlaxoSmithKline, *Part 3:* GlaxoSmithKline Employee 0.5 FTE, *Part 5:* I work half-time for GlaxoSmithKline and half-time for the University of Cambridge

Blynn Bunney: *Part 1:* NeoSync

Ronald Burch: *Part 1:* I am an employee of Naurex, Inc, the Sponsor of the clinical study reported herein., *Part 2:* Naurex, Inc, *Part 3:* Naurex, Inc

Marc Caron: *Part 1:* Lundbeck, Omeros, Roche, Forest Laboratories., *Part 2:* Omeros, *Part 4:* Lundbeck, Roche, Forest Laboratories

William Carpenter: *Part 1:* I have served as a consultant to Lundbeck, Eli Lilly and Company, Bristol-Myers Squibb, Shire Pharmaceuticals, and AstraZeneca

Kiki Chang: *Part 1:* Dr. Chang is a consultant for GSK, Merck, BMS, and Lilly. He receives research funding from GSK and Merck., *Part 4:* GSK, Merck

Charles Chavkin: *Part 1:* Consulting for Trevena 2010-2011, *Part 4:* Contract with Trevena 2010-2011

Alon Chen: See insert

Guang Chen: *Part 1:* Janssen Pharmaceutical Companies of Johnson and Johnson, *Part 2:* Janssen Pharmaceutical Companies of Johnson and Johnson, *Part 3:* Janssen Pharmaceutical Companies of Johnson and Johnson, *Part 4:* Janssen Pharmaceutical Companies of Johnson and Johnson

David Collier: *Part 1:* David Collier will become an employee of Eli Lilly on April 23rd 2012.

Wilson Compton: *Part 1:* Pfizer, General Electric, *Part 2:* None. No income and equity is less than \$10,000 in total for both entities listed above (General Electric and Pfizer)., *Part 3:* None.

P. Jeffrey Conn: *Part 2:* Consulting fee and licensing income through Vanderbilt University from Karuna Pharmaceuticals Licensing income through Vanderbilt University from Johnson and Johnson, Seaside Therapeutics Own equity that does not generate income in Seaside Therapeutics, Karuna Pharmaceuticals, *Part 4:* Johnson and Johnson, Seaside Therapeutics

Edwin Cook: *Part 1:* Consultation - Seaside Therapeutics - 2010, *Part 4:* Seaside Therapeutics - support for site in a multi-site clinical trial

Christopher Correll: *Part 1:* Dr. Correll has been a consultant and/or advisor to or has received honoraria from: Actelion, Alexza; AstraZeneca, Biotis, Boehringer-Ingelheim, Bristol-Myers Squibb, Cephalon, Desitin, Eli Lilly, GSK, IntraCellular Therapies, Lundbeck, Medavante, Medicare, Medscape, Merck, National Institute of Mental Health, Novartis, Ortho-McNeill/Janssen/J&J, Otsuka, Pfizer, ProPhase, Schering-Plough, Sepracor/Sunovion, Supernus, Takeda, Teva and Vanda. He has received grant support from BMS, Feinstein Institute for Medical Research, Janssen/J&J, National Institute of Mental Health (NIMH), National Alliance for Research in Schizophrenia and Depression (NARSAD), and Otsuka., *Part 2:* AstraZeneca, Bristol-Myers Squibb, GSK, Otsuka, Pfizer, ProPhase., *Part 3:* AstraZeneca, Bristol-Myers Squibb, GSK, Otsuka, Pfizer, ProPhase., *Part 4:* BMS, Janssen/J&J, and Otsuka.

ACNP 2012 Presenter Disclosures (continued)

David Cotter: *Part 1:* honorarium research presentation x1 Lilly

Joseph Coyle: *Part 2:* Abbott Pharmaceutical, *Part 4:* BristolMeyerSquibb

John Csernansky: *Part 1:* Membership on a Data Monitoring Committee for Eli Lilly and Co.

Bruce Cuthbert: *Part 2:* NIMH Salary

Kenneth Davis: *Part 1:* My wife, Bonnie Morrison Davis, MD, is a patent holder on the use patent for galantamine for Alzheimer's disease and dementias that has been licensed to Janssen-Pharma, a subsidiary of Johnson & Johnson. She receives royalty income from this license., *Part 2:* My wife, Bonnie Morrison Davis, MD, is a patent holder on the use patent for galantamine for Alzheimer's disease and dementias that has been licensed to Janssen-Pharma, a subsidiary of Johnson & Johnson. She receives royalty income from this license., *Part 3:* My wife, Bonnie Morrison Davis, MD, is a patent holder on the use patent for galantamine for Alzheimer's disease and dementias that has been licensed to Janssen-Pharma, a subsidiary of Johnson & Johnson. She receives royalty income from this license.

Geraldine Dawson: *Part 1:* Professional Advisory Board for Integragen, Inc. from which I receive compensation for scientific advice.

Steven DeKosky: *Part 1:* Helicon Therapeutics Genzyme Merck, *Part 2:* none other than primary employment at the University of Virginia, *Part 4:* Elan Pharmaceuticals Novartis Janssen Forest

Melissa DelBello: *Part 1:* Eli Lilly, Amylin, AstraZeneca, Pfizer, Bristol Myers Squibb, Janssen, Johnson and Johnson, Somerset, Shire, Novartis, *Part 2:* Bristol Myers Squibb, Merck, *Part 3:* Bristol Myers Squibb, *Part 4:* Eli Lilly, Amylin, AstraZeneca, Pfizer, Bristol Myers Squibb, Janssen, Johnson and Johnson, Somerset.

Michael Detke: *Part 1:* MedAvante, Sonkei, Rhine, Columbia, Roche, NIH Pharamet/i3/ Inventiv (partner), *Part 2:* MedAvante, *Part 3:* MedAvante

Wayne Drevets: *Part 1:* Johnson & Johnson, consultant, Esai, Inc., consultant, Myriad/ Rules Based Medicine, consultant, *Part 2:* Johnson & Johnson

Ronald Duman: *Part 1:* Lundbeck, Lilly, Taisho, Forest, J&J, Bristol Myers Squibb, Pfizer, *Part 4:* Lundbeck, Lilly, Forest, J&J

Elliot Ehrich: *Part 1:* Alkermes plc, Full time employee, *Part 2:* Alkermes plc, Full time employee, *Part 3:* Alkermes plc, Full time employee

Wissam El-Hage: *Part 1:* WEH acted as a speaker or consultant for AstraZeneca, Bristol-Myers Squibb, Eli Lilly & Co., Janssen, Lundbeck, and Servier France.

Mary-Anne Enoch: *Part 2:* Government salary, two homes, one car

Cagla Eroglu: *Part 4:* Mazorx Inc provided a grant that supported our efforts to find activity blocking antibodies against TSPs.

ACNP 2012 Presenter Disclosures (continued)

Amit Etkin: *Part 4:* Grant from Brain Resource, Inc. for taking part in the International Study to Predict Optimized Treatment in Depression (iSPOT-D)

A. Eden Evins: *Part 1:* Pfizer, GSK, Envivo Pharmaceuticals, *Part 4:* Pfizer, GSK, Envivo Pharmaceuticals

Peter Falkai: *Part 1:* Until the end of 2010 I have been giving paid lectures and was on the advisory boards of the following pharmaceutical companies: Lilly, Servier, AstraZeneca, Janssen-Cilag, Pfizer and Lundbeck., *Part 2:* 20.000 US dollars., *Part 3:* No financial involvement of this amount., *Part 4:* I am currently having a grant from Servier.

Ellen Frank: *Part 1:* Servier International (Consultant) Vanda Pharmaceuticals (Consultant) Guilford Press and American Psychological Association Publishing (Royalties), *Part 2:* Servier International (Consultant) Spouse: Consultant to the American Psychiatric Association

Alan Frazer: *Part 1:* Lundbeck Takeda Lilly Cyberonics, *Part 4:* Lundbeck

Marlene Freeman: *Part 1:* Investigator-Initiated Trials (grants to MGH): Forest, Lilly, GlaxoSmithKline; 1 day advisory board: Bristol Myers Squibb, *Part 2:* Journal of Clinical Psychiatry, *Part 4:* Investigator-Initiated Trials (grants to MGH): Forest, Lilly, GlaxoSmithKline; 1 day advisory board: Bristol Myers Squibb

Jeffrey Friedman: *Part 1:* Envoy Therapeutics, Bay City Capital, Anglian Pharmaceutical, *Part 2:* Envoy Therapeutics, Bay City Capital, Anglian Pharmaceutical

Daniel Geschwind: *Part 1:* Synapdx -- scientific advisory board/consultant Roche -- ad hoc advisory board., *Part 2:* Synapdx 2011-2012

Donald Goff: *Part 1:* In the past 2 years Dr. Goff has received honoraria for consulting or speaking from Hoffman-La Roche, Eli Lilly, Takeda, Dainippon Sumitomo, Endo Pharmaceuticals, Janssen, Cypress Bioscience, Bristol Myer Squibb, Abbott Laboratories and served on a DMC for Otsuka., *Part 4:* Dr. Goff received research funding from Pfizer, GlaxoSmithKline, Janssen, Novartis and PamLab

David Goldman: *Part 2:* NIH - salary Elsevier - book contract American Century - Mutual Fund Thrift Savings Plan - IRA Home Car

Anthony Grace: *Part 1:* Johnson & Johnson, Lundbeck, Pfizer, GSK, Puretech Ventures, Merck, Takeda, Dainippon Sumitomo, Otsuka, Lilly, Roche, *Part 2:* Johnson & Johnson, *Part 3:* Johnson & Johnson, *Part 4:* Lundbeck, GSK, Lilly

Guy Griebel: *Part 1:* Employee of Sanofi.

Roland Griffiths: *Part 1:* Heffter Research Institute - Member of Board of Directors - 2 grants for clinical trials with psilocybin Transept Pharmaceutical - consulting Alexza Pharmaceuticals - grant for clinical trial Bristol-Myers Squibb - consulting Merck and Co - consulting Vanda Pharmaceuticals - consulting, *Part 4:* Alexza Pharmaceutical --A grant was provided to Johns Hopkins to conduct an abuse liability evaluation with a novel drug delivery system Heffter Research Institute -- Two grants to conduct clinical trials with psilocybin

ACNP 2012 Presenter Disclosures (continued)

Dimitri Grigoriadis: *Part 1:* I am a full time employee of Neurocrine Biosciences Inc., *Part 2:* Salary and Equity associated with full time employment at Neurocrine Biosciences Inc., *Part 3:* I am a full time employee of Neurocrine Biosciences Inc.

Ruben Gur: *Part 2:* Defender's association and Federal and State courts (detailed in my disclosure to the University), *Part 4:* Investigator-initiated grants from Pfizer and AstraZeneca

Jan-Ake Gustafsson: *Part 1:* Consultant for KaroBio AB., *Part 2:* Consultancy fee from KaroBio AB., *Part 3:* No., *Part 4:* No.

Suzanne Haber: *Part 1:* Pfizer

Chang-Gyu Hahn: *Part 4:* Pfizer Pharmaceuticals

Paul Harrison: *Part 1:* Honoraria for scientific talks: AstraZeneca, Otsuka, Takeda Advisory board: Merck, *Part 2:* University of Oxford, *Part 4:* Unrestricted educational grant from Takeda (Cambridge, UK) to the Department of Psychiatry, University of Oxford

Markus Heilig: *Part 2:* none other than primary employment by the US Federal Government

W. Joseph Herring: *Part 1:* Presenter is an employee of Merck., *Part 2:* Merck, *Part 3:* Merck

Ian Hickie: *Part 1:* Paid Educational Seminars/Resources Servier Astra Zeneca Pfizer Eli-Lilly Travel Support from Pharmaceutical or Business Companies Servier Astra Zeneca Price Waterhouse Cooper Research Support from Pharmaceutical Companies: Servier Pfizer, *Part 2:* Professor of Psychiatry, University of Sydney Executive Director, Brain & Mind Research Institute, University of Sydney Clinical Consultant in Psychiatry, Sydney Local Health District (NSW Government Services) Bupa Australia (Private Health Insurance) - Member of the Medical Advisory Panel Headspace: the National Youth Mental Health Foundation- Director on behalf of the University of Sydney, which is a member of the Company.(ENDED JAN 2012, *Part 4:* Hickie I. (2011-2012) Project: Does circadian disturbance predict response to sleep-wake interventions in early-onset depression. Servier, \$100,000.

Florian Holsboer: *Part 1:* Cofounder of HMNC GmbH (Biotech Company) Cofounder and shareholder of Affectis AG (Biotech Company)

Steven Hyman: *Part 1:* Novartis Science Board, *Part 2:* Novartis Fidelity Biosciences (Venture) Access BridgeGap (Venture)

Iliyian Ivanov: *Part 1:* Memeber of Data Safety Monitoring Board for Lundbeck., *Part 4:* Co-investigator on a neuroimaghg grant from Shire awarded to Dr Jeffrey Newcorn at Mont Sinai School of Medicine.

Daniel Javitt: *Part 1:* Solvay, Sepracor, AstraZeneca, Pfizer, Cypress, Merck, Sunovion, Lilly, BMS, Takeda, Glytech, AASI, Promentis, *Part 2:* Pfizer, Glytech, *Part 3:* Glytech, *Part 4:* Pfizer, Roche,

Jarcho Johanna: *Part 2:* National Institute of Health postdoctoral fellowship stipend

Rene Kahn: *Part 1:* AstraZeneca, BMS, Lilly, Otsuka, Roch, Sunovion

ACNP 2012 Presenter Disclosures (continued)

Ned Kalin: *Part 1:* Neuronetics CeNeRx BioPharma Corcept Therapeutics CME Outfitters Elsevier, *Part 2:* Elsevier, *Part 3:* Not applicable, *Part 4:* APIRE/Janssen Resident Psychiatric Mentor Grant National Institute of Mental Health The Stanley Medical Research Institute

Peter Kalivas: *Part 2:* Medical University of South Carolina Medical University Associates

Shitij Kapur: *Part 1:* Has served as a one-off consultant and/or speaker for AstraZeneca, Bioline, Bristol Meyers Squibb, Eli Lilly, Envivo, Janssen - Johnson and Johnson, NeuroSearch, Otsuka, Pfizer, Sanofi-Aventis, Schering-Plough, Servier, Solvay Wyeth and serves on the Scientific Advisory Boards for Lundbeck and Roche., *Part 4:* GSK, Lundbeck.

Richard Keefe: *Part 1:* Abbott, Amgen, Astellas, Asubio, Boehringer-Ingelheim, BiolineRx, Bristol-Myers Squibb, Eli Lilly, EnVivo, Helicon, Lundbeck, Merck, Mitsubishi, Novartis, Otsuka, Pfizer, Roche, Shire, Sunovion, Takeda, Targacept, *Part 2:* Abbott, Amgen, BiolineRx, Eli Lilly, EnVivo, Lundbeck, Mitsubishi, Pfizer, Roche, Shire, Sunovion, Takeda, Targacept, *Part 3:* NeuroCog Trials, Inc., *Part 4:* GSK, Novartis, PsychoGenics, Sunovion

Matcheri Keshavan: *Part 1:* I have received two grants, one from Sunovion and another from GSK, in the last two years., *Part 4:* I have received two grants, one from Sunovion and another from GSK, in the last two years.

Thomas Kilduff: *Part 1:* Since I am not a member of ACNP, I have no idea what “companies doing business with or proposing to do business with ACNP over past 2 years.”, *Part 4:* CHDI EMD Serono Research Institute F. Hoffman-La Roche, Ltd. Sunovion

Michael Kilgard: *Part 1:* I am a consultant for and have a financial interest in MicroTransponder, Inc., *Part 2:* I am a consultant for MicroTransponder, Inc., *Part 3:* I am a consultant for MicroTransponder, Inc., *Part 4:* I have a sub-contract from MicroTransponder, Inc. to conduct research that is supported in part by the NIH.

Lori Knackstedt: *Part 2:* MUSC

Scott Kollins: *Part 1:* Research Support: Rhodes Pharmaceuticals, Shire Pharmaceuticals, Otsuka Pharmaceuticals, Addrenex/Shionogi Pharmaceuticals Consultant/Advisory Board: Shire Pharmaceuticals, Otsuka Pharmaceuticals, Addrenex/Shionogi Pharmaceuticals, WebMD/Medscape, *Part 2:* Shire Pharmaceuticals, Otsuka Pharmaceuticals, *Part 4:* Shire Pharmaceuticals

George Koob: *Part 1:* Addes, Alkermes, Arkeo, Embera, Psychogenics

Andrew Krystal: *Part 1:* Abbott, Astellas, AstraZeneca, BMS, Cephalon, Eisai, Eli Lilly, GlaxoSmithKline, Jazz, Johnson and Johnson, MECTA Corp, Merck, Neurocrine, Novartis, Ortho-McNeil-Janssen, Respiroics, Roche, Sanofi-Aventis, Somnus, Sunovion/Sepracor, Somaxon, Takeda, Transcept, Kingsdown Inc., *Part 4:* NIH, Cephalon, Pfizer, Sunovion/Sepracor, Takeda, Transcept, Phillips-Respiroics, Astellas, Abbott, Neosynch, Brainsway.

David Kupfer : *Part 2:* University of Pittsburgh American Psychiatric Association

ACNP 2012 Presenter Disclosures (continued)

Steven Kymes: *Part 1:* I am a consultant and receive (or have received) research funding for Pfizer, Bayer and Genentech., *Part 2:* Consulting fees from Bayer in excess of \$10,000 in 2011 and 2012, *Part 4:* As noted above, Pfizer and Genenech in 2010 and 2011.

Paul Lombroso: *Part 1:* President, STEP Solutions, New Haven, CT, *Part 2:* \$0

Dan Lubman: *Part 1:* Honorarium for lectures (AstraZeneca, Janssen)

Angus MacDonald: *Part 2:* University of Minnesota

Robert Malenka: *Part 1:* Circuit Therapeutics, Inc. SAB member

Craig Mallinckrodt: *Part 1:* Employee of Eli Lilly and Co, *Part 2:* Employee of Eli Lilly and CO, *Part 3:* Employee of Eli Lilly and Co

Husseini K. Manji: *Part 1:* Husseini Manji is a full time salaried employee of Johnson & Johnson, *Part 2:* Husseini Manji is a full time salaried employee of Johnson & Johnson, *Part 3:* Husseini Manji is a full time salaried employee of Johnson & Johnson, *Part 4:* Husseini Manji is a full time salaried employee of Johnson & Johnson

Stephen Marder: *Part 1:* Consultant: Abbott, Amgen, Targacept, Lundbeck, Pfizer, Roche, Bristol Meyers Squibb, Otsuka., *Part 4:* Research Support: Novartis, GSK, Sunovion, Psychogenics

Barbara Mason: *Part 1:* SAB: Addex Pharmaceuticals, Lohocla Research Corporation; Consultant: Johnson & Johnson Pharmaceutical Research & Development, LLC, Lilly USA, LLC; Speaker: Merck KGaA

Colleen McClung: *Part 1:* I have received honoraria from Johnson & Johnson, Servier, and Pfizer, *Part 4:* We received research funding from GlaxoSmithKline and Pfizer

James Meador-Woodruff: *Part 2:* Primary employer University of Alabama School of Medicine and University of Alabama Health Servces Foundation; ACNP provides an honorarium for service as Editor in Chief of Neuropsychopharmacolgy, *Part 4:* American Foundation for Suicide Prevention and Ortho-McNeil. Served as PI on an Investigator Initiated project (jointly funded by both) for three month transition between original and current PIs.

Lin Mei: *Part 1:* SAB member, Mind-NRG

Kalpna Merchant: *Part 1:* I am an employee of Eli Lilly and company, a major pharmaceutical company., *Part 2:* As an employee of Eli Lilly and company, I receive income and equity from the company., *Part 3:* I am an employee of Eli Lilly and company, a major pharmaceutical company.

ACNP 2012 Presenter Disclosures (continued)

Jeffrey Meyer: *Part 1:* The author has been a consultant for companies that make antidepressants, including Eli Lilly, SK Life Sciences, Lundbeck, Takeda, Bristol-Myers Squibb, and GlaxoSmithKline, and has received operating grants from some of these companies. It is likely that he will in the future receive consulting contracts or operating grants from companies that make MAO-A inhibitors. He has applied for a patent to use MAO-A measures to predict the course of illness and to treat major depressive disorder., *Part 2:* The author has been a consultant for companies that make antidepressants, including Eli Lilly, SK Life Sciences, Lundbeck, Takeda, Bristol-Myers Squibb, and GlaxoSmithKline, and has received operating grants from some of these companies., *Part 4:* The author has been a consultant for companies that make antidepressants, including Eli Lilly, SK Life Sciences, Lundbeck, Takeda, Bristol-Myers Squibb, and GlaxoSmithKline, and has received operating grants from some of these companies.

David Michelson: *Part 1:* Employee of Merck and Co., *Part 2:* Merck salary and stock, *Part 3:* Merck employee

Emmanuel Mignot: *Part 1:* Novo Nordisk - consulting Jazz Pharmaceuticals - consulting GSK - Advisory Board

Jennifer Mitchell: *Part 1:* I am a founding member of a company that will eventually provide ex vivo GPCR screening.

Lisa Monteggia: *Part 1:* Speaker for Roche and Sepracor

Elaine Morrato: *Part 1:* I have received research grant funding through my university from Janssen Pharmaceuticals, Inc., *Part 4:* I have received research grant funding through my university from Janssen Pharmaceuticals, Inc (2012).

John Morrison: *Part 2:* I have developed monoclonal antibodies that are sold as research reagents that generate royalties in excess of \$10,000 per year.

Charles Nemeroff: *Part 1:* Research/Grants: National Institutes of Health (NIH), Agency for Healthcare Research and Quality (AHRQ) Speakers Bureau: None Consulting: Xhale, Takeda, SK Pharma, Shire, Roche, Lilly Stockholder: CeNeRx BioPharma, PharmaNeuroBoost, Revaax Pharma, Xhale, NovaDel Pharma Other Financial Interests: CeNeRx BioPharma, PharmaNeuroBoost Patents: Method and devices for transdermal delivery of lithium (US 6,375,990B1) Method of assessing antidepressant drug therapy via transport inhibition of monoamine neurotransmitters by ex vivo assay (US 7,148,027B2) Scientific Advisory Boards: American Foundation for Suicide Prevention (AFSP), CeNeRx BioPharma, National Alliance for Research on Schizophrenia and Depression (NARSAD), Xhale, PharmaNeuroBoost, Anxiety Disorders Association of America (ADAA), Skyland Trail, AstraZeneca Pharmaceuticals (2009) Board of Directors: AFSP, Mt. Cook Pharma (2010), NovaDel (2011), Skyland Trail, Gratitude America Income sources or equity of \$10,000 or more: AstraZeneca Pharmaceuticals (2009), PharmaNeuroBoost, CeNeRx BioPharma, NovaDel Pharma, Reevax Pharma, American Psychiatric Publishing, Xhale, *Part 2:* Income sources or equity of \$10,000 or more: AstraZeneca Pharmaceuticals (2009), PharmaNeuroBoost, CeNeRx BioPharma, NovaDel Pharma, Reevax Pharma, American Psychiatric Publishing, Xhale

ACNP 2012 Presenter Disclosures (continued)

Eric Nestler: *Part 1:* PsychoGenics, SAB chair Merck, Consultant Berg Pharma, Consultant, *Part 2:* PsychoGenics, SAB chair Merck, Consultant Berg Pharma, Consultant

John Newcomer: *Part 1:* AstraZeneca Pharmaceuticals Bristol-Myers Squibb BioVail H. Lundbeck Obecure Otsuka Pharmaceuticals Sepracor, Inc. Solvay Pharma, Inc. Teva Pharmaceutical Dainippon Sumitomo Pharma America, Inc. Organon Pharmaceuticals USA Inc. Schering-Plough / Merck Vivus, Inc. Consultant to Litigation Boehringer-Ingelheim, *Part 3:* AstraZeneca Pharmaceuticals Bristol-Myers Squibb BioVail H. Lundbeck Obecure Otsuka Pharmaceuticals Sepracor, Inc. Solvay Pharma, Inc. Teva Pharmaceutical Dainippon Sumitomo Pharma America, Inc. Organon Pharmaceuticals USA Inc. Schering-Plough / Merck Vivus, Inc. Consultant to Litigation Boehringer-Ingelheim, *Part 4:* The National Institute of Mental Health (NIMH) Bristol-Myers Squibb Pfizer, Inc.

Ginger Nicol: *Part 1:* Dr. Nicol has received research funding from the National Institute of Mental Health (NIMH), NARSAD, the Dana Brown Charitable Trust Foundation, the Sidney R. Baer, Jr. Foundation, and the CHADS Coalition for Mental Health. She also receives grant support from Pfizer, Inc. for an investigator-initiated clinical trial. She receives royalties from Jones & Barlett Learning for development of a pediatric metabolic monitoring form and has consulted to MedScape. She does not participate in speakers' bureaus., *Part 4:* Investigator initiated clinical trial grant support: Pfizer, Inc.

Michael Nitsche: *Part 1:* Advisory Boards: UCB, Eisai, GSK, Starstim

Charles O'Brien: *Part 1:* Alkermes consultant Reckitt consultant Embera consultant, *Part 2:* Alkermes Consultant

Charles O'Brien: *Part 1:* Alkermes-Consultant Embera- Consultant Reckitt-Consultant, *Part 2:* Alkermes- Consultant over 2 years

M. Foster Olive: *Part 1:* April - November 2011 - Principal Investigator of contract work for Gilead Sciences

Alvaro Pascual-Leone: *Part 1:* Neosync вЂ“ Member of Scientific Advisory Board Starlab вЂ“ Member of Scientific Board Neuronix вЂ“ Member of Medical and Scientific Advisory Board Johnson & Johnson, Codman вЂ“ Member of Medical Advisory Board on Neurological and Neurosurgical Technology Nexstim вЂ“ Advisory Board Member Novavision вЂ“ Chair, Advisory Board Member Institut Guttmann, Spain - Member, Board of Scientific Advisors *Part 4:* Investigator initiated grants from Neuronix, Nexstim, Neuronetics

Sanjeev Pathak: *Part 1:* AstraZeneca; *Part 2:* AstraZeneca; *Part 3:* AstraZeneca; *Part 5:* AstraZeneca

Mani Pavuluri: *Part 2:* AstraZeneca speaker, *Part 4:* Abbott Laboratories and Janssen Pharmaceuticals supplied placebo and active drug for NIH Funded Divalproex vs. Risperdal Double-Blind Placebo Controlled Trial.

Katharine Phillips: *Part 1:* Forest Laboratories (medication only for an NIMH-funded study) Transcept Pharmaceuticals (research funding) Oxford University Press (royalties) Guilford Publications and The Free Press (potential royalties) Elsevier (future honorarium), *Part 2:* NIMH, FDA, Rhode Island Hospital, Alpert Medical School of Brown University, *Part 4:* Forest Laboratories (medication only for an NIMH-funded study) Transcept Pharmaceuticals (research funding)

ACNP 2012 Presenter Disclosures (continued)

Daniel Pine: *Part 2:* Clinical Practice; Editor, American Journal of Psychiatry; NIMH Researcher

William Potter: *Part 1:* Consultant to: AgeneBio, Amgen, Astellas, BMS, EnVivo, Envoy, Index Ventures, J&J, MedAvante, Orasi, Otsuka, Pfizer, Sonkei, Takeda, Theravance, *Part 2:* Merck and Eli LillyStock, *Part 3:*Merck in 2010

Joseph Price: *Part 2:* Faculty salary from Washington University in St. Louis

Eugenii Rabiner: *Part 1:* Until October 2011, was a full time employee of GlaxoSmithKline. I am currently a consultant for GlaxoSmithKline, Biotie and Takeda., *Part 2:* Full time employee of GlaxoSmithKline up to October 2011

Scott Rauch: *Part 1:* UTSW Grand Rounds - Honorarium (2010) Cleveland Clinic Grand Rounds - Honorarium (2010) NIMH Conte Center - OCD Advisory Board - Honorarium (2010) NIMH RDoC - Honorarium (2011) Hall Mercer - Board Member (2010 & 2011) Oxford Univeristy Press - Royalty (2010 & 2011) APPI - Royalty (2010 & 2011), *Part 2:* None other than primary employer: McLean Hospital/Partners Healthcare., *Part 3:* None other than primary employer: McLean Hospital/Partners Healthcare., *Part 4:* Cyberonics Medtronic

Darrel Regier: *Part 4:* Dr. Darrel A. Regier, Director of APIRE and Director of the Division of Research, American Psychiatric Association, oversees all Federal and industry sponsored research and research training grants in APIRE but receives no external salary funding or honoraria from any government or industry sources.

Perry Renshaw: *Part 1:* I am a Consultant and stockholder for Ridge Diagnostics, I am a Consultant for Kyowa Hakko Kirin. I have received royalties on a patent describing the use of uridine to treat bipolar depression from Repligen.

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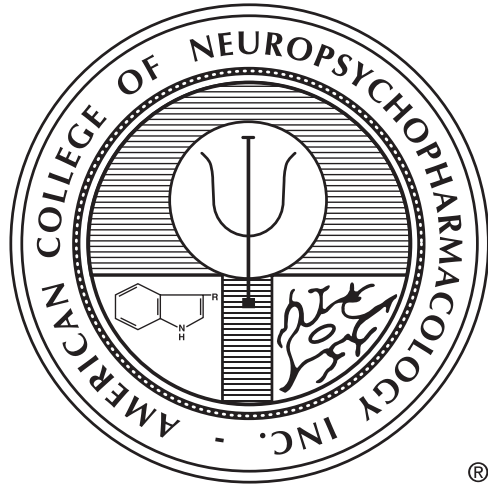
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